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Coronary Artery Ectasia as a Predictor of Major Adverse Cardiac Events in Patients with Acute ST Elevation Myocardial Infarction

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Coronary artery ectasia (CAE) is characterised as localised or widespread non-obstructive lesions of the epicardial coronary arteries with a luminal dilation more than 1.5 times the neighbouring normal segments or vessel diameter. Isolated CAE is CAE in the absence of severe coronary artery stenosis. This aberrant dilatation of coronary arteries can produce angina pectoris and even myocardial infarction in people without coronary artery disease owing to vasospasm, dissection, or thrombus. The purpose of this study was to evaluate the connection between CAE and major adverse cardiac events (MACE) following acute myocardial infarction with ST elevation.

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Methods: This was a prospective cohort study which was carried out on 300 cases. Cases were divided into two groups: Group I: including about 22 STEMI cases with CAE and Group II: including about 278 STEMI cases without CAE. All cases in this study were subjected to full history taking, clinical examination, laboratory tests, standard 12-leads ECG, resting transthoracic echocardiography (TTE), and coronary angiography.
Results: Regarding MACE in the studied groups, Incidence of reinfarction and cardiac death were significant higher in cases with STEMI and CAE than cases with STEMI alone. In univariate regression analysis, CAE (OR: 3.59, p value =0.022) was a significant predictor of cardiac death but age, male sex, and EF were not. Also, in multivariate regression analysis CAE (OR: 3.49, p value =0.029) was a significant predictor of cardiac death but age, male sex, and EF were not smoking with high warfarin consumption. Further, Markis classification 1 and 3 were the most frequent phenotype among cases. In STEMI cases, the incidence of reinfarction and cardiac death were significantly higher in cases with STEMI and CAE than cases with STEMI alone. So, CAE is a significant predictor of cardiac death.

Keywords: ST elevation myocardial infarction; coronary artery ectasia; adverse cardiac events.

1. INTRODUCTION

Myocardial infarction (MI) is described as the death of myocardial cells through persistent ischemia. MI is utilized clinically when there are signs of myocardial necrosis consistent with acute myocardial ischemia and an increase in cardiac troponin. Acute myocardial infarction is associated with a high death rate, although immediate coronary revascularization has significantly improved the prognosis. [1].

“Coronary artery ectasia (CAE) is characterized as localized or widespread non-obstructive lesions of the epicardial coronary arteries with a luminal dilation more than 1.5 times the neighboring normal segments or vessel diameter. Isolated CAE is CAE in the absence of severe coronary artery stenosis. This aberrant dilatation of coronary arteries can produce angina pectoris and even myocardial infarction in individuals without coronary artery disease [2] owing to vasospasm, dissection, or thrombus”.

“The prevalence is as high as 12 percent in the Indian population, which may have distinct demographic features. The male-to-female ratio is 3:1 [3], indicating a preponderance of men”.

“CAE is connected with inflammation, neuro-hormonal processes, and cardiovascular risk factors. Although it has been hypothesized that CAE is a subtype of coronary artery disease, a conclusive relationship between atherosclerosis and ectasia has not been established” [4].

However, it is unknown why equivalent risk factors result in arterial dilatation in some cases but arterial blockage in others [5]. “Cases with CAE often had significant vascular inflammatory infiltration involving all layers of the vascular wall in the coronary circulation, according to postmortem histopathologic investigations” [2].

According to previous research, “CAE risk variables include sex, BMI, diastolic blood pressure, d-dimer, triglyceride, and LDL/HDL ratio. Hyperlipidemia is a strong predictor of CAE” [6].

“CAE might be a lesion with a high risk of triggering acute coronary events. In addition, the increased thrombogenicity [7] in CAE implies that pharmacological treatments that modulate the coagulation cascade may be useful in preventing CAE-related coronary events”. In individuals with acute STEMI, the presence of CAE indicated subsequent cardiac events. Prior research revealed that acute MI cases with CAE constituted a high-risk subgroup that would benefit from a pharmaceutical strategy to regulate the coagulation cascade.

The purpose of this study was to evaluate the connection between CAE and major adverse cardiac events (MACE) following acute myocardial infarction with ST elevation.

2. MATERIALS AND METHODS

This prospective cohort observational study was carried out on 310 cases more than 18 years with acute STEMI who underwent primary percutaneous coronary intervention according to the recent guidelines of myocardial revascularization.

Exclusion criteria were cases with a history of previous stroke, patient with a history of

congestive heart failure or left ventricular systolic dysfunction, patient with serious arrhythmias and cases with rheumatic or congenital heart disease.

Cases were further divided in to two groups: group I: STEMI cases with CAE and group II: age and sex matched control group with STEMI only.

All cases were subjected to: Thorough history taking including [Age, sex, and risk factors for ischemic heart disease], medication, clinical examination and laboratory tests.

Resting Transthoracic Echocardiography (TTE): During a TTE, cases lied on their back or on their left lateral position on a bed or table.

Before discharge, TTE was conducted to detect wall motion abnormalities and assessment of LV systolic function. Using standard echocardiographic views, EDD, ESD, PWD, IVSD, FS and LVEF were measured using Philips Epic 7 and Vivid S5 Echocardiographic machines and the results were recorded according to ASE recommendations [8].

Coronary Angiography: "CAE was described as a coronary artery segment having a diameter more than 1.5 times the neighboring normal segment" [9]. "During the index coronary angiogram, cases with CAE in any coronary channel were found. The anatomical distribution of CAE on angiography was classified according to the Markis classification [10]: Type I: was classified as diffuse CAE in two or three coronary vessels, Type II: as diffuse CAE in one coronary artery and localized CAE in another vessel, Type III: as diffuse CAE in just one coronary vessel, and Type IV: as localized or segmental CAE".

Multivessel disease was defined as the presence of coronary stenosis greater than fifty percent in at least two main coronary arteries. Thrombolysis In Myocardial Infarction (TIMI) frame count approach was utilized to measure coronary artery flow [11]. According to the TIMI-thrombus scale [12], thrombus load was rated from 0 to 5. High thrombus load was defined as a TIMI-thrombus score of four or above. Angiographical success was defined as final TIMI 3 distal flows with less than 20% vascular stenosis and the absence of acute mechanical problems. No-reflow phenomena was classified as TIMI flow 2 without angiographic evidence of mechanical vascular occlusion [13].

Follow-up for significant cardiac adverse events (MACE): "MACE was defined as cardiac or non-cardiac mortality, death in hospital, re-infarction, readmission for acute coronary syndrome, and repeat coronary revascularization". Doi et al., [7]. In the current study cases were followed up our cases for one year for MACEs including re-infarction, stroke, serious arrhythmias and sudden cardiac death.

2.1 Statistical Analysis

Version 25 of the SPSS (Statistical Package for the Social Sciences) was used for statistical analysis (IBM Inc., Chicago, IL, USA). Using the Shapiro-Wilks normality test and histograms, the distribution of quantitative data was examined in order to identify the appropriate kind of statistical testing: parametric or nonparametric. Unpaired t. test was used to compare variables that were reported as mean and standard deviation (SD), such as age.

Categorial variables (e.g., sex) were expressed as frequency and percentage and were statistically analyzed by Chi-square or Fisher's exact test when appropriate. Logistic regression was performed to assess the probability of an event occurring based on one or more independent variables (predictors). A two-tailed P value ≤ 0.05 was considered statistically significant.

3. RESULTS

Baseline characteristics (age and gender) were insignificantly different between both groups. Regarding risk factors of CAD, DM and smoking were significantly higher in ectasia group compared to non-ectasia group (P value = 0.015 and 0.029 respectively) while hypertension, dyslipidaemia, and obesity BMI >30kg/m2 were insignificantly different between both groups. Regarding previous medications, warfarin use was significantly higher in ectasia group compared to non-ectasia group (P value = 0.011) while aspirin, DAPT, DAPT + warfarin, ACE-I/ARB, β-Blocker and Statin were insignificantly different between both groups (Table 1).

Vital signs (systolic blood pressure, diastolic blood pressure, HR and RR) and troponin levels were insignificantly different between both groups (Table 2).

There was no significant difference in main culprit lesion, and number of vessels involved between STEMI cases with or without CAE. Of STEMI cases with CAE, 12 (54.5%) had ectatic

LAD, 17 (77.3%) had ectatic RCA, and 9 (40.9%) had ectatic LCx. Markis classification was 1 in 6 (27.3%) cases, 2 in 3 (13.6%) cases, 3 in 7 (31.8%) cases, and 4 in 6 (27.3%) cases. Cases with 0-1 pre-PCI TIMI flow were significantly higher in STEMI cases with CAE than STEMI cases without CAE (0.037). There was no significant difference in cases with 2 and 3 pre-PCI TIMI flow between both groups (Table 3).

ECG was insignificantly different between both groups. EF was significantly lower in ectasia group compared to non-ectasia group (P value = 0.033). Regional wall motion abnormalities were significantly higher in CAE group compared to non-ectasia group (p <0.001). There was no significant difference in 2D echo parameters (LV ESV, LV EDV, PWD, and IVSD) between STEMI cases with or without CAE (Table 4).

Regarding MACE in the studied groups, Incidence of reinfarction and cardiac death were significantly higher in cases with STEMI and CAE than cases with STEMI alone (p =0.015, 0.031 respectively) Incidence if serious arrythmia was insignificantly different between the studied groups (Table 5).

In univariate regression analysis, CAE (OR: 3.59, p value =0.022) was a significant predictor of cardiac death but age, male sex, and EF were not. Also, in multivariate regression analysis CAE (OR: 3.49, p value =0.029) was a significant predictor of cardiac death but age, male sex, and EF were not (Table 6).

4. DISCUSSION

Coronary ectatic arteries (CAE) are characterised by a high thrombus load that predisposes to propagation into more distal coronary vasculature. This elevated thrombus load is presumably predisposed by the sluggish blood flow observed in ectatic arteries. Notably, coronary artery ectasia [14] has also been associated with angina pectoris and left ventricular dysfunction.

Determining the characteristics related with the existence and severity of CAE may thus be advantageous for the therapy of these individuals. Previous research has demonstrated that inflammation and atherosclerosis play important roles in the development of CAE, although the fundamental causes of ectasia production are still not entirely understood [15].

Since CAE is related with inflammation and usually coexists with CAD, it has been hypothesised that CAE is a subtype of CAD. On the basis of the results of earlier investigations, it has been hypothesised that a more severe inflammation may play a role in the aetiology of CAE [16].

In the present study, it was found that vital signs such as (systolic blood pressure, diastolic blood pressure, respiratory rate, and heart rate) were insignificantly different between ectatic group and non- ectatic group. In agreement with the present results, Baldi et al. [17] highlighted that there were insignificantly different between ectatic group and non- ectatic group regarding systolic blood pressure and diastolic blood pressure.

In the present study, it was found that there was no significant difference in main culprit lesion (LAD, RCA, and LCx) between ectasia and non-ectasia group (P = 0.839). On the contrary, Popovic et al. [18] highlighted that there was significant difference in main culprit lesion (LAD, RCA, and LCx) between ectasia and non-ectasia group (P <0.05). The contradiction between both studies can be justified by the larger sample size in the other study.

In the present study it was found that there was insignificant difference in number of vessels involved between STEMI cases with or without CAE.

Regarding multivessel disease number, Schram et al. [19] highlighted there was no significant difference in number of vessels involved between STEMI cases with or without CAE (P = 0.48).

In the present study, it was found that of the STEMI cases with CAE, 12 (54.5%) had ecstatic LAD, 17 (77.3%) had ecstatic RCA, and 9 (40.9%) had ecstatic LCx. In consistent with our results, Baldi et al. [17] highlighted that CAE involved the RCA in 79.2% of cases, LAD in 40.3%, and LCx in 35.1%.

In the present study, it was found that Markis classification was 1 in 6 (27.3%) cases, 2 in 3 (13.6%) cases, 3 in 7 (31.8%) cases, and 4 in 6 (27.3%) cases.

Table 1. Demographic data and previous medications of the studied groups

		Total (n =300)	STEMI with CAE (n =22)	STEMI (n =278)	P value
Age (years)	Mean ± SD	65.67 ± 8.77	63.05 ± 10.22	65.87 ± 8.63	0.293
Gender	Male	227 (76%)	18 (82%)	209 (75%)	0.611
	Female	73 (24%)	4 (18%)	69 (25%)	
Comorbidities	HTN	197 (66%)	15 (68%)	182 (65%)	1.000
	DM	91 (30%)	12 (55%)	79 (28%)	**0.015***
	Dyslipidemia	125 (42%)	8 (36%)	117 (42%)	0.659
	Smoking	212 (71%)	20 (91%)	192 (69%)	**0.029***
	Obesity (BMI >30kg/m^2)	32 (11%)	3 (14%)	29 (10%)	0.73
Previous medications	Aspirin	277 (92%)	21 (95%)	256 (92%)	1.000
	Warfarin	47 (16%)	8 (36%)	39 (14%)	**0.011***
	DAPT	84 (28%)	5 (23%)	79 (28%)	0.805
	DAPT + warfarin	15 (5%)	2 (9%)	13 (5%)	0.302
	ACE-I/ ARB	214 (71%)	14 (64%)	200 (72%)	0.463
	β-Blocker	168 (56%)	10 (45%)	158 (57%)	0.182
	Statins	163 (54%)	13 (59%)	150 (54%)	1.000

*STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, CAD: coronary artery disease, HTN: hypertension, DM: diabetes mellitus, BMI: body mass index, DAPT: dual antiplatelet therapy, ACE: angiotensin-converting enzyme, ARB: angiotensin receptor blocker, *Statistically significant as P value ≤ 0.05.*

Table 2. Clinical and laboratory data of the studied groups

		Total (n =300)	STEMI with CAE (n =22)	STEMI (n =278)	P value
Systolic blood pressure (mmHg)	**Mean ± SD**	143 ± 16	141 ± 16	143 ± 16	0.578
Diastolic blood pressure (mmHg)	**Mean ± SD**	80 ± 9	81 ± 7	80 ± 9	0.976
HR (beat/min)	**Mean ± SD**	75 ± 12	77 ± 17	74 ± 12	0.319
RR (breath/min)	**Mean ± SD**	16 ± 2	16 ± 3	16 ± 2	0.958
Troponin (pg/ml)	**Mean ± SD**	51 ± 25	57 ± 24	50 ± 25	0.232

STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, HR: heart rate, RR: respiratory rate

Table 3. Coronary angiography data of the studied groups

		Total (n =300)	STEMI with CAE (n =22)	STEMI (n =278)	P value
Main culprit lesion treated with PCI	**LAD**	119 (40%)	10 (45%)	109 (39%)	0.839
	RCA	102 (34%)	7 (32%)	95 (34%)	
	LCx	79 (26%)	5 (23%)	74 (27%)	
Number of vessels involved	**Single vessel**	184 (61.3%)	12 (54.5%)	172 (61.9%)	0.842
	Multi-vessel	116 (38.7%)	10 (45.5%)	106 (38.1%)	
Ectatic vessels	**LAD**	-	12 (54.5%)	-	-
	RCA	-	17 (77.3 %)	-	
	LCx	-	9 (40.9%)	-	
Markis classification	**1**	-	6 (27.3%)	-	-
	2	-	3 (13.6%)	-	
	3	-	7 (31.8%)	-	
	4	-	6 (27.3%)	-	
Pre-PCI TIMI flow	**0-1**	198 (66%)	19 (86.4%)	179 (64.4%)	**0.037***
	2	24 (12.5%)	0 (0%)	24 (8.63%)	0.235
	3	78 (3.85%)	3 (13.6%)	75 (26.9%)	0.212

*STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, LAD: Left anterior descending, RCA: Right coronary artery, LCx: Left circumflex, TIMI: Thrombolysis in Myocardial Infarction, *: statistically significant as P value ≤ 0.05.*

Table 4. ECG and Echocardiographic data of the studied groups

		Total (n =300)	STEMI with CAE (n =22)	STEMI (n =278)	P value
ECG	**Anterior STEMI**	119 (40%)	10 (45%)	109 (39%)	0.912
	Inferior STEMI	65 (22%)	5 (23%)	60 (22%)	
	Posterior STEMI	37 (12%)	2 (9%)	35 (13%)	
	Lateral STEMI	79 (26%)	5 (23%)	74 (27%)	
EF (%)	**Mean ± SD**	48 ± 6	45 ± 6	48 ± 6	**0.033***
	Range	35 – 62	35 – 54	37 – 62	
LV ESV (mL)	**Mean ± SD**	91 ± 10	93 ± 9	90 ± 10	0.101
	Range	64 – 105	64 – 103	74 – 105	
LV EDV	**Mean ± SD**	149 ± 18	153 ± 26	149 ± 17	0.307

		Total (n =300)	STEMI with CAE (n =22)	STEMI (n =278)	P value
(mL)	**Range**	106 – 188	106 – 188	120 – 176	
PWD	**Mean ± SD**	10 ± 1	10 ± 2	10 ± 1	0.408
(mm)	**Range**	7 – 13	7 – 13	8 – 12	
IVSD	**Mean ± SD**	9 ± 1	9 ± 1	9 ± 1	0.573
(mm)	**Range**	7 – 13	8 – 10	7 – 13	
Regional wall motion	**Yes**	24 (8%)	8 (36.4%)	16 (5.75%)	**<0.001***
abnormalities	**No**	276 (92%)	14 (63.6%)	262 (94.24%)	

*STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, ECG: electrocardiogram, STEMI: ST-elevation myocardial infarction, EF: ejection fraction, LV: Left ventricle, ESV: End systolic volume, EDV: End diastolic volume, PWD: Posterior wall diameter, IVSD: Interventricular septal diameter. *: statistically significant as p value ≤ 0.05*

Table 5. Major Adverse Cardiac Events (MACE) in the study participants during the follow up period

		Total (n =300)	STEMI with CAE (n =22)	STEMI (n =278)	P value
MACE	Reinfarction	22 (7%)	5 (23%)	17 (6%)	**0.015***
	Serious arrythmia	74 (25%)	6 (27%)	68 (24%)	0.798
	Cardiac death	26 (8.7%)	5 (22.7%)	21 (8%)	**0.031***

*STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, MACE: Major adverse cardiac events, *: statistically significant as P value ≤ 0.05.*

Table 6. Univariate logistic regression analysis for the prediction of death among the study participants

	Univariate regression analysis		Multivariate regression analysis	
	OR (95% CI)	**P value**	**OR (95% CI)**	**P value**
Age	0.99 (0.95 – 1.04)	0.919	1.003 (0.957 – 1.05)	0.956
Sex (male)	1.07 (0.42 – 2.79)	0.876	1.04 (0.396 – 2.726)	0.937
EF	0.98 (0.92 – 1.04)	0.483	0.99 (0.926 – 1.051)	0.678
CAE	3.59 (1.21 – 10.72)	**0.022***	3.49 (1.139 – 10.618)	**0.029***

*STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, STEMI: ST-elevation myocardial infarction, CAE: Coronary artery ectasia, MACE: Major adverse cardiac events, *: statistically significant as P value ≤ 0.05.*

In agreement with the current results, Baldi et al. [17] highlighted that Markis classification was 1 in 44 (28.6 %) cases, 2 in 16 (10.4%) cases, 3 in 75 (48.7 %) cases, and 4 in 19 (12.3)% cases.

Similarly to the present results, Kundi et al. [20] analyzed retrospective electronic patient data recording. 330 individuals were enrolled in the trial, comprising 110 cases with isolated CAE, 110 cases with obstructive CAD without CAE, and 110 cases with NCA. The results showed that Markis classification was 1 in 34 (30.9) %) cases, 2 in 19 (17.2 %) cases, 3 in 15 (13.6 %) cases, and 4 in 42 (38.1 %) cases.

In the present study, it was found that cases with 0-1 pre-PCI TIMI flow were significantly higher in STEMI cases with CAE than STEMI cases without CAE (0.037). There was no significant difference in cases with 2 and 3 pre-PCI TIMI flow between ectasia and non-ectasia group.

In accordance with our results, Popovic et al. [18].highlighted that patient with 0-1 pre-PCI TIMI flow were significantly higher in STEMI cases with CAE than STEMI cases without CAE (P = 0.0179). There was no significant difference in cases with 2 and 3 pre-PCI TIMI flow between ectasia and non-ectasia group.

Also, Schram et al. [19] highlighted that cases with 0-1 pre-PCI TIMI flow were significantly higher in STEMI cases with CAE than STEMI cases without CAE (P < 0.001).

However, Baldi et al. [17] highlighted that IMI flow grade was not statistically different between groups (P = 0.283). Larger sample size in the other study may be the cause of this contradiction between both studies.

Regarding MACE in the studied groups, it was observed that the incidence of reinfarction and cardiac death were significant higher in cases with STEMI and CAE than cases with STEMI alone (p =0.015, 0.031 respectively). In agreement with our results, Doi et al. [7] highlighted that the incidence of MACE was considerably higher in CAE cases compared to cases without CAE where cardiac death was significantly higher in cases with STEMI and CAE 14 (28%) than cases with STEMI alone 130 (8%) (P=0.03).
In univariate regression analysis, it was found that CAE (OR: 3.59, p value =0.022) is a significant predictor of cardiac death but age, male sex, and EF were not. In agreement with our results, Doi et al. [7] highlighted that that CAE (p value < 0.001) is a significant predictor of cardiac death but age, and male sex, were not.

However, Mir et al. [14] performed a meta-analysis where studies comparing outcomes of PCI in CE versus no-ectasia (NE) STEMI cases were identified. The results showed that both groups had comparable follow-up mortality results with [OR: 0.83, 95% CI 0.39–1.78; p = 0.63]. where they did not find any significant differences in cardiac death for STEMI in cases with and without coronary ectasia. The contradiction between both studies can be justified by the fact that all included studies were observational retrospective, with variable follow-up durations and different selection criteria. The predictive odds of all the components could not be calculated due to insufficient reporting of the stratified event rates.

Limitations were relatively small sample size, this study was observational cohort study that may include some bias and lack of some clinical investigation (e.g., Inflammatory biomarkers).

5. CONCLUSIONS

In ectasia patient there was a significant increase in CAD, DM and smoking with high warfarin consumption. Further, Markis classification 1 and 3 were the most frequent phenotype among cases. In STEMI cases, the incidence of reinfarction and cardiac death were significant higher in cases with STEMI and CAE than cases with STEMI alone. CAE is a significant predictor of cardiac death.

ETHICAL APPROVAL AND CONSENT

An informed written consent was obtained from the patient or relatives of the cases. The study was done after approval from the Cardiology department, Tanta university hospital and Mahalla Cardiac Centre Cath. Lab.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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