



One-Year Outcome of Patients with Coronary Artery Ectasia Undergoing Percutaneous Coronary Intervention: Clinical Implications and Question Marks

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Abstract

Background: Coronary artery ectasia (CAE) is a rare condition with unclear pathophysiology, optimal treatment, and prognosis. We aimed to determine the prognostic implications of CAE following coronary angioplasty.

Methods: We conducted a retrospective cohort study on 385 patients, including 87 subjects with CAE, who underwent percutaneous coronary intervention (PCI). Major adverse cardiovascular events (MACE) were considered to consist of mortality, nonfatal myocardial infarction (MI), repeated revascularization, and stroke.

Results: The mean age of the participants was 57.31±6.70 years. Multivariate regression analysis revealed that patients with diabetes, ST-segment-elevation MI at presentation, and high thrombus grades were more likely to have suboptimal post-PCI thrombolysis in myocardial infarction (TIMI) flow. However, CAE was not a predictor of a decreased TIMI flow (OR: 1.46, 95% CI: 0.78–8.32; P=0.391). The Cox-regression model showed that CAE, the body mass index, and a family history of MI were risk factors for MACE, while short lesion lengths (<20 vs >20 mm) had an inverse relationship. The adjusted hazard ratio (HR) for the prediction of MACE in the presence of CAE was 1.65 (95% CI: 1.08–4.78; P=0.391). All-cause mortality (HR: 1.69, 95% CI: 0.12–3.81; P=0.830) and nonfatal MI (HR: 1.03, 95% CI: 0.72–4.21; P=0.341) occurred similarly in the CAE and non-CAE groups. Conversely, CAE increased urgent repeat revascularization (HR: 2.40; 95% CI: 1.13–5.86; P=0.013).

Conclusion: Although CAE had no substantial short-term prognostic effects on post-PCI TIMI flow, considerable concerns regarding adverse outcomes emerged during our extended follow-up. Stringent follow-ups of these patients should be underscored due to the high likelihood of urgent revascularization.

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Introduction

Among patients undergoing coronary arteriography, coronary artery ectasia (CAE), defined as a diameter in the coronary arteries exceeding the normal segments by 1.5 times,¹ is a rare condition with a reported prevalence of between 0.2% and 6.0% in several studies.¹⁻⁵ While it has been postulated that CAE might be primarily a congenital atherosclerotic entity, connective tissue disorders and inflammatory diseases are also considered possible etiologies. Nonetheless, the true in-depth pathophysiology of CAE remains to be determined.⁶

The clinical advent of CAE, as well as its therapeutic and prognostic implications, mostly depends on the features of the adjacent coronary anatomy. In patients with segments of severe coronary artery stenosis, the clinical manifestation resembles that of an acute coronary syndrome.^{1, 7, 8} On the other hand, in patients without hemodynamically significant coronary artery stenosis, the clinical picture is expressed in the same manner as stable angina with positive exercise stress tests, which is mostly attributed to the reduced coronary flow velocity in ectatic segments.^{6, 7, 9} There are published case reports regarding ectatic lesions responsible for episodes of acute myocardial infarction (MI). These papers signify that despite its relatively low prevalence, CAE may bring on notable prognostic consequences.^{10, 11} Many authors consider these lesions high risk for performing PCI due to the enhanced activation of the thrombin cascade. Moreover, turbulent blood flow in dilated coronary arteries can lead to increased coagulation vulnerability.¹²

There are scarce published materials on the outcome of patients with CAE and concomitant coronary artery stenosis in ectatic segments undergoing percutaneous coronary intervention (PCI). Furthermore, these lesions are considered diagnostic and therapeutic challenges even to the most seasoned of interventionalists. Accordingly, we designed the present study to investigate the outcomes of revascularizations performed on patients with CAE and assess their adverse cardiovascular events in months following their angioplasty.

Methods

The Data Registry System of Tehran Heart Center was drawn upon to identify 87 patients diagnosed with CAE undergoing PCI. A second group of patients without CAE (n=298) but with a history of PCI were selected for the initial analysis. The clinical, angiographic, procedural, and follow-

up data (including office and emergency department visits) of all the subjects were gathered. A retrospective cohort study was designed and performed by using the records of patients between June 2016 and October 2018. The median follow-up period was 11 (9–12) months. Stringent eligibility criteria were meticulously defined and adhered to for the enrollment process. The exclusion criteria were comprised of age under 18 or above 80 years; a history of prior coronary artery bypass graft surgery (CABG) at any time or PCI within 6 months preceding the index PCI, cardiopulmonary resuscitation, or severe renal dysfunction (glomerular filtration rate <30 mL/min/1.73 m²); the consumption of thrombolytic agents; stent thrombosis; balloon angioplasty without stenting; and unsuccessful procedures due to guidewire-passage failure.

The main angiographic parameter employed to evaluate the result of reperfusion was the thrombolysis in myocardial infarction (TIMI) flow grade as defined by the TIMI Working Group.¹³ In the mentioned scale, TIMI subtends 4 grades: TIMI-0 refers to no antegrade coronary flow beyond the atherosclerotic lesion; TIMI-1 displays a poor antegrade stream, portending the minimal delayed filling of the distal part; TIMI-2 describes a delayed runoff, leading to the partial filling of the distal segments of the vessel; and TIMI-3 depicts a normal and complete coronary blood flow.¹⁴

Additionally, the Markis classification was used to determine the extent of CAE, which was defined as a dilated segment with a diameter 1.5 times or more than that of the contiguous normal segments.¹⁵ It comprises 4 subgroups: Type I: diffuse ectasia, involving 2 or 3 coronary arteries; Type II: diffuse dilatation, observed in a vessel with concomitant localized ectasia in another one; Type III: diffuse CAE in a single vessel; and Type IV: discrete segmental ectasia.

Cardiac troponin-T was assessed before and after the procedure as a surrogate measure indicating myocardial damage. The primary endpoint of the study was a composite of cardiovascular mortality, repeat revascularization either via PCI or via CABG, nonfatal acute MI, and acute stroke in both groups, which was abbreviated as major adverse cardiovascular events (MACE). The potential risks of MACE and its components such as nonfatal MI and recurrent revascularization were also determined for both CAE and non-CAE groups. The secondary endpoint of the study was suboptimal reperfusion, defined as a persistent TIMI flow grade of II or less. The potential relationship between CAE and favorable TIMI flow after PCI was also assessed.

All the patients received similar medical regimens in keeping with the standard guideline-directed medical treatment for coronary artery disease (CAD). Hence, loading doses of clopidogrel pertaining to P2Y₁₂ inhibitors



(600 mg initially and 75 mg once daily, thereafter), unfractionated heparin (70–100 unit /kg), aspirin (300 mg followed by 80 mg once daily), and atorvastatin (80 mg) were administered. Decision-making about thrombosuction and the administration of glycoprotein IIb/IIIa inhibitors was at the discretion of the interventional cardiologist. The distributions of other consumed drugs such as angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, beta-blockers, and nitrates as well as procedural techniques were identical in the CAE and non-CAE groups.

The study protocol adhered to the principles of the Declaration of Helsinki and the Ethics Committee of Tehran University of Medical Sciences.

Continuous variables were presented as the mean \pm the standard deviation (SD), and categorical variables were described as percentages. The Kolmogorov–Smirnov test was recruited to evaluate the normality of the distribution of different variables. The independent t-test was utilized to evaluate differences in continuous variables. Categorical variables were compared with the use of the χ^2 test. A P value of less than 0.05 was considered significant. All the statistical analyses were conducted with SPSS Statistics, version 25.0 (SPSS Inc, Chicago, IL). Additionally,

univariate and multivariate regression analyses were carried out to determine the associations between the predictors of a reduced TIMI flow grade and MACE. The multivariate regression analysis was performed to determine the adjusted association between the potential risk factors of a low ejection fraction and MACE. The Kaplan–Meier graphs were used to show event-free survival between the groups with and without ectasia at 1 year's follow-up. The Cox multivariate regression models were also applied to depict the cumulative adjusted hazards of total MACE, nonfatal MI, and further revascularization attributable to CAE against the non-CAE group.

Results

The Data Registry System of Tehran Heart Center was thoroughly reviewed, and 87 patients with a history of CAE undergoing PCI were identified. A total of 298 patients without CAE who underwent PCI were also identified. The demographic and clinical characteristics of these 2 groups are depicted in Table 1. Primary PCI for ST-segment-elevation myocardial infarction (STEMI) was performed in

Table 1. Baseline demographic, clinical, and angiographic characteristics of the patients enrolled in the study*

| | Non-CAE Group (n=298) | CAE Group (n=87) | P |
|--|-----------------------|--------------------|-------|
| Sex (male) | 285 (95.6) | 82 (94.3) | 0.453 |
| Age (y) | 57.46 \pm 10.33 | 57.06 \pm 9.78 | 0.910 |
| BMI (kg/m ²) | 28.69 \pm 5.17 | 28.83 \pm 5.25 | 0.874 |
| GFR (Cockcroft–Gault, mL/min/1.73 m ²) | 114.85 \pm 16.33 | 109.32 \pm 19.33 | 0.325 |
| LVEF (%) | 41.7 \pm 11.8 | 39.1 \pm 9.1 | 0.096 |
| STEMI | 57 (19.1) | 14 (16.1) | 0.232 |
| NSTEMI | 73 (24.5) | 21 (24.1) | 0.862 |
| Unstable angina | 106 (35.6) | 29 (33.3) | 0.341 |
| Stable angina | 62 (20.8) | 23 (26.4) | 0.121 |
| Lesion length (mm) | 22.54 \pm 9.89 | 23.41 \pm 11.68 | 0.121 |
| Lesion Type (ACC/AHA classification) | | | |
| B1 | 94 (31.5) | 25 (28.7) | 0.254 |
| B2 | 33 (11.1) | 10 (11.5) | 0.543 |
| C | 171 (57.4) | 52 (59.8) | 0.386 |
| Stent diameter (mm) | 3.16 \pm 0.51 | 3.63 \pm 0.65 | 0.054 |
| Stent length (mm) | 23.98 \pm 8.86 | 25.57 \pm 6.61 | 0.158 |
| Stent inflation pressure (atm) | 12.69 \pm 2.62 | 13.74 \pm 3.30 | 0.083 |
| High thrombus grade | 22 (7.4) | 14 (16.1) | 0.022 |
| Declined initial TIMI (<III) | 79 (26.5) | 40 (46.0) | 0.005 |
| Heavy calcification of the target vessel | 21 (7.0) | 3 (3.4) | 0.167 |
| Cigarette Smoking | | | |
| Current smoker | 148 (49.7) | 45 (51.7) | 0.218 |
| Former smoker | 40 (13.4) | 12 (13.8) | 0.876 |
| Diabetes mellitus | 52 (17.4) | 14 (16.1) | 0.613 |
| Positive family history of IHD | 48 (15.6) | 15 (19.5) | 0.092 |
| Hyperlipidemia | 138 (46.3) | 42 (48.3) | 0.314 |
| Hypertension | 114 (38.3) | 38 (43.7) | 0.067 |
| Chronic lung disease | 11 (3.7) | 1 (1.2) | 0.305 |
| ESRD (dialysis) | 1 (0.3) | 0 | 0.111 |
| Cerebrovascular disease | 9 (3.0) | 2 (2.3) | 0.743 |
| Opium use | | | |
| Current | 88 (29.5) | 22 (25.3) | 0.218 |
| Former | 9 (3.0) | 2 (2.3) | 0.867 |

*Continuous variables are expressed as mean \pm SD or n (%).

IHD, Ischemic heart disease; TIMI, Thrombolysis in myocardial infarction; LVEF, Left ventricular ejection fraction; ESRD, End-stage renal disease; ACC/AHA, American College of Cardiology/American Heart Association; STEMI, ST-segment-elevation myocardial infarction; GFR, Glomerular filtration rate; NSTEMI, Non-ST-segment-elevation myocardial infarction; BMI, Body mass index; atm, atmosphere

16.1% of the CAE group and 19.1% of the non-CAE group (P=0.237). The 2 study groups had similar rates of non-STEMI. Among the patients with CAE, 24.1% experienced non-STEMI, whereas 24.5% of the control group underwent PCI in such a setting. Stable angina and unstable angina were diagnosed for 26.4% and 33.3% of the subjects with CAE, which resembled the rates in the non-CAE group (20.8% and 35.60%, respectively).

The distributions of culprit vessels for angioplasty were similar between the CAE and non-CAE groups. The involvement rates of the left anterior descending, left circumflex, obtuse marginal, ramus intermedius, and right coronary arteries in the patients with ectasia were 45.6%, 7.0%, 2.0%, 1.0%, and 44.3%, correspondingly. Similar findings were observed vis-à-vis the aforementioned coronary territories in the control group (43.7%, 6.9%, 1.1%, 1.1%, and 47.2%, respectively). The difference between the groups in this respect, therefore, did not constitute statistical significance. The anatomic segments of culprit lesions were also similar in both groups: stenosis in the ostial and proximal parts was reported, respectively, in 7.1% and 21.1% of the CAE group and, correspondingly, 6.5% and 20.8% of the control group.

None of the subjects with CAE had coronary dissection and fibromuscular dysplasia. Two patients in the non-CAE group consumed warfarin, whereas none of the CAE group patients received anticoagulants. According to the Markis classification, the relative frequencies of CAE Types I-IV were 35 (40.2%), 12 (13.80%), 36 (41.4%), and 4 (4.6%), respectively. Note that in a patient with 2 or 3 ectatic vessels, PCI might be required for only 1 or 2 vessels, while angioplasty might be performed on a non-ectatic coronary artery.

The early results of PCI in the participants were identical regarding the elimination of the target lesion stenosis and optimal reperfusion. The primary success rates of angioplasty for the CAE and non-CAE groups were 96.4% and 95.7%, correspondingly (P=0.816). An unfavorable final TIMI flow grade (<III) was observed in 2.3% of the patients in the CAE group, which was similar to that of the controls (1.3%) (P=0.525). Table 2 presents the construction of 2 related models of multiple logistic regression to reveal the association between a suboptimal coronary flow and various factors, especially CAE. Declined preprocedural TIMI flow grades were more frequent in the CAE group than the non-CAE group (46.4% vs 26.5%; P=0.005). Reduced final TIMI flow grades (II or less) occurred in 2.3% of the CAE group as

Table 2. Multivariate logistic regression models for predicting suboptimal reperfusion after PCI (post-PCI TIMI <III)

| Predictors | Model I | | Model II | |
|---|-------------------|-------|------------------|-------|
| | OR (95% CI) | P | OR (95% CI) | P |
| Diabetes mellitus | 1.36 (0.88-3.39) | 0.072 | 1.52 (1.15-4.18) | 0.019 |
| Primary PCI (vs other diagnoses) | 1.11 (0.87-5.13) | 0.244 | 1.32 (1.06-2.12) | 0.021 |
| Thrombus grade (high vs low) | 2.74 (0.92-6.86) | 0.061 | 4.70 (1.02-8.63) | 0.045 |
| CAE | 1.85 (0.39-7.83) | 0.642 | 1.46 (0.78-8.32) | 0.391 |
| Severity of stenosis (per 10% increase) | 1.02 (0.81-1.23) | 0.840 | - | - |
| Stent length (per 5 mm increase) | 0.97 (0.71-1.31) | 0.852 | - | - |
| Dyslipidemia | 1.37 (0.49-11.28) | 0.761 | - | - |
| LV ejection fraction (per 5% increase) | 0.96 (0.90-1.07) | 0.652 | - | - |

Model I, Odds ratio values were adjusted for age, sex, BMI, lesion length, serum creatinine, cigarette smoking, family history of CAD, opium use, hypertension, stent generations, and ACC-AHA classification for the complexity of PCI (A, B1, B2, and C).

Model II, Adjustments were applied for the variables above in addition to target vessels, stent inflation pressure, and pre-PCI TIMI flow.

PCI, Percutaneous coronary intervention; TIMI, Thrombolysis in myocardial infarction, BMI, Body mass index; CAD, Coronary artery disease; ACC-AHA, American College of Cardiology- American Heart Association, LV, Left ventricle

Table 3. Cox multivariate regression analysis of the predictors of 1 year's MACE

| | Adjusted HR | 95% CI | P |
|-------------------------------------|-------------|------------|-------|
| Age (y) | 1.02 | 0.97 -1.06 | 0.471 |
| BMI (kg/m ²) | 0.90 | 0.82 -0.99 | 0.031 |
| Cigarette smoking | 1.85 | 0.61-5.57 | 0.184 |
| Family history of MI or CAD | 3.16 | 1.31 -7.61 | 0.011 |
| Dyslipidemia | 1.92 | 0.86-4.27 | 0.109 |
| Hypertension | 1.35 | 0.56-3.26 | 0.502 |
| LVEF (per 10% increase) | 0.97 | 0.94 -1.00 | 0.061 |
| Short lesion length (<20 vs >20 mm) | 0.94 | 0.90 -0.98 | 0.012 |
| CAE (CAE vs non-CAE) | 1.65 | 1.08 -4.78 | 0.028 |
| Stent diameter | 0.67 | 0.28 -1.57 | 0.353 |
| Calcification (high vs low burden) | 0.85 | 0.19 -3.82 | 0.827 |
| Total occlusion | 0.34 | 0.06 -1.93 | 0.224 |
| Creatinine (per 0.1 mg/dL increase) | 0.74 | 0.18 -3.03 | 0.671 |

The model was adjusted for opium use, PCI-treated vessel, initial severity of stenosis, stent generation, PCI location (ostial, proximal, and non-proximal parts), thrombus grade, setting of PCI (primary PCI vs others), and Pre-PCI TIMI.

MACE, Major adverse cardiovascular events; MI, Myocardial infarction; CAD, Coronary artery disease; LVEF, Left ventricular ejection fraction; CAE, Coronary artery ectasia; PCI, Percutaneous coronary intervention; TIMI, Thrombolysis in myocardial infarction



compared with 1.3% of the control group (P=0.527).

The multivariate analysis demonstrated the association between CAE and MACE (Table 3). In addition, a propensity score-matching model was constructed through the selection of 77 patients from each category. In this model, there were no significant differences in clinical characteristics between the 2 groups.

Apropos the outcomes in the CAE and non-CAE groups, both in the primary analysis and in the propensity score-matching analysis, despite a considerable blunting effect after propensity-score matching, the cumulative incidence of MACE was still greater in the CAE group. The hazard ratio

(HR) of MACE was 1.65 (95% CI: 1.08–4.78; P=0.016) before propensity-score matching, but it dropped to 1.33 (95% CI: 1.04–3.342; P=0.038) after this process. The Kaplan–Meier survival chart also showed this significant divergence between the 2 groups starting from the second month. Figures 1A and 1B depict the estimates for the HR of MACE and all-cause mortality in the presence of CAE. Figures 2A and 2B also illustrate the adjusted HR of nonfatal MI and repeat revascularization (target vessel and/or target lesion) in both groups, with and without CAE.

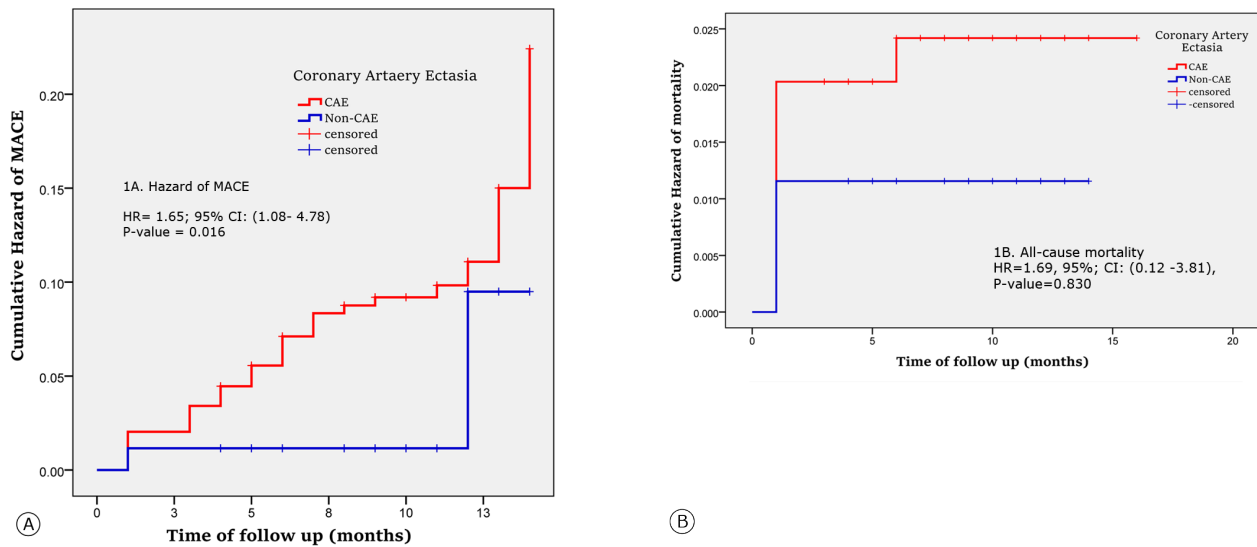


Figure 1. A) Kaplan–Meier curves illustrate the cumulative hazards of total MACE following the index PCI in the CAE and non-CAE groups. B) Kaplan–Meier graph shows the cumulative hazard of all-cause death.

MACE, Major adverse cardiovascular events; PCI, Percutaneous coronary intervention; CAE, Coronary artery ectasia

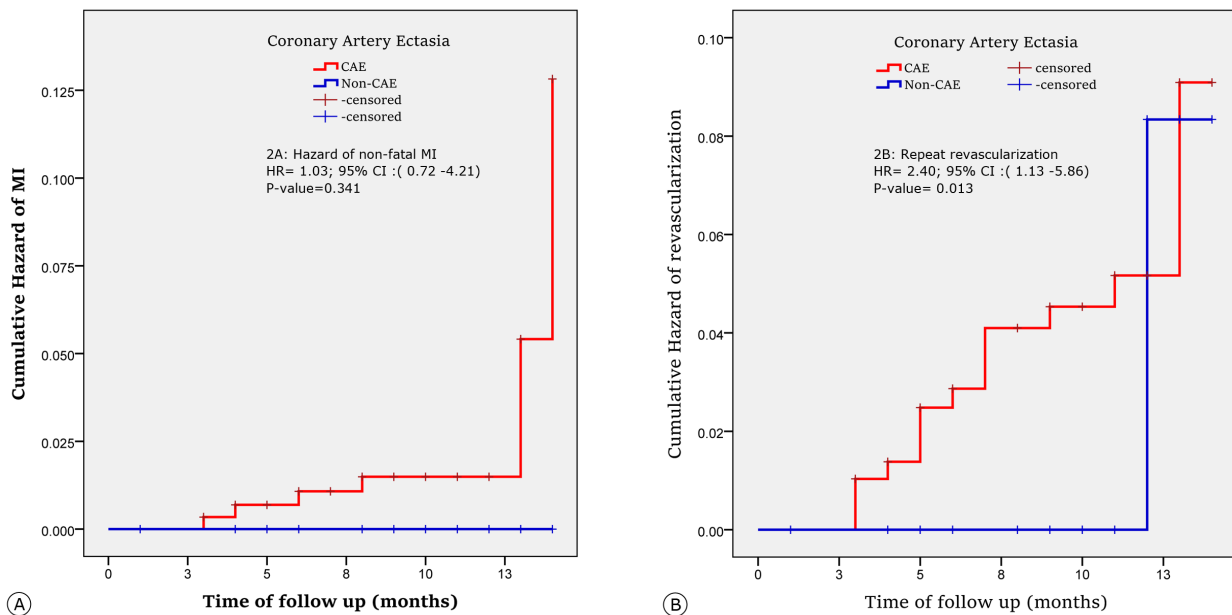


Figure 2. A) The image illustrates the cumulative hazards pertaining to non-fatal MI and B) repeated revascularization following PCI in patients with and without CAE. MI, Myocardial infarction; CAE, Coronary artery ectasia

Discussion

It is a well-known fact that CAE often co-exists with obstructive CAD and the overall outcome of patients with CAE mostly depends on the presence and severity of obstructive CAD.^{1,8,14} Still, there are reports from other studies indicating that patients with only isolated CAE might have different epidemiological, clinical, and prognostic features from those with mixed CAE.¹⁵ These findings necessitate due attention to isolated CAE as an independent clinical entity. PCI is the accepted method for the management of patients with CAE and MI. Its superiority over medical and surgical options and its related effects on the long-term prognosis are well established in the literature.^{16, 17} In clinical situations other than acute MI (symptomatic non-acute coronary syndrome-related ischemic heart disease), the efficacy and safety of PCI are less clear, however, which might be explained by the fact that most studies on the role of different management strategies in CAE patients have been underpowered by small sample sizes.¹⁸

The mere presence of ectasia and aneurysm in a coronary artery does not affect overall survival at 5 years.¹ Nevertheless, there are reports supporting the evidence that angioplasty on stenosis relating to an ectatic segment may increase the rate of clinical adverse events as compared with non-CAE cases.^{19, 20} In line with these findings, we detected a higher rate of MACE at 1 year's follow-up after angioplasty in our CAE group. Major effects were delivered via incident repeat revascularization (both surgical and percutaneous) rather than nonfatal MI or cumulative mortality. Notably, cardiovascular mortality was slightly higher in our CAE group, even though the statistical significance was borderline. Although CAE was associated with relatively poor long-term PCI outcomes (1 year's MACE), the short-term prognosis was not affected significantly either by diminished TIMI or by in-hospital mortality. Consistent with most of the previous reports, our results demonstrated that diabetes mellitus, STEMI as the clinical setting of PCI, and high thrombus burden were the independent predictors of decreased TIMI flow grades after angioplasty.^{21, 22} However, CAE did not contribute to the risk of slow flow, which is scintillating in that it is antithetical to the published evidence. Schram et al²³ revealed a robust relationship between CAE and the no-reflow phenomenon in a case-control study. Nevertheless, the authors enrolled only STEMI patients with greater frequencies of pre-PCI TIMI flow grades of 0 and 1 in cases than controls, while only a small proportion of these cases underwent PCI (47% vs 74%). The difference in our results might be explained by the fact that not only was our sample heterogeneous but also all of our subjects underwent PCI.

The single-center and retrospective nature of this study poses some caveats. The methodological barrier precluded us from determining whether CAE is an innocent bystander,

an intermediate part of the process, a risk factor, or a robust causative agent. We treated the majority of our study subjects via angioplasty in the setting of acute coronary syndrome, which might have restricted the effect size attributed to other categories and led to the underestimation of the true influence of CAE in patients with stable ischemic heart disease and unstable angina. Furthermore, the inherent mortality of MI is greater than that of stable angina. A heterogeneous sample of patients, as was investigated here, may reflect the overall picture of CAD. However, extrapolation of the results is far from simple since the paradigm of the emerging proportional incidence rates of various categories of CAD exhibits rapid alterations. The total number of events pertaining to cardiovascular mortality and MI was relatively low, lessening the power of our study in sub-analyses. Moreover, the male predominance observed in the present study may warrant caution in the extrapolation of the results to all CAD patients with CAE.

Conclusion

There are still notable questions left unanswered regarding the management and prognosis of CAE. Indeed, although recent years have witnessed great strides toward addressing many issues surrounding ectasia, comprehensive explanations for many of our unknowns are still lacking. The results of the current investigation showed that the long-term PCI outcome was worse in patients with CAE than patients without CAE, even though the outcome appears to be mainly dependent on the subsequent need for angioplasty. On the other hand, long-term follow-ups may reveal greater differences apropos all-cause mortality and nonfatal MI, which were similar in frequency in the present study.

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