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ORIGINAL ARTICLE

Relation between coronary artery anatomy and location of culprit lesions in patients with ST segment elevation myocardial infarction

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ABSTRACT

Background: In patients with STEMI, culprit lesions are frequently located immediately distal to bifurcations and in proximity to major vascular curvatures. Culprit lesions in the LCA are more proximal, are closer to bifurcation branches, and result in larger infarctions and greater in-hospital mortality than culprit lesions in the RCA .

Objective: The aim was to evaluate the frequency and distribution of culprit lesions in patients with ST-segment elevation acute myocardial infarction.

Methods: In this prospective cohort study 222 patients with STEMI were included in the period from June 2014 to June 2019 at the cardiology department in Agouza hospital. Patients were divided into 3 groups:

Group (A): includes patients with culprit lesion is in LAD Group (B):includes patients with culprit lesion is in LCX .Group (C) :includes patients with culprit lesion is in the RCA. We survey the number of culprit lesions in every 10 mms extent from ostium , bifurcation and curvature using angiography, intravascular ultrasound and instantaneous wave-free ratio(IFR)

Result: Culprit lesions were within 20 mm of a bifurcation in 77% of patients and closer to the bifurcation in the LCA compared with the RCA. RCA culprit lesions, 45% were within 20 mm of a major curvature. Compared with those in the RCA, culprit lesions in the LCA were more proximally and were associated with larger myocardial infarctions .

Conclusion: In patients with STEMI, culprit lesions are frequently located immediately distal to bifurcations and in proximity to major curvatures

INTRODUCTION

Coronary atherosclerosis continues to be a leading cause of morbidity and mortality all over the world **[1]** Plaque can give rise to a wide spectors of adverse clinical events, including myocardial infarction and Sudden cardiac death **[2].**Lifestyle improvements, modification of risk factors and therapeutic interventions helped to decrease the incidence of myocardial infarctions in developed countries **[3]** Therefore, deep understanding of pathological mechanisms of underlying coronary plaque development, progression and vulnerable plaque rupture is critically important for clinical and pharmacological research for new diagnostic and therapeutic strategies .

PATIENTS AND METHODS

This prospective cohort study was conducted in Cardiology Department, agouza police Hospitals. We included **222** consecutive patients with STEMI undergoing diagnostic cardiac catheterizations and percutaneous coronary interventions in our Cath Lab, within a time period from June 2014 to June 2019. Patients were

divided into 3groups: Group (A): patients with STEMI with culprit is in LAD ,Group (B): patients with STEMI with culprit is in LCX and Group (c):patients with STEMI with culprit is in RCA . In addition, the following anatomic features of lesions were considered 1.The distribution of culprit lesions as indicated by number of culprit lesions in each 10 mm distance from ostium, bifurcation or major curvatures 2. Calculation of the degree of axial bending and deformity of the vessel 3. We evaluated other non-culprit plaques into culprit and non-culprit vessels using (quantitative coronary angiography-coronary measurement system [QCA-CMS], intravascular ultrasound (IVUS) and instantaneous wave-free ratio (IFR).(Written consent of acceptance of sharing in the study was taken from all patients. The study was approved by the research ethical committee of Faculty of Medicine, Zagazig University and agouza police Hospital. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Inclusion Criteria: 1. Patients between 18 – 70 years. 2. Rise in cardiac biomarkers of myocardial necrosis (Troponin T) above reference level .3. Patients should have a definite culprit lesion.

Exclusion Criteria: 1. Patients with significant valvular heart disease **2.**Patients who had a previous angioplasty at the site of the culprit lesions.3.Previous MI.4.Patients without a clearly identifiable culprit lesions.5.Previous CABG.

Methods: the patients were subjected to full history taking, clinical examination, and imaging studies. Imaging studies included echocardiography, resting 12 lead surface ECG, coronary angiography. Digital coronary angiograms were analyzed offline with an automated edge detection system (Philips Integris 5000, Netherland) by using the dye-filled guiding catheter as a reference. with facilities of using IVUS and IFR. Serum creatinine and blood urea nitrogen were done before coronary angiography while troponin and CK-MB were done every 8 hours for 1st 24 hours then daily for 3 days and the presence of complications such as coronary dissection, perforation, abrupt vessel closure or others were recorded. All patients were followed-up during the hospital period with special emphasis on all causes of mortality and recurrent ischemic symptoms with or without re-elevation of cardiac enzymes which necessitate second angiography.

Statistical analysis:

Data were analyzed using the Statistical Package for Social Sciences (SPSS) release16.Data showing normal distribution were presented as the means and standard deviation. For comparison between the means of two groups, the t-test was used. The non-parametric values were tested using the Mann–Whitney-U test. Qualitative data are represented by frequency and relative percentage and chi-square test was used for testing the association of the qualitative data. In all analyses, *P* values <0.05 were considered statistically significant.

RESULTS

The cardiac enzyme presented in **Table(1).**There were statistically significant differences between them in the **cardiac biomarkers (troponin and CK-MB)** with higher levels in LAD group compared to RCA and LCX groups **respectively. (P<0.05).**

*Also There were **statistical significant difference** between the study groups regarding Ejection fraction **p value<0.05**.There was lower ejection fraction in LAD group than in LCX and RCA groups***.** Regarding other vulnerable plaque in multivessel disease using angiography ,IVUS and IFR , there were 56(65%), 17(68%) and 76(68%) in the three groups respectively. There was no significant statistical difference **Table (1)**

Seventy-four percent of culprit lesions were in the proximal 40 mm of the vessel. This was most evident in the LCA, where 78% of culprit lesions were in the proximal 40 mm from the ostium of the LAD or LCX. In contrast,64% of RCA culprit lesions were within the proximal 40 mm of the vessel, with a bimodal peak incidence The mean distances from the Ostia of individual vessels to culprit lesions were 22.7 ± 18.2 mm in the LAD, 28.2 ± 11.9 mm in the LCX, and 44.7 ± 28.8 mm in the RCA. Culprit lesions in the LCA were closer to the ostium compared with the lesions located in the RCA **(P b .0011). Figure (1)Table (2)** Culprit lesions were in proximity to major proximal bifurcations and the frequency of culprit lesions decreased with increasing distance from major bifurcations Forty-five percent of culprit lesions were within 10 mm and 77 % were within 20 mm of a bifurcation. Culprit.lesions were closer to the bifurcation in the LCA compared with the RCA. This finding was most evident in the LAD, where 23% of culprit lesions were within 10 mm and 77% were within 20 mm distal to a bifurcation. Similar results were noted in the LCX, where 61% of culprit lesions were found within 10 mm and 90% within 20 mm, and In the RCA, 13.5% were within 10 mm and 45.04% were within 20 mm distal to a curvatures. **Figure (2,3) Table (3)**There was statistically significant difference between LV systolic function as expressed by EF% ,cardiac

enzyme and axial deformity and lesion distance from LAD ostium . lesions in the LCA are located more proximally, closer to bifurcations, and associated with larger myocardial infarctions, lower ejection fractions, and higher in-hospital mortality.**Table (4)** There was **no statistically significant differences** regarding luminal percent stenosis as assessed by **IVUS, or iFR** of non-culprit palques in multivessel disease patients between groups. **Figure (4,5) Table (5)**The data of clinical outcomes are presented in **Table (6)** Patients with totally occluded LAD vessel had higher incidence of MACE during hospital stay (20patients, 23%) versus (three and thirteen patients, 12,11%) in patients with occluded LCX and RCA vessel and higher recurrent ischemic symptoms and decompensated **HF (Table 6)**

Table (1) EF, Troponin and cardiac enzymes in different study groups:

Variable	LAD	LCX	RCA	P value
EF (%)	46.8(±8.5))	58.8 (±6.8)	52.4% (±8)	0.02
Troponin	64.6(±61.25)	26 (±22.6)	46.55(±29.7)	0.03
CK_MB	251.2 (±1)	149.56(±69.2)	154.8 (±63.9)	0.005
MVD *	56(65%)	17(68%)	76(68%)	0.8

*MVD (multi-vessel disease, other non-culprit significant plaques present in same or other vessels using IVUS and IFR)

Table (2) Anatomic characteristics of culprit lesions sites on LAD, LCX and RCA

Anatomical parameters	LAD	LCX	RCA	P.value
Distance from ostium(mm)	**22.7±18.2**	**28.2 ± 11.9**	**44.7 ± 28.8**	**0.011**
Distance from bifurcation(mm)	**12 ±5.1**	**14±4.8**	**17.6±4.5**	**0.023**
More proximal plaque present (N,%)	**13(23%)**	**2(11.7%)**	**26(34%)**	**0.017**
***Degrees of angulation on lesion**	**155±20.5**	**130.9±17.7**	**161.7±19.5**	**0.077**
****Axial deformity (degrees)**	**25.1±12.3**	**13.1±5.5**	**26.3±9.8**	**0.032**

***In the projection that show most severe angle in diastole
****axial deformity(difference in angle between systole and diastole)

Table (3) The frequency distribution of culprit lesions' distance from the most proximal bifurcation to culprit lesion into LCA(LAD ,LCX) and RCA

Culprit Artery	Number	ratio
LAD		
(0_10mm)	20	23%
(10_20mm)	66	77%
LCX		
(0_10mm)	6	24%
(10_20)	19	76%
RCA		
(0_10mm)	15	13.5%
(10_20mm)	50	45.04%
(20_30mm)	46	41.4%

Table (4) correlation between proximity of culprit lesion from LAD ostium and other studied variables:

Variable	r	P value
Age	0.14	0.2
Weight	0.1	0.35
Height	-0.05	0.7
EF%	0.49	0.02
Troponin	-0.753	<0.001
CK-MB	-0.42	0.02

Variable	r	P value
Non culprit proximity to bifurcation (IVUS)	-0.107	0.07
iFR	-0.019	0.5
Angulation on lesion	0.49	0.02
Axial deformity	-0.61	0.04

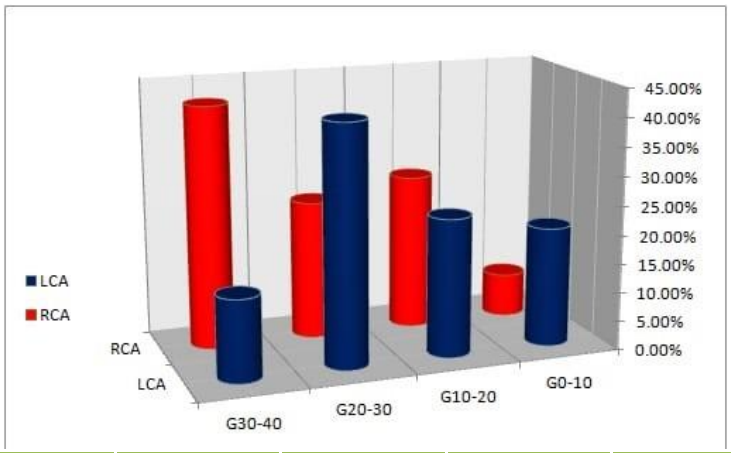
Table (5) comparison between LAD and RCA regarding luminal percent stenosis as assessed by **IVUS, or iFR** of non-culprit palques in multivessel disease patients:

variable	LAD lesions N=56	RCA lesions N=76	Significance
Luminal stenosis (%) (IVUS)	62.5%±18.5	57.2%±18.8	**0.64**
iFR	0.89±0.17	0.93±0.12	**0.37**

Table (6) Showing specific MACE related to each group:

Culprit lesion	*LAD*	*LCX*	*RCA*	*P-value*
HF	*10*	*1*	*2*	*0.005*
VT&VF	*7*	*2*	*9*	*0.03*
DEATH	*3*	*0*	*2*	*0.04*

Culprit lesion	MACE	P- value
LAD	20 (23%)	0.039
LCX	3 (12%)	
RCA	13 (11%)	



	G 0-10	G 10-20	G 20-30	G 30-40
LCA	14.31%	41.30%	23.91%	20.65%
RCA	41.79%	23.88%	26.87%	7.46%

Figure (1) Culprit lesion distance from ostium

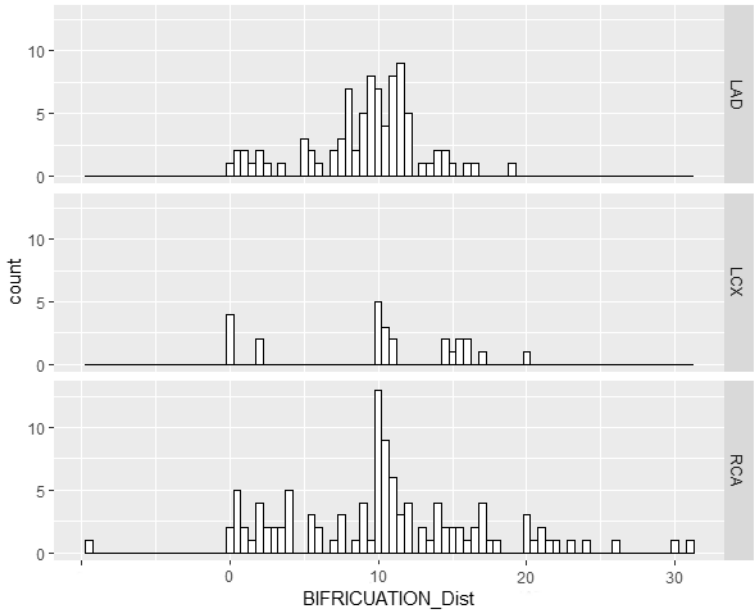


Figure (2) Bar chart showing; the frequency distribution of culprit lesions' distance from the bifurcation to culprit. Culprit lesions were more proximally located in the LCA compared with the RCA.

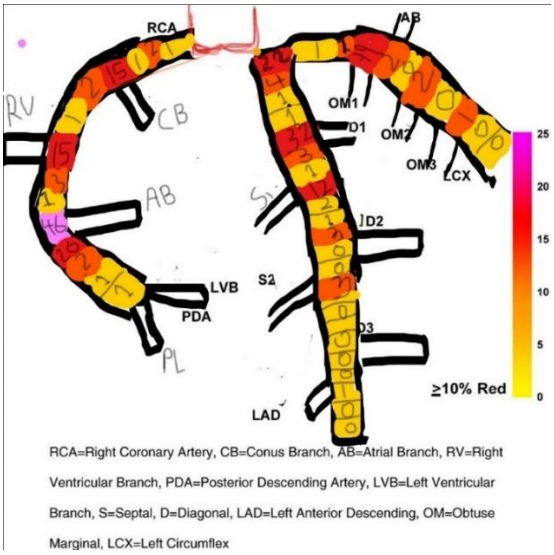


Figure (3) The relationship of culprit lesions to the specific bifurcations in the left and right coronary arteries.

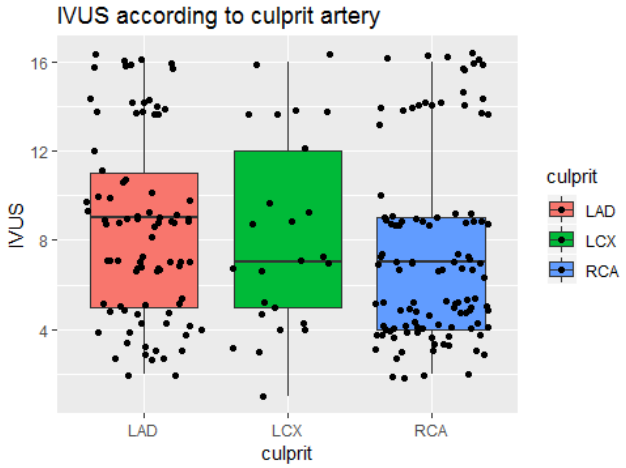


Figure (4) Bar chart shows using IVUS to assess proximal plaque into three different study group which shows insignificant plaque (insignificant lesion) in same vessel or other vessel

Figure (5) Bar chart shows using IFR to assess proximal plaque into three different study group which shows insignificant plaque (insignificant lesion) in same vessel or other vessel

DISCUSSION

This study demonstrates that, in patients with STEMIs undergoing cardiac catheterization, culprit lesions resulting in STEMIs are frequently located in the proximal coronary arteries, immediately distal to bifurcations, and in proximity to the major curvatures (in the RCA) where blood flow is known to be disturbed. In addition, compared with culprit lesions in the RCA, lesions in the LCA are located more proximally, closer to bifurcations, and associated with larger myocardial infarctions, lower ejection fractions, and higher in-hospital mortality.

Atherosclerosis is the most common cause of CAD. Coronary plaque disruption with overlaying thrombosis is the underlying cause of ACS, including unstable angina, NSTEMI, STEMI and sudden cardiac death. Thin cap fibroatheroma (The fibrous cap that is <65 µm in thickness) has been postulated as the precursor lesion of plaque rupture. It usually occurs in lesions showing < 50% diameter stenosis and is mostly observed in the proximal LAD, left circumflex and proximal RCA, followed by mid and is less frequent in distal coronary arteries [2]

It is well known that patients post STEMI are expected to experience more cardiac events both during index hospitalization and that continue short term after the index MI [5]

Regarding demographic characteristics of study patients and risk factors for CAD, we found no statistically significant difference between the studied three groups. This reflects the global nature of coronary atherosclerosis and that all three epicardial coronaries are prone relatively equally to same demographic and risk factors regarding the risk of plaque rupture[6] Our findings agree with those of McDaniel et al ., who demonstrated the same proximal clustering of STEMI and Non _STEMI culprits in epicardial coronaries and immediately distal to bifurcation especially in left coronary circulation .

In addition, in an optical coherence tomographic investigation of 55 patients with acute coronary syndromes, carried out by Fujii et al ., [7] there were 51% of the thin cap fibroatheroma that were near to a significant bifurcation. They demonstrated a mean distance of 20 ± 1.4 mm between the side branch and minimum fibrous-cap thickness site.

Previous studies have demonstrated the same concept in STEMI secondary to LAD culprits in the presence or absence of ramus artery, they showed more proximal location of culprit in LAD in those with Ramus intermedius artery present and subsequently larger myocardial infarction and more MACE and complications [8]

Thus in RCA culprit lesions plaques had a mean distance to proximal curve of 15,2 ±6.4 mm and mean distance to distal curve of 13± 8.2 , This finding goes hand in hand with that of MC Daniel et al ., [6] study which showed that peak incidence of culprit lesions in the RCA occur 10_20mm from the first curvature. They studied that culprit lesion cluster at the ostium and at the first and second major curvatures In addition, they demonstrated that the incidence of culprit lesions decreased with increasing distance from the curves.

Regarding the presence of a more proximal non culprit plaque in relation to bifurcation distance, there were 13(23%) cases in the LAD group, 2(11.7%) in the LCX group and 26(34%) cases in the RCA groups. We demonstrated that in RCA group, non-culprit lesions were significantly more proximally located in relation to bifurcations compared to both LAD and LCX non-culprit lesions groups. To the best of our knowledge, this is the first study to address this finding.

During cardiac cycle coronary arteries undergo cyclic longitudinal deformation and axial bending (flexion) and stretching. Plaque lesion angulation and axial deformity during cardiac cycle significantly affect wall shear stress and may extend mechanical distortion of atherosclerotic plaque [9]

Such finding of less axial deformity in the LCX artery could partially explain the fact that STEMI involving acute occlusion of the LCX occur less often than those involving the RCA or LAD (Fuster et al .,). Such lower frequency of ischemic events was attributed to difference in LCX geometry that lead to changes in wall shear stress that might make plaques rupture and thrombosis less common [10]

Another explanation of that was suggested by other authors to be due to ECG insensitivity in ACS events involving LCX artery, that some of those ACS events pass clinically unnoticed [11]

Conclusion:

Culprit lesions in STEMI tend to cluster in the proximal portion of epicardial coronaries (especially left coronary arteries) and. culprit lesions are frequently located immediately distal to bifurcations and in proximity to major vascular curvatures. Axial deformity imparted by the lesion angulation in relation to vessel may participate in vulnerable plaque rupture. Presence of more proximal non_culprit plaques in relation to ostium or bifurcation in same culprit coronary or other coronaries was noted and that merits further investigation.

REFERENCES

1. *Sacco RL*, *Roth GA*, *Reddy KS*, Arnett DK, Bonita R, Gaziano TA , et al , The heart of 25 by 25: achieving the goal of reducing global and regional premature deaths from cardiovascular diseases and stroke: a modeling study from the American Heart Association and World Heart Federation Circulation **2016** ;133 pp. e674-e690
2. Libby P, Pasterkamp, Jia H, Abtahian F, Aguirre AD, Lee S, Chia S , et al Requiem for the "vulnerable plaque."Eur Heart J **2015;** *36*:2984–2987. 10.1093/eurheartj/ehv3
3. *Thom T*, Haase N, Rosamond W, Howard VJ, Rumsfeld J, Manolio T, et al A Report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2006;113(6):e85–e151.
4. *Tang X*, *Yang Y*, *Luo S*, Zhao *Y*, Lu C, *Luo Y*, et al The effect of statin therapy on plaque regression following acute coronary syndrome: A meta-analysis of prospective trials. Coronary. Artery Disease **2016**; 27, 636–649
5. Maehara A, Mintz GS, Bui AB, Walter OR, Castagna MT, Canos D, et al. Morphologic and angiographic features of coronary plaque rupture detected by intravascular ultrasound. J Am Coll Cardiol 2002.;40:904–910.
6. McDaniel, Erin M. Galbraith, Kashlan OR, Eshtehardi P, Suo J, Dhawan S, et al Localization of culprit lesions in coronary arteries of patients with ST-segment elevationmyocardial infarctions: Relation to bifurcations and curvature Am Heart J.2011;161(3):508-15. doi: 10.1016/j.ahj.
7. Fujii K, Kawasaki D, Masutani M, Okumura T, Akagami T, Sakoda T et al OCT assessment of thin-cap fibroatheroma distribution in native coronary arteries. JACC Cardiovasc Imaging 2010;3:168-75.
8. Galbraith EM, McDaniel MC, Jeroudi AM, Kashlan OR, Suo J, Giddens D, et al Am J Cardiol. 2010;106(2):162-6. doi: 10.1016/j.amjcard.

9. Katritsis DG, Pantos J, Efstathopoulos E. Tzanalaridou *E*, De Waha *A*, Siontis et al Hemodynamic factors and atherosclerotic plaque rupture in the coronary arteries: From vulnerable plaque to vulnerable coronary segment.Progr Coronay Artery Disease 2008.;18:229–237.

10. Wang JC, Normand S-LT, Mauri L, Kuntz RE, Williams DO. Coronary artery spatial distribution of acute myocardial infarction occlusions circulation 2004;110:27884.d10.1161/01.

11. Hong MK, Mintz GS, Lee CW, Park DW, Choi BR, Park KH, et al A three-vessel virtual histology intravascular ultrasound analysis of frequency and distribution of thin-cap fibroatheromas in patients with acute coronary syndrome or stable angina pectoris. Am J Cardiol 2007;101(5):568–572.

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