Coronary Artery Ectasia Predicts Future Cardiac Events in Patients With Acute Myocardial Infarction

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Objective—Coronary artery ectasia (CAE) is an infrequently observed vascular phenotype characterized by abnormal vessel dilatation and disturbed coronary flow, which potentially promote thrombogenicity and inflammatory reactions. However, whether or not CAE influences cardiovascular outcomes remains unknown.

Approach and Results—We investigated major adverse cardiac events (MACE; defined as cardiac death and nonfatal myocardial infarction [MI]) in 1698 patients with acute MI. The occurrence of MACE was compared in patients with and without CAE. CAE was identified in 3.0% of study subjects. During the 49-month observation period, CAE was associated with 3.25-, 2.71-, and 4.92-fold greater likelihoods of experiencing MACE (95% confidence interval [CI], 1.88–5.66; *P*<0.001), cardiac death (95% CI, 1.37–5.37; *P*=0.004), and nonfatal MI (95% CI, 2.20–11.0; *P*<0.001), respectively. These cardiac risks of CAE were consistently observed in a multivariate Cox proportional hazards model (MACE: hazard ratio, 4.94; 95% CI, 2.36–10.4; *P*<0.001) and in a propensity score–matched cohort (MACE: hazard ratio, 8.98; 95% CI, 1.14–71.0; *P*=0.03). Despite having a higher risk of CAE-related cardiac events, patients with CAE receiving anticoagulation therapy who achieved an optimal percent time in target therapeutic range, defined as ≥60%, did not experience the occurrence of MACE (*P*=0.03 versus patients with percent time in target therapeutic range <60% or without anticoagulation therapy).

Conclusions—The presence of CAE predicted future cardiac events in patients with acute MI. Our findings suggest that acute MI patients with CAE are a high-risk subset who might benefit from a pharmacological approach to controlling the coagulation cascade.

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Key Words: cardiac events ■ coronary artery disease ■ ectasia ■ myocardial infarction ■ propensity score

Coronary artery ectasia (CAE) is a vascular phenotype that is infrequently observed in patients who undergo coronary angiography.[1–6] It is characterized by abnormal vessel dilatation associated with disturbed coronary flow.[7,8] Although CAE has been shown to be associated with enhanced thrombogenicity and inflammatory reactions, such as activation of tumor necrosis factor-α and interleukin-1β,[8] the clinical significance of CAE has not been fully elucidated yet.

Although CAE is uncommonly seen on coronary angiography, several case reports showed acute occlusion at the ectatic coronary segment within infarct-related arteries in patients with acute myocardial infarction (AMI).[9,10] In these cases, thrombectomy catheters retrieved substantial amounts of thrombus around ectatic lesions.[10] Postmortem

pathological studies also demonstrated spontaneous occlusion of dilated coronary arteries because of the presence of massive thrombi.[1,5] Because dilatation of the coronary arteries disturbs coronary flow and thereby increases blood viscosity and activates coagulation,[11] CAE may be a high-risk lesion causing acute coronary events. Furthermore, the enhanced thrombogenicity in CAE suggests a potential benefit of pharmacological agents for modulating the coagulation cascade to prevent CAE-related coronary events.[12] However, the association of CAE with cardiac events and the clinical efficacy of anticoagulation therapy remain uncertain. Therefore, the current study sought to (1) investigate clinical outcomes in AMI patients with CAE, and (2) evaluate the efficacy of anticoagulation therapy on major adverse cardiac events (MACE).

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Nonstandard Abbreviations and Acronyms	
AMI	acute myocardial infarction
CAE	coronary artery ectasia
CI	confidence interval
HR	hazard ratio
MACE	major adverse cardiac events
%TTR	percent time in target therapeutic range

Materials and Methods

Materials and Methods are available in the online-only Data Supplement.

Results

Prevalence and Angiographic Characteristics of CAE

Patient disposition is shown in Figure 1. In the current study, CAE was observed in 89 coronary arteries of 51 patients (3.0% of patients; 95% confidence interval [CI], 2.1–4.3). Representative cases of classical and alternative definition of CAE is illustrated in Figure 2. The conventional definition of CAE was applied in 67% (n=34) of CAE subjects, and the alternate definition was applied in the remaining cases (33%, n=17). Overall, the CAEs were located in 29 infarct-related arteries and 60 noninfarct related arteries, respectively. One patient each in CAE subjects had polycystic kidney disease and Ehlers–Danlos syndrome. None of patients with CAE had any angiographic coronary dissection and fibromuscular dysplasia. Table 1 summarizes the clinical characteristics of CAE. CAE was more frequently observed within the right coronary artery (39 of 51; 76%), followed by the left circumflex artery (28 of 51; 55%), left anterior descending artery (22 of 51; 43%), and left main trunk (10 of 51; 20%). Fifty-nine percent of CAE subjects showed multivessel CAE involvement. With regard to angiographic features of CAE, oscillatory and static flow were observed in 47% (24 of 51) and 27% (14 of 51) of patients with CAE, respectively. Of particular interest was that 37% (19 of 51) of patients with CAE exhibited concomitant dilatation of other vessels, including thoracic aorta (8%), abdominal aorta (8%), iliac artery (4%), and intracranial arteries (8%). One patient (2%) had both iliac and intracranial

artery aneurysms. Dissecting aortic aneurysm was identified in 4 patients (8%).

Baseline Clinical Demographics in Patients With CAE

Baseline clinical demographics in patients with and without CAE are shown in Table 2. Patients with CAE, compared with those without, were more likely to be younger (63±13 versus 68±12 years; P=0.005), men (84% versus 71%; P=0.04), more obese (body mass index ≥30 kg/m²; 12% versus 4%; P=0.008), and smokers (86% versus 71%; P=0.02) and to exhibit an increased number of coronary risk factors (2.9±1.1 versus 2.6±1.2; P=0.04) and higher left ventricular ejection fraction (49±8% versus 45±10%; P=0.003). Warfarin was more frequently used in patients with CAE (37% versus 13%; P<0.001), but these patients were less likely to receive dual antiplatelet therapy (22% versus 34%; P=0.04). The use of other established medical therapies was comparable between the 2 groups.

Lesion Characteristics and Percutaneous Coronary Intervention Procedures

Lesion characteristics and details on percutaneous coronary intervention procedures are summarized in Table 2 and Table II in the online-only Data Supplement. There were no significant differences in the numbers of diseased coronary vessels (1.8±0.7 versus 1.7±0.8; P=0.30) and the prevalence of triple vessel disease (20% versus 22%; P=0.64) between patients with and without CAE. The Gensini score in patients with CAE was significantly lower compared with those without CAE (48 [27–81] versus 58 [40–87]; P=0.03). Primary percutaneous coronary intervention was conducted in 82% and 74% of patients with and without CAE, respectively (P=0.17). Lesion characteristics and number of treated vessels were comparable between the 2 groups. In patients receiving primary percutaneous coronary intervention, the frequency of stent use was lower in individuals with CAE than those without (62% versus 91%; P<0.001), whereas those with CAE were more likely to be treated with an intraaortic balloon pump (29% versus 14%; P=0.01). In patients not treated with stents, balloon angioplasty without stent use (31% versus 7%; P<0.001) and thrombectomy (31% versus 2%; P<0.001) were more frequently used in the CAE group.
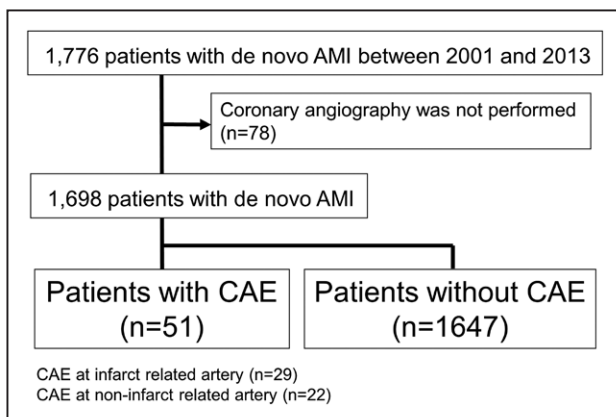
Figure 1. Flowchart of the study subjects. AMI indicates acute myocardial infarction; and CAE, coronary artery ectasia.
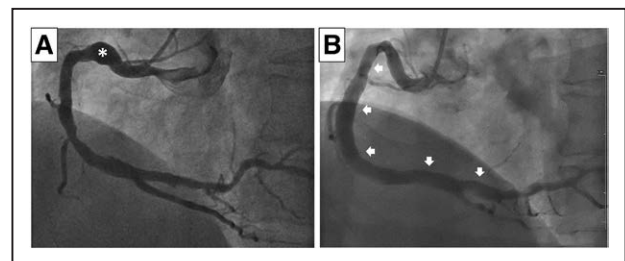
Figure 2. Definition of coronary artery ectasia (CAE). CAE was defined as coronary artery dilatation of >1.5-fold the diameter of adjacent normal coronary segments (**asterisk**; **A**). In subjects who did not have adjacent normal segments (**arrows**; **B**), the mean diameter of each coronary segment in 51 age- and sex-matched patients without heart disease were assigned as a normal reference diameter, and a 1.5-fold difference in diameter between affected and normal reference diameter was used to define CAE.

Table 1.   Clinical Characteristics of CAE

	CAE (n=51)
Involved vessel, n (%)	
Right coronary artery	39 (76)
Left anterior descending artery	22 (43)
Left circumflex artery	28 (55)
Left main trunk	10 (20)
Multivessel involvement	30 (59)
The presence of CAE in infarct-related artery	29 (57)
Angiographic features of CAE, n (%)	
Oscillatory flow	24 (47)
Static flow	14 (27)
Other vessels with concomitant dilatation, n (%)	
Overall	19 (37)
Thoracic aorta	4 (8)
Abdominal aorta	4 (8)
Iliac artery	2 (4)
Intracranial artery	4 (8)
Iliac and intracranial arteries	1 (2)
Dissecting aortic aneurysm	4 (8)

Categorical variables are expressed as n (%). CAE indicates coronary artery ectasia.

As a consequence, the proportion of patients who achieved Thrombolysis in Acute Myocardial Infarction flow grade 3 was significantly lower in CAE subjects (45% versus 88%; *P*<0.001; Figure 1).

Long-Term Outcomes in Patients With CAE

Figure 3, Tables 3 and 4, and Tables IV and V in the online-only Data Supplement compare the occurrence of MACE, cardiac death, and nonfatal myocardial infarction (MI) in the 2 groups. During the observation period (median, 49 months; interquartile range, 19–93 months), significantly higher incidences of MACE (hazard ratio [HR], 3.25; 95% CI, 1.88–5.66; *P*<0.001), cardiac death (HR, 2.71; 95% CI, 1.37–5.37; *P*=0.004), and nonfatal MI (HR, 4.92; 95% CI, 2.20–11.0; *P*<0.001) were observed in patients with CAE (Table 4; Table IV and Table V in the online-only Data Supplement). Of note, 5 of 7 nonfatal MI events in patients with CAE occurred at the segment with CAE in infracted-related artery (Table 3; Table III in the online-only Data Supplement). In patients with CAE, 2 patients occurred nonfatal MI at culprit lesion (29%=2/7), whereas 13 patients (33%=13/40) occurred in patients without CAE (29% versus 33%; *P*=0.84). After adjusting for covariates, CAE was still an independent predictor of MACE (HR, 4.94; 95% CI, 2.36–10.4; *P*<0.001), cardiac death (HR, 7.97; 95% CI, 2.85–22.3; *P*<0.001), and nonfatal MI (HR, 3.90; 95% CI, 1.61–9.46; *P*=0.003; Table 4; Tables IV and V in the online-only Data Supplement). These associations were further analyzed using propensity score–matching analysis, which selected 49 patients in each of the CAE and

Table 2.   Baseline Clinical Demographics

	CAE (+) (n=51)	CAE (−) (n=1647)	*P* Value
Age, y	63±13	68±12	0.005
Male, n (%)	43 (84)	1173 (71)	0.04
Hypertension, n (%)	38 (75)	1086 (66)	0.20
Diabetes mellitus, n (%)	15 (29)	652 (40)	0.14
Dyslipidemia, n (%)	24 (47)	896 (54)	0.30
Smoking, n (%)	44 (86)	1163 (71)	0.02
BMI ≥30 kg/m², n (%)	6 (12)	67 (4)	0.008
No. of major coronary risk factors, n	2.9±1.1	2.6±1.2	0.04
STEMI, n (%)	43 (84)	1318 (80)	0.45
Killip class ≥2, n (%)	4 (8)	289 (18)	0.07
Infarct-related artery, n (%)			
Left anterior descending artery	19 (37)	791 (48)	
Left circumflex artery	10 (20)	219 (13)	
Right coronary artery	21 (41)	585 (36)	
Left main coronary artery	0 (0)	44 (3)	
Unknown	1 (2)	8 (0)	
No. of diseased vessels, n	1.8±0.7	1.7±0.8	0.30
Triple vessel disease, n (%)	10 (20)	368 (22)	0.64
Gensini score	48 (27–81)	58 (40–87)	0.03
Primary PCI, n (%)	42 (82)	1214 (74)	0.17
Peak level of CK, units/L	2109 (1089–3694)	2142 (1114–3924)	0.66
LVEF, %	49±8	45±10	0.003
Medications, n (%)			
Aspirin	50 (98)	1525 (93)	0.09
Thienopyridine	11 (22)	612 (37)	0.02
Warfarin	19 (37)	206 (13)	<0.001
DAPT	11 (22)	565 (34)	0.04
DAPT+warfarin	1 (2)	50 (3)	0.64
ACE-I/ARB	37 (73)	1075 (65)	0.25
β-Blocker	27 (53)	976 (59)	0.26
Statin	28 (55)	909 (55)	0.85
Laboratory data, mg/dL			
Total cholesterol	180±37	173±35	0.12
LDL cholesterol	118±34	112±30	0.18
HDL cholesterol	40±9	39±10	0.71
Triglyceride	127±61	124±57	0.69
CRP	0.48 (0.21–1.25)	0.49 (0.19–1.39)	0.95

Categorical variables are expressed as n (%), and continuous variables as mean±SD or median (interquartile range). Major coronary risk factors include hypertension, diabetes mellitus, dyslipidemia, and smoking. ACE-I indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; CAE, coronary artery ectasia; CK, creatine kinase; CRP, C-reactive protein; DAPT, dual antiplatelet therapy; HDL cholesterol, high-density lipoprotein cholesterol; LDL cholesterol, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; and STEMI, ST-segment–elevation myocardial infarction.

Table 3.   Summary of MACE During Follow-Up Period

	Overall (n=1698)	CAE (+) (n=51)	CAE (−) (n=1647)
MACE, n (%)	146 (9)	14 (28)	130 (8)
Cardiac death, n (%)	97 (6)	7 (14)	90 (5)
Nonfatal MI, n (%)	47 (3)	7 (14)	40 (2)
Nonfatal MI at segment with CAE, n (%)	…	5 (10)	…

Categorical variables are expressed as n (%).

CAE indicates coronary artery ectasia; MACE, major adverse cardiac events; and MI, myocardial infarction.

non-CAE groups. In this model, there were no significant differences in clinical characteristics between the 2 groups (Table VI in the online-only Data Supplement). Even in this propensity score–matched cohort, the frequency of MACE was still significantly higher in patients with CAE (HR, 8.98; 95% CI, 1.14–71.0; P=0.03).

Anticoagulation Therapy and MACE in Subjects With CAE

Of the 51 patients with CAE, 19 were treated with warfarin at the time of discharge. Of these, 8 achieved the percent time in the target therapeutic range (%TTR ≥60%). During the observation period, the occurrence of MACE was not observed in these patients. By contrast, the incidence of MACE was considerably higher in patients with %TTR <60% or in those who did not take warfarin (14 of 43; 33%; P=0.03; Figure 4).

Discussion

Although CAE is associated with abnormal vessel dilatation, disturbed coronary flow, and enhanced thrombogenicity, its association with cardiac events has not been fully characterized. Our study demonstrated that (1) 3% of patients with AMI harbored CAE, and (2) patients with CAE more frequently developed nonfatal MI and cardiac death. These findings suggest that CAE may be an important morphological characteristic of coronary artery causing cardiac events.

CAE has generally been defined according to the ratio of the size of ectatic segment to that of the adjacent normal reference segment.[4,13] However, as shown in Figure 2, this method is not applicable to patients with diffuse CAE because of the absence of normal reference segments. Krüger et al[14] developed another definition of CAE that used the mean size of each coronary segment in an age- and sex-matched cohort without heart disease as a reference value. In our study, the conventional definition of CAE was inapplicable to 17 patients (33%), who were, therefore, diagnosed using Kruger's definition. Consequently, we identified CAE in 3% of the study population, which was within the range of the previously reported prevalence (0.9%–5.3%).[4,6,13,15,16] As such, patients with diffuse CAE might be underdiagnosed when the conventional definition alone is used. The use of both definitions is more rigorous but still practical for properly identifying ectatic segments within coronary arteries.

As reported previously, patients with CAE in this study exhibited distinct clinical characteristics, such as more frequent involvement of the right coronary artery,[6,13,16,17] a lower

Table 4.   Univariate and Multivariate Analysis of Predictors for MACE

	Univariate Analysis			Multivariate Analysis			Bootstrap Validation Reliability, %
	HR	95% CI	P Value	HR	95% CI	P Value	
Age, y	1.02	1.01–1.04	0.003				10.5
Male	1.37	0.97–1.94	0.08	2.02	1.10–3.05	0.007	53.0
Smoking	0.97	0.67–1.40	0.86				3.0
BMI ≥30 kg/m²	0.54	0.17–1.70	0.29				27.5
STEMI	1.34	0.86–2.10	0.20				4.5
Killip class ≥2	5.12	3.65–7.18	<0.001				18.0
Triple vessel disease	1.89	1.33–2.68	<0.001	1.83	1.21–3.35	0.02	57.5
Gensini score	1.00	1.00–1.00	0.34				6.5
Primary PCI	1.07	0.74–1.56	0.71				7.5
Peak levels of CK, log	1.76	1.43–2.15	<0.001				18.5
LVEF, %	0.97	0.94–0.99	0.002	0.95	0.93–0.97	<0.001	87.5
CAE	3.25	1.88–5.66	<0.001	4.94	2.36–10.4	<0.001	88.5

Unadjusted hazard ratios for MACE were calculated by a univariate Cox proportional hazards model. Adjusted hazard ratios were calculated by a multivariate Cox proportional hazards model using stepwise selection with a P value of 0.05 for backward elimination. This model included the following variables: age, sex, smoking, BMI ≥30 kg/m², STEMI, Killip class ≥2, triple vessel disease, Gensini score, primary PCI, peak levels of CK, LVEF, and CAE. Variable selection in multivariable models was guided by bootstrap selection. A resampling analysis with 200 iteration was performed to identify the variables that entered into 50% of the Cox proportional hazards models to determine the independent predictor of MACE (selection criterion: P<0.10). The 95% CIs of the covariates in the final predictive models were also obtained from the bootstrapped resampling model. BMI indicates body mass index; CAE, coronary artery ectasia; CI, confidence interval; CK, creatine kinase; HR, hazard ratio; LVEF, left ventricular ejection fraction; MACE, major adverse cardiac events; PCI, percutaneous coronary intervention; and STEMI, ST-segment–elevation myocardial infarction.
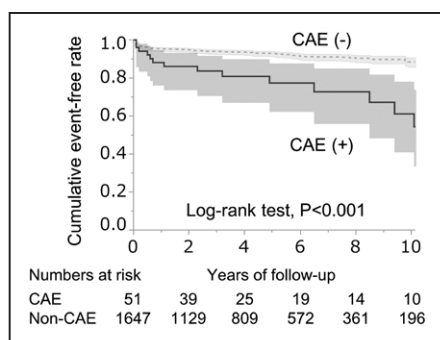
Figure 3. Comparison of long-term outcomes between patients with and without coronary artery ectasia (CAE). Kaplan–Meier curves show the 5-y rates of survival free from major adverse cardiac event (MACE). The solid and dotted black lines indicate the event-free survival curve in patients with and without CAE, respectively. The dark and light gray fields indicate 95% confidence interval of the event-free survival curve in patients with and without CAE, respectively.

frequency of stent implantation, and final Thrombolysis in Acute Myocardial Infarction flow grade 3.[16,17] Moreover, enlargement of other vessels was identified in 37% of patients with CAE. Mechanistically, both atherosclerosis and nonatherosclerotic pathogeneses[18–21] have been considered to cause CAE, and these potentially affect other systemic arteries as well. Although our CAE subjects were more likely to show clustering of coronary risks, 2 CAE cases had polycystic kidney disease or a connective tissue disorder accompanied by dilated intracranial arteries or descending thoracic aorta. As such, the influence of CAE-related pathogeneses on noncoronary vessels underscores the importance of meticulous screening of systemic arteries in CAE subjects.

The current study analyzing 1698 patients with AMI demonstrated that the presence of CAE was significantly associated with an increased risk of MACE during the 49-month observation period. Moreover, >70% of nonfatal MI in CAE subjects occurred at nonobstructive ectatic coronary segments. Mechanistically, dilatation of vessel size has been shown to slow coronary flow velocity, which increases blood viscosity and promotes coagulation.[12] This pathophysiological element might result in thrombotic occlusion of ectatic coronary arteries. Another possible mechanism is an increased level of
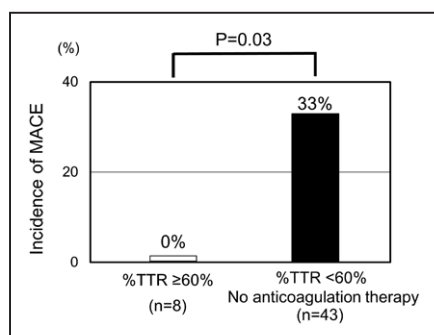

Figure 4. Anticoagulation therapy and major adverse cardiac event (MACE) in patients with coronary artery ectasia (CAE). The occurrence of MACE was compared between CAE subjects who achieved percent time in target therapeutic range (%TTR) ≥60% under warfarin use and those with either suboptimally controlled %TTR (<60%) or who did not take any anticoagulation therapy.

inflammatory cytokines, such as tumor necrosis factor-α and interleukin-1β, in CAE subjects.[8] Because these cytokines stimulate the synthesis of tissue factor, which activates the coagulation cascade, a more inflamed substrate in CAE subjects might account for an increased risk of acute coronary events.

Our findings are different from previous studies which did not identified association between CAE and cardiac events.[4,13,16,17,22–24] This might be partly because of analyzing patients with stable coronary artery disease in other published studies. Because risk of cardiac events in stable coronary artery disease subjects is lower compared with those with AMI, it may be difficult to detect significant differences in clinical outcomes between stable coronary artery disease subjects with and without CAE. Longer observational period in our study might have enabled to elucidate worse outcomes in patients with CAE.

In this study, subjects who achieved %TTR ≥60% with warfarin therapy exhibited a lower occurrence of MACE compared with those with %TTR <60% or without anticoagulation therapy. Given a significant reduction of thrombotic events in patients with optimal control of %TTR with warfarin,[9] this observation might underscore the importance of carefully monitoring the international normalization ratio in CAE subjects. Further investigation is warranted to evaluate the clinical efficacy of anticoagulation therapy in CAE subjects.

Several caveats should be noted. This was an observational study at a single center that included relatively small numbers of patients with CAE and cardiac events. Despite these limitations, a multivariate Cox proportional hazards model and propensity score–matched analyses consistently showed a significant relationship between CAE and cardiac events. The frequency of statin use was only 55% because guidelines before 2007 did not recommend early statin therapy in patients with AMI. Management based on current therapeutic guidelines might affect the clinical outcomes in our study. Anticoagulation therapy was initiated according to each physician's discretion, a procedure that is susceptible to selection bias.

In conclusion, the presence of CAE predicted the future occurrence of nonfatal MI and cardiac death in the setting of AMI. Our findings highlight CAE as a high-risk vascular phenotype requiring additional pharmacological approaches to optimally control the coagulation cascade.

Disclosures
None.

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Highlights

- Coronary artery ectasia (CAE) is characterized as abnormal vessel dilatation associated with disturbed coronary flow. Enhanced thrombogenicity and inflammatory reactions at ectatic coronary segment suggest that the presence of CAE may cause future coronary events.
- In the current analysis, CAE was observed in 3.0% of patients with acute myocardial infarction.
- The presence of CAE was associated with a higher occurrence of cardiac death and nonfatal myocardial infarction. This association was consistently observed by multivariate Cox proportional hazards model and a propensity score–matched cohort analysis.
- Despite a higher risk of cardiac events in patients with CAE, the use of anticoagulation therapy which achieved an optimal percent time in target therapeutic range, defined as ≥60%, did not experience any occurrence of major adverse cardiac events.
- Evaluation of CAE may enable to identify high-risk subjects who are more likely to develop cardiac death and nonfatal myocardial infarction. The efficacy of pharmacological intervention, such as anticoagulation therapy on cardiovascular outcomes, in patients with CAE should be investigated in future prospective randomized trial.

Coronary Artery Ectasia Predicts Future Cardiac Events in Patients With Acute Myocardial Infarction

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The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://atvb.ahajournals.org/content/37/12/2350

Materials and Methods

Study Population

The current study retrospectively analyzed 1,776 consecutive patients with de novo

acute myocardial infarction (AMI) who were hospitalized at the National Cerebral and

Cardiovascular Center from January 2001 to December 2013. Of these, 78 patients

were excluded because they did not undergo coronary angiography. The remaining

1,698 patients were included in the current analysis (Figure 1). AMI was defined as

cardiac ischemic symptoms with biomarker evidence of myocardial ischemia and

electrocardiographic changes suggestive of new ischemia (new ST-T changes or new

left bundle-branch block), or the development of pathological Q waves on

electrocardiography. [1] The current study was approved by the institutional review board

of the National Cerebral and Cardiovascular Center.

Coronary Angiography and Percutaneous Coronary Intervention

Coronary angiography was performed in multiple projections after administration of

intracoronary nitroglycerin (0.125–0.25 mg) using 5-French diagnostic catheters.

Following identification of culprit lesions on coronary angiography, primary

percutaneous coronary intervention (PCI) was conducted using 6- or 7-French

catheters. All procedural decisions, including device selection and adjunctive

pharmacotherapy, were made according to the discretion of the individual PCI operator.

In addition to the Gensini score, [2, 3] which has been validated previously, coronary

angiographic measurement were performed using computerized quantitative coronary

angiography (QAngio XA Version 7.1; Medis Medical Imaging System BV, Leiden, The

Netherlands).

Definition of CAE

In the current study, we used the conventional definition of coronary artery ectasia

(CAE), namely coronary segment dilatation of more than 1.5-fold the diameter of

adjacent normal coronary segments (Figure 2). [4, 5] In patients who did not have any

adjacent normal segment due to diffuse ectatic coronary artery, CAE was defined

according to normal reference value of corresponding segment from data in age-sex-

matched patients without heart disease (n=51) as previously reported [6] (Table I in the

Data Supplement).

Patients with CAE were further analyzed with regard to angiographic features,

specifically oscillatory and static flow. Oscillatory flow was defined as a segmental

backflow phenomenon with a whirling appearance of contrast material in the ectatic

segment. (Video I in the Data Supplement). Static flow was defined as the local

deposition of contrast material on the wall of the ectatic segment after the majority of

the contrast material had been flushed from the coronary lumen (Video II in the Data

Supplement) [6-8] Kappa coefficients were calculated to assess intra-reader and inter-

reader agreement for coronary angiographic signs in all patients who had CAE (n=51).

Two independent, experienced cardiologists evaluated coronary angiograms to

determine inter-reader agreement. With regard to intra-reader agreement, images were

re-analyzed by the same experienced cardiologist at least 4 weeks after the initial

evaluation. The intra- and inter-reader Kappa coefficients for oscillatory flow were 0.84

(95% confidence interval [CI]: 0.69–0.99) and 0.84 (95% CI: 0.64–0.97), and those for

static flow were 0.75 (95% CI: 0.54–0.96) and 0.76 (95% CI: 0.56–0.96), respectively.

Anticoagulation Therapy in Patients with CAE

The use of anticoagulation therapy was investigated in patients with CAE. Warfarin was

commenced according to each physician's discretion. The optimal therapeutic range of

the international normalized ratio (INR) was defined as between 1.6 and 2.5 according

to data from a recently published study. [9, 10] The percent time in the target therapeutic

range (%TTR) was calculated in patients with CAE receiving anticoagulation therapy

using the Rosendaal method. [11] This method utilizes linear interpolation to estimate

time within a therapeutic INR range. In each individual, time within the therapeutic INR

range was added and then divided by the total time of observation.

Clinical Outcome

The primary outcome was a composite of major adverse cardiac events (MACE), specifically cardiac death and non-fatal myocardial infarction (MI). The secondary outcome was the occurrence of each component of MACE (cardiac death or non-fatal MI). These outcomes were determined through medical record review, and when necessary, through a questionnaire by mail and telephone follow-up.

Statistical Analysis

Continuous variables were expressed as the mean ± standard deviation and compared using the t test if data were normally distributed. Non-normally distributed continuous data were summarized as the median (interquartile range) and compared using the Mann-Whitney test. Categorical variables were compared using the Fisher exact test or the Chi-square test as appropriate. The Kaplan-Meier method was used to estimate survival curves for primary and secondary outcomes, and the log-rank test was used to assess differences between patients with and without CAE. Stepwise selection for a multivariate Cox hazards model with a P value of 0.05 for backward elimination was used to identify independent predictors of primary and secondary outcomes. This analysis included the following variables: age, sex, smoking, body mass index (BMI) ≥30 kg/m^2, ST-elevation MI, Killip class ≥2, triple vessel disease, Gensini score,

primary percutaneous coronary intervention (PCI), peak levels of creatine kinase, left

ventricular ejection fraction (LVEF) and CAE.. Variable selection in multivariable models

was guided by bootstrap selection with the correction of bias due to small sample size

by Harrell et al. [12] A resampling analysis with 200 iteration was performed to identify the

variables that entered into 50% of the Cox proportional hazards models to determine

the independent predictor of MACE, cardiac death, and non-fatal MI (selection criterion:

P<0.10) using mfp commands of STATA. [13] The 95% CI of the covariates in the final

predictive models were also obtained from the bootstrapped resampling model. The

propensity score was estimated using probit regression models,[14, 15] with MACE as the

outcome and baseline clinical demographics as predictors (covariates: age, sex,

smoking, BMI ≥30 kg/m^2, and LVEF). Patients with and without CAE were matched by

propensity score on a 1:1 basis by using the nearest-neighbour matching method

within a calliper of 0.01 of the propensity score with the psmatch2 procedure in the

STATA program. [16] All P values <0.05 were considered statistically significant. All

analyses were performed with SPSS (SPSS Japan, Tokyo, Japan) or STATA 13

(StataCorp, College Station, TX).

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SUPPLEMENTAL MATERIAL

Supplemental Figure I-A.

Supplemental Figure I-B.

Kaplan-Meier curve showing cumulative event-free rate over years of follow-up comparing CAE (-) and CAE (+) groups. Log-rank test, P<0.001.

Numbers at risk	0	2	4	6	8	10
CAE	51	39	25	19	14	10
Non-CAE	1647	1129	809	572	361	196
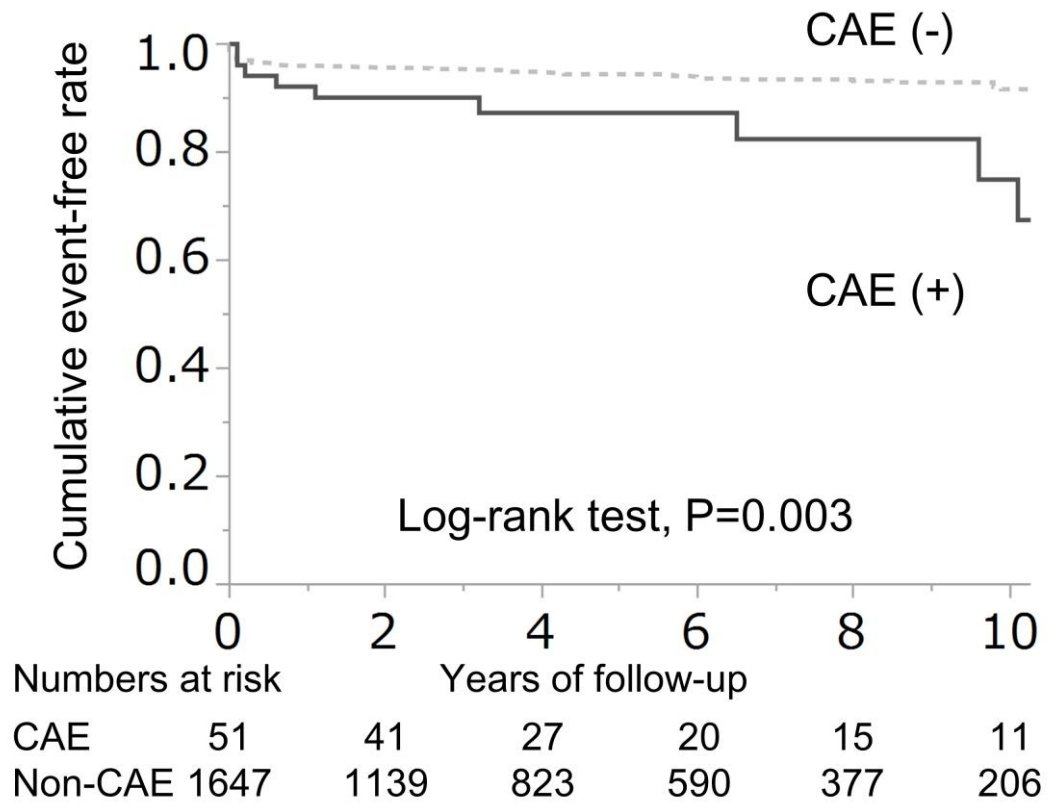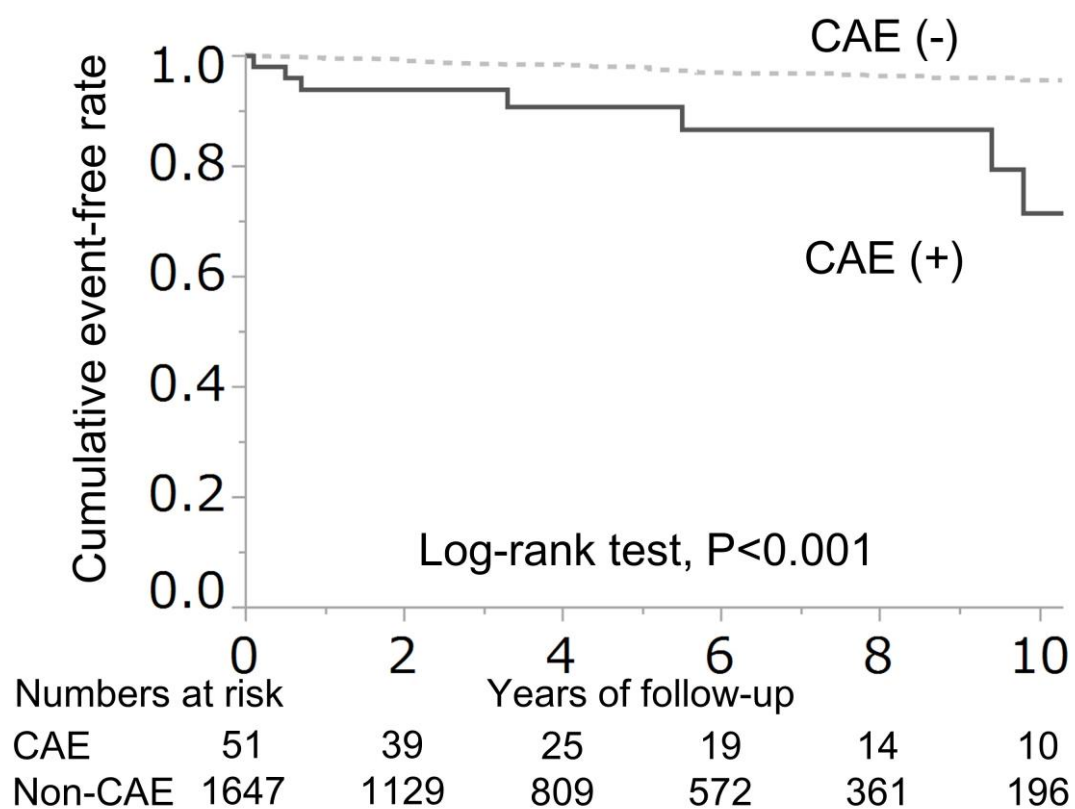
Supplemental Figure Legends

Supplemental Figure I. Comparison of Long-term Outcomes between Patients with and without CAE.

Kaplan-Meier curves show the 5-year rates of survival free from cardiac death (A), and non-fatal MI (B). The solid and dotted black lines indicate the event-free survival curve in patients with and without CAE, respectively.

CAE = coronary artery ectasia, CI = confidence interval, MI = myocardial infarction.

Supplemental Table I. Coronary Artery Diameter in Age-Sex-Matched Subjects without Heart Disease

	Control subjects (n=51)
	diameter (mm)
Right coronary artery	
Proximal	3.8±0.6
Middle	3.5±0.6
Distal	3.3±0.5
Right atrioventricular	1.9±0.4
Posterior descending	1.7±0.3
Left anterior descending artery	
Proximal	3.4±0.6
Middle	2.6±0.5
Distal	1.7±0.3
Left circumflex artery	
Proximal	3.0±0.7
Middle	1.7±0.8
Obtuse marginal	1.4±0.4
Left main trunk	4.2±0.7

Supplemental Table II. Comparison of Lesion Characteristics and PCI Procedures

	CAE (+)	CAE (-)	
	(n=42)	(n=1214)	P value
Number of treated vessels, n	1.1±0.4	1.1±0.3	0.46
Lesion characteristics			
Calcification, n (%)	7 (17)	327 (27)	0.17
Thrombus, n (%)	10 (24)	208 (17)	0.11
Lesion length, mm	10±10	11±9	0.56
Stent implantation, n (%)	26 (62)	1106 (91)	<0.001
Bare metal stent, n (%)	23 (55)	971 (80)	<0.001
Drug-eluting stent, n (%)	3 (7)	135 (11)	0.38
Number of stents used, n	1.2±0.8	1.2±0.8	0.93
Non-stent PCI, n (%)	16 (38)	108 (9)	<0.001
POBA without stent use, n (%)	13 (31)	88 (7)	<0.001
Thrombectomy, n (%)	13 (31)	30 (2)	<0.001
Mechanical circulatory support			
Intra-aortic balloon pumping, n (%)	12 (29)	167 (14)	0.01
Final TIMI flow grade 3, n (%)	19 (45)	1070 (88)	<0.001

Staged PCI, n (%)	8	(19)	464 (38)	0.05

Categorical variables are expressed as n (%), and continuous variables as mean±SD.

CAE = coronary artery ectasia, PCI = percutaneous coronary intervention, POBA = plain

old balloon angioplasty, SD = standard deviation, TIMI = thrombolysis in acute myocardial

infarction.

Supplemental Table III. Association of CAE Location and MACE

	CAE location		
	Infarct-related artery (n=29)	Non-infarct related artery (n=22)	P value
MACE, n (%)	9 (31)	5 (23)	0.51
Cardiac death, n (%)	4 (14)	3 (14)	0.99
Non-fatal MI, n (%)	5 (17)	2 (9)	0.39
Non-fatal MI at segment with CAE, n (%)	5 (17)	0 (0)	0.01

Categorical variables are expressed as n (%). CAE = coronary artery ectasia, MACE =

major adverse cardiac events, MI = myocardial infarction

Supplemental Table IV. Univariate and multivariate analysis of predictors for cardiac death

	Univariate Analysis			Multivariate			Bootstrap validation
	HR	95% CI	P value	HR	95% CI	P value	reliability, %
Age, y	1.04	(1.02-1.06)	<0.001				
Male	1.74	(1.18-2.56)	0.005	4.16	(2.21-7.85)	<0.001	55.0
Smoking	0.87	(0.57-1.32)	0.51				2.5
BMI ≥ 30kg/m^2	0.72	(0.23-2.26)	0.57				28.0
STEMI	1.47	(0.86-2.50)	0.16				4.5
Killip class ≥2	9.03	(6.05-13.5)	<0.001				19.0
Triple vessel disease	2.13	(1.44-3.16)	<0.001	2.33	(1.22-4.43)	0.01	58.5
Gensini score	1.00	(1.00-1.00)	0.52				6.0

Primary PCI	1.14	(0.73-1.77)	0.56				7.5
Peak levels of CK, log	2.15	(1.70-2.71)	<0.001				20.0
LVEF, %	0.94	(0.91-0.96)	<0.001	0.91	(0.88-0.95)	<0.001	87.5
CAE	2.71	(1.37-5.37)	0.004	7.97	(2.85-22.3)	<0.001	88.5

Unadjusted hazard ratios for cardiac death were calculated by a univariate Cox proportional hazards model. Adjusted hazard ratios were calculated by a multivariate Cox proportional hazards model using stepwise selection with a P value of 0.05 for backward elimination. This model included the following variables: age, sex, smoking, BMI ≥30 kg/m^2, STEMI, Killip class ≥2, triple vessel disease, Gensini score, primary PCI, peak levels of CK, LVEF and CAE. Variable selection in multivariable models was guided by bootstrap selection. A resampling analysis with 1000 iteration was performed to identify the variables that entered into 50% of the Cox proportional hazards models to determine the independent predictor of cardiac death (selection criterion: P<0.10). The 95% CI of the covariates in the final predictive models were also obtained from the bootstrapped resampling model.

BMI = body mass index, CAE = coronary artery ectasia, CI = confidence interval, CK = creatine kinase, HR = hazard ratio, LVEF = left

ventricular ejection fraction, PCI = percutaneous coronary intervention, STEMI = ST-segment elevation myocardial infarction

Supplemental Table V. Univariate and multivariate analysis of predictors for non-fatal MI

	Univariate Analysis			Multivariate			Bootstrap validation
	HR	95% CI	P value	HR	95% CI	P value	reliability, %
Age, y	0.99	(0.96-1.01)	0.28	0.96	(0.94-0.99)	0.02	67.0
Male	0.84	(0.43-1.64)	0.61				8.5
Smoking	1.01	(0.52-1.94)	0.98				6.0
BMI ≥ 30kg/m^2	0		0.99				31.0
STEMI	1.13	(0.55-2.34)	0.74				19.5
Killip class ≥2	0.65	(0.23-1.80)	0.41				43.0
Triple vessel disease	1.59	(0.85-2.96)	0.15	2.16	(1.04-4.49)	0.04	53.5
Gensini score	1.00	(0.99-1.00)	0.29				3.0

Primary PCI	1.01	(0.54-1.89)	0.99				6.0
Peak levels of CK, log	0.91	(0.65-1.29)	0.61				37.0
LVEF, %	1.01	(0.98-1.05)	0.42				6.0
CAE	4.92	(2.20-11.0)	<0.001	3.90	(1.61-9.46)	0.003	62.0

Unadjusted hazard ratios for non-fatal MI were calculated by a univariate Cox proportional hazards model. Adjusted hazard ratios were calculated by a multivariate Cox proportional hazards model using stepwise selection with a P value of 0.05 for backward elimination.

This model included the following variables: age, sex, smoking, BMI ≥30 kg/m^2, STEMI, Killip class ≥2, triple vessel disease, Gensini score, primary PCI, peak levels of CK, LVEF and CAE. Variable selection in multivariable models was guided by bootstrap selection. A resampling analysis with 1000 iteration was performed to identify the variables that entered into 50% of the Cox proportional hazards models to determine the independent predictor of non-fatal MI (selection criterion: P<0.10). The 95% CI of the covariates in the final predictive models were also obtained from the bootstrapped resampling model.

BMI = body mass index, CAE = coronary artery ectasia, CI = confidence interval, CK = creatine kinase, HR = hazard ratio, LVEF = left

ventricular ejection fraction, MI = myocardial infarction, PCI = percutaneous coronary intervention, STEMI = ST-segment elevation

myocardial infarction

Supplemental Table VI. Baseline Demographics of Patients after Propensity Score-matching

	Propensity Score-Matched Groups		
	CAE (+) (n=49)	CAE (-) (n=49)	P value
Age, years	62±12	62±13	0.84
Male, n (%)	43 (88)	43 (88)	1.00
Hypertension, n (%)	36 (73)	28 (57)	0.09
Diabetes mellitus, n (%)	15 (31)	17 (35)	0.67
Dyslipidemia, n (%)	24 (49)	26 (53)	0.69
Smoking, n (%)	43 (88)	45 (92)	0.51
BMI ≥30 kg/m^2, n (%)	6 (12)	7 (14)	0.77
STEMI, n (%)	41 (84)	43 (88)	0.56
Killip class ≥2, n (%)	2 (4)	6 (12)	0.14
Triple vessel disease, n (%)	10 (20)	5 (10)	0.16
Gensini score	47 (24-80)	47 (38-69)	0.52
Primary PCI, n (%)	41 (84)	34 (69)	0.10

Peak level of CK, units/L	2109 (1055-3590)	2039 (1120-4281)	0.57
LVEF, %	49±8	49±9	0.71

The propensity score was estimated using probit regression models, with MACE as the outcome and baseline clinical demographics as predictors (covariates: age, sex, smoking, BMI ≥30 kg/m^2, left ventricular ejection fraction). Categorical variables are expressed as n (%), and continuous variables as mean±SD or median (interquartile range). BMI = body mass index, CAE = coronary artery ectasia, CK = creatine kinase, LVEF = left ventricular ejection fraction, PCI = percutaneous coronary intervention, SD = standard deviation, STEMI = ST-segment elevation myocardial infarction.

Supplemental Video Legends

Supplemental Video I. Representative Patient with CAE who Exhibited Oscillatory Flow

A 65-year-old man developed acute posterolateral myocardial infarction. Left coronary angiography identified total occlusion of the middle segment of the left circumflex artery. In addition, an extremely dilated left anterior descending artery was observed. This ectatic coronary segment was accompanied by oscillatory flow, manifesting as a segmental backflow phenomenon with a whirling appearance of contrast material within the ectatic segment.

CAE = coronary artery ectasia

Supplemental Video II. Representative Patient with CAE who Exhibited Static Flow.

A 67-year-old man was hospitalized due to acute inferior myocardial infarction. Coronary angiography revealed ectasia of the proximal coronary segment in the left anterior descending artery. This segment exhibited static flow that was characterized by local deposition of contrast material after most of the contrast material had been flushed from the coronary lumen.

CAE = coronary artery ectasia