# Prevalence and long-term outcomes of patients with coronary artery ectasia presenting with acute myocardial infarction

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# Prevalence and Long-term Outcomes of Patients with Coronary Artery Ectasia Presenting with Acute Myocardial Infarction

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**Coronary artery ectasia (CAE) is described in 5% of patients undergoing coronary angiography. Previous studies have shown controversial results regarding the prognostic impact of CAE. The prevalence and prognostic value of CAE in patients with acute myocardial infarction (AMI) remain unknown. In 4788 patients presenting with AMI referred for coronary angiography the presence of CAE (defined as dilation of a coronary segment with a diameter ≥1.5 times of the adjacent normal segment) was confirmed in 174 (3.6%) patients (age 62 ± 12 years; 81% male), and was present in the culprit vessel in 79.9%. Multivessel CAE was frequent (67%). CAE patients were more frequently male, had high thrombus burden and were treated more often with thrombectomy and less often was stent implantation. Markis I was the most frequent angiographic phenotype (43%). During a median follow-up of 4 years (1-7), 1243 patients (26%) experienced a major adverse cardiovascular event (MACE): 282 (6%) died from a cardiac cause, 358 (8%) had a myocardial infarction, 945 (20%) underwent coronary revascularization and 58 (1%) presented with a stroke. Patients with CAE showed higher rates of MACE as compared to those without CAE (36.8% versus 25.6%; p <0.001). On multivariable analysis, CAE was associated with MACE (HR 1.597; 95% CI 1.238-2.060; p <0.001) after adjusting for risk factors, type of AMI and number of narrowed coronary arteries. In conclusion, the prevalence of CAE in patients presenting with AMI is relatively low but was independently associated with an increased risk of MACE at follow-up.**  **(Am J Cardiol 2021;156:9−15)**

Coronary artery ectasia (CAE) is defined as a dilation of a coronary artery segment with at least 1.5 times the diameter of the adjacent normal segments.[1] The prevalence of CAE in patients undergoing coronary angiography ranges from 0.3% to 5.3% .[2] CAE may be detected as an incidental finding in asymptomatic patients during coronary angiography (i.e. prior to valve surgery or atrial fibrillation ablation) or in the context of an acute myocardial infarction (AMI).[3] Clinical symptoms could be caused by the presence of concomitant obstructive atherosclerotic disease or distal embolization due to local thrombosis in the lumen of a large aneurysmatic coronary segment.[4] In patients presenting with AMI, the presence of CAE may influence the procedural success and the long-term outcome. However, current knowledge is based on small sample size studies which showed contradictory results.[5-9] Accordingly, we aimed at: 1) assessing the prevalence of CAE in a large cohort of patients presenting with AMI, 2) defining the main phenotypical angiographic characteristics of patients with and without CAE and 3) at investigating the long-term prognostic impact of CAE.

## Methods

Consecutive patients presenting with AMI at the Leiden University Medical Center (Leiden, the Netherlands) between February 2004 to October 2015, who underwent acute invasive coronary angiography, were included in the analysis. Patients with previous history of coronary artery bypass grafting were excluded. Invasive coronary angiography was performed in a standard fashion and revascularization of the culprit lesion was performed according to contemporary recommendations. Patients were subsequently treated according to the institutional protocol,[10] remaining hospitalized for at least 48 hours. Baseline demographic and clinical data, including cardiovascular risk factors and medications at discharge, were retrospectively collected from the Departmental Cardiology Information System (EPD-Vision: Leiden University Medical Center, Leiden, The Netherlands). This retrospective study of clinically acquired data was approved by the Institutional Review Board and the need for patient written informed consent was waived.

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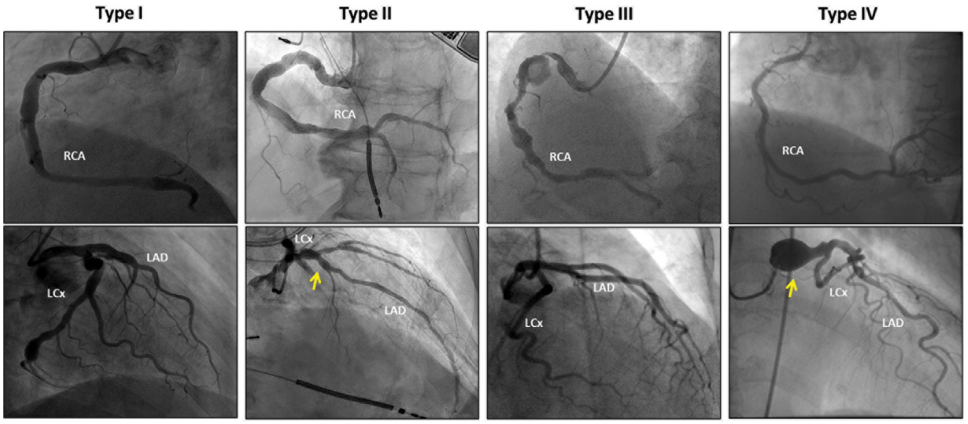


Figure 1. Angiographic characterization of CAE distribution according to the Markis classification. Type I: diffuse CAE in 2 or 3 coronary vessels. In these case, all 3 vessels present diffuse CAE. Type II: diffuse CAE in one coronary vessel (RCA) and localized CAE in another vessel (proximal LAD, arrow). Type III: diffuse CAE in only 1 coronary vessel (RCA, arrows). Type IV: localized or segmental CAE (in this case, massive dilatation of the LMCA, arrow). CAE = coronary artery ectasia; LAD = left anterior descending; LCx = left circumflex; LMCA = left main coronary artery; RCA = right coronary artery.

CAE was defined as a dilation of a coronary artery segment with a diameter ≥1.5 times of the adjacent normal segment. Patients with CAE in any of the coronary vessels during index coronary angiography were identified. The study cohort was divided into two groups, according to the presence or absence of CAE. Coronary angiograms obtained during the index procedure were retrospectively evaluated by two independent interventional cardiologists blinded to the clinical outcomes. The angiographic anatomical distribution of CAE was categorized according to the Markis classification[11]: type I was defined as the presence of diffuse CAE in 2 or 3 coronary vessels; type II as diffuse CAE in one coronary vessel and localized CAE in another vessel; type III as diffuse CAE in only one coronary vessel and type IV as localized or segmental CAE (Figure 1).

Multivessel disease was defined by the presence of a coronary stenosis >50% in ≥2 major coronary arteries. Coronary artery flow was evaluated by using the Thrombolysis In Myocardial Infarction (TIMI) frame count method.[12] Thrombus burden was graded from 0 to 5 according to the TIMI-thrombus scale.[13] High thrombus burden was defined as a TIMI-thrombus scale ≥4. Angiographic success was defined as final TIMI 3 distal flow with less than 20% of vessel stenosis and no immediate mechanical complications. No-reflow phenomenon was defined as TIMI flow ≤2 at the end of the procedure without angiographic evidence of mechanical vessel obstruction.[14]

Patients were followed-up according to the institutional guideline-based care-track protocol.[10] The primary endpoint was composite of major adverse cardiovascular events (MACE) which included cardiac death, myocardial infarction, stroke and repeated coronary revascularization, including percutaneous coronary intervention or coronary artery bypass grafting. Secondary endpoints were the individual components of the composite outcome. Deaths were considered to be attributable to a cardiac cause unless a noncardiac death could be confirmed. Myocardial infarction was defined as an increase of cardiac troponin with at least 1 value above the 99th percentile upper reference limit and ischemic symptoms and/or new or presumed new ST−segment, T−wave changes or new left bundle branch block.[15] Stroke was defined as any cerebrovascular event (intracranial hemorrhage or non-hemorrhagic stroke) meeting the following criteria: 1) rapid onset of neurological deficit; 2) duration ≥24 hours or <24 hours if therapeutic intervention, neuro-imaging or death; 3) absence of non-stroke cause; 4) confirmation by neurologist/neurosurgeon, neuro-imaging or lumbar puncture. Medical records review and survival status information were obtained through the hospital information systems (EPD-Vision and EZIS; Leiden University Medical Centre, Leiden, The Netherlands).

Normally distributed continuous variables are presented as mean ± standard deviation while non-normally distributed continuous variables are presented as median with interquartile range. Categorical data are presented as numbers and percentages. Unpaired Student's t-test was used for comparison of normally distributed continuous variables, Mann-Whitney U test for non-normally distributed continuous variables, and chi-square test for categorical data. The cumulative events were calculated using the Kaplan−Meier curves and comparison between groups was performed using the log-rank test. Uni- and multivariable Cox regression analyses were performed to identify independent demographic, clinical and angiographic variables associated with MACE. The hazard ratio (HR) and 95% confidence interval are presented. All statistical tests were two-sided, and a P-value <0.05 was considered statistically significant. Data analyses were performed using SPSS version 25.0 software (IBM SPSS Statistics for Windows. Armonk, NY, USA)

## Results

Among 4788 patients (62 ±12 years old, 74% men), CAE was observed in 174 (3.6%) patients. Baseline characteristics of patients with and without CAE are shown in Table 1. Patients with CAE were more frequently men as compared to patients without CAE. There were no other significant differences in clinical variables. Angiographic and procedural data are summarized in Table 2. Regarding distribution of the culprit vessels, the right coronary artery (RCA) was the most frequent culprit vessel in patients with

Table 1
Baseline clinical characteristics.

| Variable | Total population(n = 4788) | Coronary Artery Ectasia | | p Value |
|---|---|---|---|---|
| | | Yes (n = 174) | No (n = 4614) | |
| Age (years) | 63 ± 13 | 62 ± 12 | 63 ± 12 | 0.766 |
| Men | 3540 (73.9%) | 142 (81.6%) | 3398 (73.6%) | 0.019 |
| Diabetes mellitus | 620 (12.9%) | 12 (6.9%) | 608 (13.2%) | 0.052 |
| Hypertension | 1838 (38.4%) | 58 (33.3%) | 1780 (38.6%) | 0.316 |
| Dyslipidemia | 2889 (60.3%) | 112 (64.4%) | 2777 (60.2%) | 0.243 |
| Smoker | 2500 (50.2%) | 105 (60.3%) | 2395 (51.9%) | 0.089 |
| BMI ($kg/m^2$) | 27 ± 9 | 27 ± 9 | 28 ± 12 | 0.130 |
| Previous MI | 417 (8.9%) | 19 (11.1%) | 398 (8.9%) | 0.394 |
| Previous PCI | 361 (7.5%) | 13 (7.6%) | 348 (7.5%) | 0.127 |
| STEMI at presentation | 4373 (91.3%) | 158 (90.8%) | 4215 (91.4%) | 0.801 |
| Killip class >2 | 176 (3.7%) | 2 (1.1%) | 174 (3.8%) | 0.071 |
| LVEF | 47 ± 9 | 48 ± 9 | 47 ± 9 | 0.832 |
| *Laboratory data* | | | | |
| Total cholesterol (mg/dl) | 205 ± 47 | 203 ± 45 | 205 ± 48 | 0.617 |
| LDL-cholesterol (mg/dL) | 133 ± 43 | 132 ± 41 | 133 ± 43 | 0.785 |
| Peak CK (units/L) | 1392 (539-2149) | 1494 (506-2099) | 1389 (541-2151) | 0.854 |
| Creatinine ($\mu$mol /L) | 80 (68-89) | 79 (68-88) | 80 (68-89) | 0.488 |
| CRP (mg/L) | 3 (3-11) | 3 (3-11) | 4 (3-11) | 0.311 |
| *Medication at discharge* | | | | |
| Aspirin | 4419 (92.3%) | 161 (92.5%) | 4258 (92.3%) | 0.906 |
| DAPT | 4415 (92.2%) | 161 (92.5%) | 4254 (92.2%) | 0.873 |
| Oral anticoagulation | 149 (3.2%) | 8 (4.7%) | 141 (3.2%) | 0.254 |
| DAPT + oral anticoagulation | 116 (2.4%) | 5 (2.9%) | 111 (2.4%) | 0.694 |
| ACE-I/ARB | 4276 (92.9%) | 156 (92.9%) | 4120 (92.9%) | 0.967 |
| $\beta$-Blockers | 4174 (90.7%) | 153 (91.1%) | 4021 (90.7%) | 0.873 |
| Statins | 4435 (96.4%) | 163 (97.0%) | 4272 (96.4%) | 0.655 |

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; BMI, body mass index; CK=creatine kinase; CRP, C-reactive protein; DAPT, dual antiplatelet therapy; LDL, low-density lipoprotein;; LVEF, left ventricular ejection function; MI, myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; TIMI, thrombolysis in myocardial infarction.

Table 2
Angiographic and procedural characteristics.

| Variable | Total population(n = 4778) | Coronary Artery Ectasia | | p Value |
|---|---|---|---|---|
| | | Yes (n = 174) | No (n = 4614) | |
| Culprit lesion location, | | | | 0.310 |
| Left anterior descending | 1943 (40.6%) | 57 (32.8%) | 1886 (40.9%) | 0.032 |
| Left circumflex | 732 (15.3%) | 29 (16.7%) | 703 (15.2%) | 0.607 |
| Right | 1711 (35.7%) | 72 (41.4%) | 1639 (35.5%) | 0.114 |
| Left main | 65 (1.4%) | 3 (1.7%) | 62 (1.3%) | 0.570 |
| No. of narrowed coronary arteries | 2 (1-2) | 2 (1-3) | 2 (1-2) | 0.115 |
| Three-vessel disease | 1178 (24.6%) | 48 (27.6%) | 1130 (24.5%) | 0.352 |
| Mechanical hemodynamic support | 119 (2.5%) | 3 (1.7%) | 116 (2.5%) | 0.511 |
| Balloon pre-dilatation | 3757 (84.0%) | 114 (82.8%) | 3610 (78.2%) | 0.056 |
| Balloon post-dilatation | 1624 (36.5%) | 66 (37.9%) | 1558 (33.8%) | 0.208 |
| Thrombectomy | 461 (9.6%) | 33 (20.5%) | 428 (9.3%) | <0.001 |
| Stent implanted | 4246 (93.0%) | 146 (84.4%) | 4100 (93.3%) | <0.001 |
| No. of stents | 1 (1-2) | 1 (1-2) | 1 (1-2) | 0.830 |
| Stent diameter (mm) | 3.5 (3.0-3.5) | 3.5 (3.0-4.0) | 3.0 (3.0-3.5) | <0.001 |
| Total Stent length (mm) | 23 (16-34) | 23 (16-36) | 23 (16-34) | 0.884 |
| Initial TIMI flow | | | | |
| 0/1 | 3012 (68.4%) | 121 (76.1%) | 2891 (68.1%) | 0.034 |
| 2 | 603 (13.7%) | 17 (10.7%) | 586 (13.8%) | 0.261 |
| 3 | 787 (17.9%) | 21 (13.2%) | 766 (18.1%) | 0.117 |
| Final TIMI flow | | | | |
| 0/1 | 99 (2.3%) | 5 (3.2%) | 94 (2.2%) | 0.418 |
| 2 | 200 (4.6%) | 11 (7.1%) | 189 (4.5%) | 0.130 |
| 3 | 4054 (93.1%) | 139 (89.7%) | 3915 (93.3%) | 0.083 |
| Final TIMI flow < 3 | 299 (6.9%) | 16 (10.3%) | 283 (6.7%) | 0.083 |

TIMI, Thrombolysis in Myocardial Infarction.

Table 3
Anatomical angiographic features of patients with coronary artery ectasia.

| Variable | Coronary Artery Ectasia (n = 174) |
|---|---|
| CAE affected coronary artery, n (%) | |
| Right | 138 (79.3%) |
| Left anterior descending | 115 (66.1%) |
| Left circumflex | 90 (51.7%) |
| Left main | 55 (31.6%) |
| Diagonal branches | 15 (8.6%) |
| Obtuse marginal branches | 35 (20.1%) |
| Posterior descending | 64 (36.8%) |
| CAE in infarct-related artery | 139 (79.9%) |
| CAE single vessel involvement | 57 (32.8%) |
| CAE multivessel involvement | 117 (67.2%) |
| Type of CAE according to Markis classification | |
| I | 75 (43.1%) |
| II | 24 (13.8%) |
| III | 45 (25.9%) |
| IV | 30 (17.2%) |
| Large thrombus burden | 160 (91.9%) |

CAE, coronary artery ectasia.

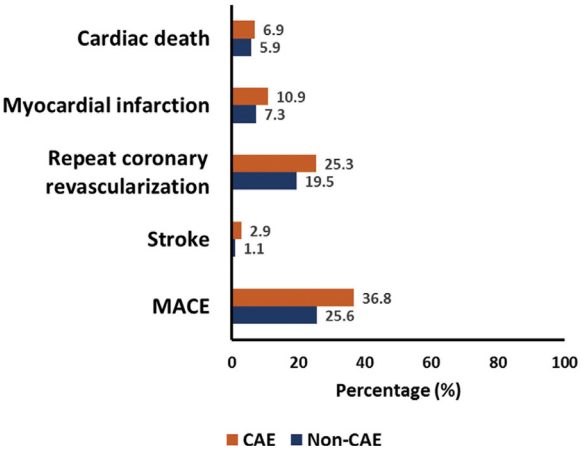


Figure 2. Distribution of individual MACE in patients with and without CAE during follow-up. CAE = coronary artery ectasia; MACE = major adverse cardiovascular events.

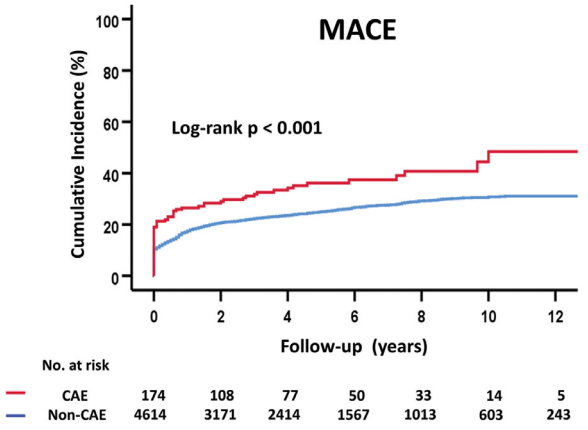


Figure 3. Kaplan-Meier survival curves of cumulative MACE incidence in patients with CAE (red) versus patients without CAE (blue). CAE = coronary artery ectasia; MACE = major adverse cardiovascular event.

CAE, whereas in patients without CAE, the left anterior descending (LAD) was the most frequent. Thrombectomy was more often used in patients with CAE whereas the rate of stent implantation in the culprit lesion was lower than in those without. Furthermore, patients with CAE were treated with stents of larger diameters as compared to patients without CAE.

The specific angiographic characteristics of patients with CAE are summarized in Table 3. CAE was predominantly observed in the RCA followed by the LAD, left circumflex artery and left main coronary artery. CAE was present in the culprit vessel in the vast majority of patients, being the presence of multivessel CAE frequently observed. Large thrombus burden was present in 92% of patients. CAE extension was assessed according to the classification proposed by Markis et al:[11] 43% patients were classified as type I (diffuse CAE in 2 or 3 coronary vessels); 14% as type II (diffuse CAE in 1 vessel and localized CAE in another vessel); 26% as type III (diffuse CAE in only 1 vessel) and 17% as type IV (localized or segmental CAE).

During a median follow-up of 4 years (IQR 1-7 years), 1243 patients (26%) presented with MACE. The individual components of MACE occurred as follows: 282 patients (6%) died from a cardiac cause, 358 (8%) had a myocardial infarction, 945 (20%) underwent coronary revascularization and 58 (1%) suffered a stroke. The distribution of events in patients with and without CAE is presented in Figure 2. Survival analysis showed higher rates of MACE in patients with CAE compared with those without CAE (Figure 3). There were no significant differences between groups regarding cardiac death rate and myocardial infarction. There were significant differences between groups in terms of any repeat revascularization and stroke, as displayed in Figure 4.

To investigate the association between CAE and the occurrence of MACE, uni- and multivariable Cox regression analyses were performed (Table 4). On univariable analysis, age, diabetes, previous myocardial infarction, ST-segment elevation myocardial infarction at presentation, three-vessel coronary artery disease, final TIMI flow <3, peak creatine kinase, creatinine, Killip class >2 and CAE showed a significant association with MACE. On multivariable analysis, diabetes, previous MI, STEMI at presentation, three-vessel coronary artery disease, TIMI flow <3, Killip class >2 and CAE remained independently associated with MACE.

## Discussion

The prevalence of CAE in a large cohort of patients presenting with AMI was 3.6%. Patients with CAE presented with ectasia affecting 2 or more coronary arteries in 67%. CAE in the culprit vessel was found in 80% of patients, representing 3.2% of the total study population. Patients with CAE presenting with AMI had an increased rate of MACE at 4-years follow-up compared with those without CAE.

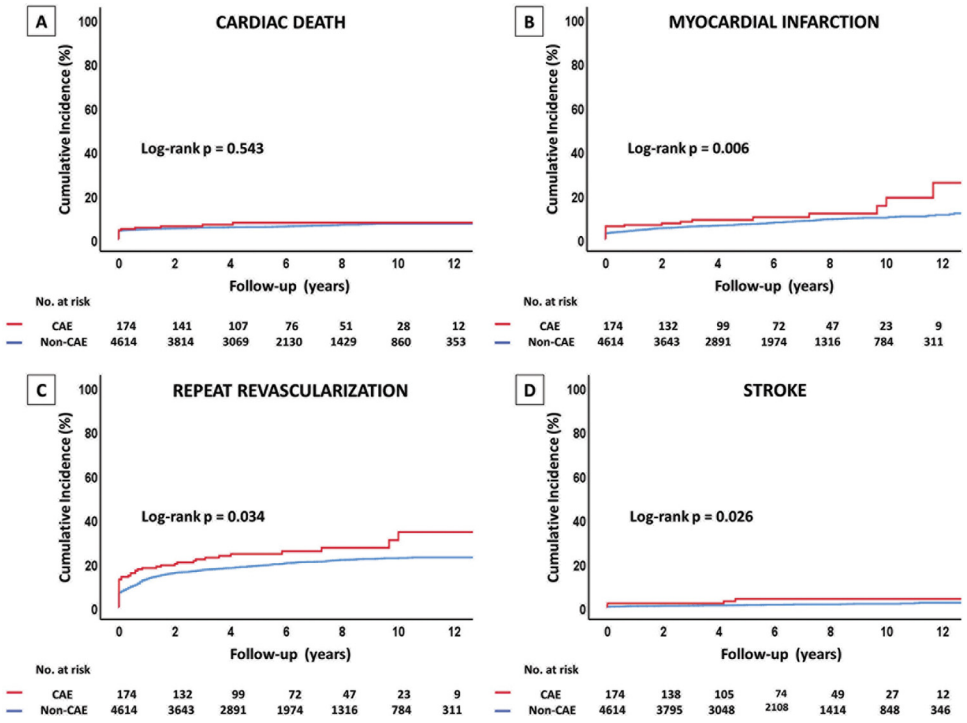


Figure 4. Kaplan-Meier survival curves of cumulative incidence of (A) cardiac death; (B) MI; (C) repeat revascularization and (D) stroke in patients with CAE (red line) versus patients without CAE (blue line). CAE = coronary artery ectasia; MI = myocardial infarction.

This association was independent from cardiovascular risk factors, type of AMI and number of diseased vessels.

The pathogenesis of CAE has not been fully elucidated, and multiple pathophysiological mechanisms have been involved.[8] Given the frequent coexistence of CAE with obstructive CAD (up to 85%), it has been suggested that CAE and atherosclerosis share a similar pathogenesis.[2,16,17] In addition, several systemic inflammatory disorders have been related to CAE, such as Kawasaki disease, Wegener's granulomatosis, lupus and rheumatic fever.[18,19] CAE has also been linked with genetic susceptibility, infections, drug use, trauma and implantation of drug-coated stents.[8]

Previous studies have reported a prevalence of CAE ranging from 0.3% to 5.3% in patients undergoing coronary angiography,[2,5,16,20] reaching up to 11% in a study including 250 patients with ischemic heart disease from India.[21] An analysis of the Coronary Artery Surgery Study (CASS) registry, which enrolled 20087 patients who underwent coronary angiography, CAE was found in 4.9%.[2] However, there are limited data regarding the prevalence of CAE in

Table 4
Univariable and multivariable analysis to evaluate the association between CAE and MACE.

| Variable | Univariable analysis | | Multivariable analysis | |
|---|---|---|---|---|
| | HR 95% CI | p Value | HR 95% CI | p Value |
| Age, (per one year increase) | 1.010 (1.006-1.015) | <0.001 | 1.001 (0.996-1.006) | 0.661 |
| Male sex | 1.097 (0.963-1.249) | 0.163 | - | |
| BMI, (per one unit increase) | 1.001 (0.994-1.008) | 0.744 | - | |
| Diabetes mellitus | 1.480 (1.274-1.720) | <0.001 | 1.330 (1.139-1.553) | <0.001 |
| Hypertension | 1.049 (0.935-1.177) | 0.413 | - | |
| Smoking history | 1.034 (0.927-1.152) | 0.551 | - | |
| Previous MI | 1.288 (1.097-1.512) | 0.002 | 0.993 (0.839-1.174) | 0.930 |
| STEMI at presentation | 2.834 (2.090-3.843) | <0.001 | 2.827 (2.078-3.846) | <0.001 |
| Three-vessel coronary artery disease | 2.443 (2.180-2.738) | <0.001 | 2.336 (2.069-2.637) | <0.001 |
| Final TIMI flow < 3 | 1.911 (1.603-2.279) | <0.001 | 1.695 (1.414-2.031) | <0.001 |
| Peak CK, units/L, (per 1000 unit increase) | 1.013 (1.008-1.019) | <0.001 | 1.004 (0.997-1.010) | 0.254 |
| Creatinine, (per one unit increase) | 1.002 (1.001-1.003) | <0.001 | 1.001 (1.000-1.002) | 0.063 |
| Killip class > 2 | 3.661 (3.007-4.457) | <0.001 | 2.326 (1.876-2.884) | <0.001 |
| LVEF | 0.980 (0.950-1.011) | 0.198 | | |
| Presence of CAE | 1.551 (1.206-1.995) | 0.001 | 1.597 (1.238-2.060) | <0.001 |

BMI, body mass index; CAE, coronary artery ectasia; CK, creatine kinase; LVEF, left ventricular ejection fraction; MACE, major adverse cardiovascular events; MI, myocardial infarction; STEMI, ST-segment elevation myocardial; TIMI, Thrombolysis in Myocardial Infarction.

patients presenting with AMI. The presence of CAE in the culprit vessel has been previously analyzed in studies with smaller sample sizes: Yip *et al.*[22] found CAE in the culprit vessel in 2.6% of a cohort of 924 patients, whereas in another study consisting of 643 patients with myocardial infarction, the frequency of CAE was 4.8%.[23] The results of the present study, with 5 times larger population, confirm previous series and reported a frequency of CAE (irrespectively of its location) of 3.6% and 3.2% when considering the presence of CAE in the culprit vessel.

Regarding the angiographic findings, CAE involved the RCA in the majority of cases (79.3%). This higher predisposition of the RCA to develop CAE as compared to the other coronary arteries has been previously described[2] but the underlying pathophysiology remains unknown. In addition, multivessel CAE is infrequent and it has been described in only 25% of patients with CAE.[16] This is contrasting to the present study, where multivessel CAE was observed in 2/3 of the patients and the Markis type I pattern the most frequently anatomical phenotype observed. This marked discrepancy might be explained by the characteristics of the study population (AMI versus stable/asymptomatic patients).

A large thrombus burden and a low initial TIMI flow was observed in patients with CAE, which is consistent with previous studies.[22,24] A large thrombus burden may result from a decreased coronary flow velocity and a turbulent flow pattern, leading to platelet activation and thrombus formation in the dilated lumen.[25] Additionally, in patients with CAE complicated by obstructive coronary artery disease, the coexistence of both dilated and stenotic coronary segments may further impair coronary flow hemodynamics,[26] favoring the progression of atherosclerotic disease. Thrombus aspiration was subsequently more often used in patients with CAE. Thrombus aspiration in acute myocardial infarction has been shown to reduce distal embolization and improve coronary perfusion, myocardial blush grade and prevent no-reflow.[27] However, although thrombus aspiration and glycoprotein IIb/IIIa inhibitors have been frequently used in patients with AMI and CAE, the occurrence of no-reflow or distal embolization is very frequent.[24,28] We observed a non-significant higher frequency of final TIMI flow <3 in patients with CAE compared to non-CAE patients. In the present study, patients with CAE were less often treated with stent implantation when compared with their counterparts and larger stents were used. Percutaneous coronary intervention for culprits lesion in ecstatic coronary segments in the setting of AMI is associated with a higher rate of procedural failure and a higher incidence of adverse events.[28,29] Proper selection of stent according to the size and extent of CAE is critical to reduce the risk of stent thrombosis and stent migration. Intracoronary imaging techniques may be helpful for the assessment of the lumen diameter and landing.[30]

Previous studies have shown conflicting results on the prognostic impact of CAE. In the CASS study, the presence of CAE showed no effect on survival at 5-years after adjusting for confounding factors.[2,16] In a retrospective study of 203 patients with CAE, CAE did not confer added risk of MACE at 2-years when compared to a control group without CAE.[16] However, among 32,372 patients undergoing coronary angiography, Baman *et al.* (2) showed that the presence of CAE was associated with 1.56-fold adjusted 5-year mortality compared to those without CAE. In patients with AMI, we observed that the presence of CAE was associated to a 1.60-fold adjusted 4-year MACE compared to patients without CAE. These differences might be explained by the different characteristics of the study population and the definitions of CAE applied in each particular case. Furthermore, there is no consensus on the optimal therapeutic approach to CAE which potentially may determine clinical outcomes. Future investigations in this field should address these challenges.

Several limitations should be acknowledged. This is a single-center, observational retrospective analysis of prospectively clinically acquired data, with all the inherent limitations associated to the nature of the study. Patients with previous coronary artery bypass graft surgery were excluded, which may imply a selection bias. Systematic evaluation of intracoronary thrombus burden according to the TIMI thrombus scale was only performed in patients with CAE. Percutaneous coronary intervention optimization with intracoronary imaging was not routinely performed, which may have impacted on the procedural outcome. Due to the relatively small sample size of patients with CAE, underestimation of the association between CAE and MACE cannot be excluded.

In conclusion, the prevalence of CAE in patients presenting with AMI was 3.6 %. The presence of CAE was independently associated with an increased risk of MACE at 4-year follow-up. This association was independent from cardiovascular risk factors, type of AMI and number of diseased vessels.

## Disclosures

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