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Keywords

Acute coronary syndrome; Significant coronary lesion; Vulnerable plaque

Understanding the Current Status of the Egyptian Coronaries; which is More Prone to Lesions?

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Abstract

Background: Atherosclerotic coronary artery disease (CAD) has been proved to have an early onset, a long latent period with heterogeneity in its phenotypic and clinical expression. It still represents the main cause of death among all cardiovascular disease. High-probability zones of coronary atherosclerosis may exist along the whole coronary tree.

We aimed to determine the nature of distribution of significant coronary lesions among our patients.

Methods: This study included 529 patients, for whom coronary angiography was done for suspected or proved coronary artery disease (CAD), they were divided into 3 groups according to the distribution of coronary lesions: LAD group (n=305) with significant LAD lesion, LCX group (n=148) with significant LCx lesion& RCA group (n=181) with significant RCA lesion.

Results: 169 (31.9%) had non-significant lesion, 166 (31.4%) had single vessel disease, with significantly higher incidence of significant LAD lesion 305((57.5%) which were proximal 52.4%, LAD lesions were more prone to be the culprit vessel 47.5%, LCx was the least vessel with significant lesion 148 (27.9%), and the least to be prone as a culprit 21.1%. Proximal culprit LAD was 63.5% and RCA55.6% had significantly higher incidence, mid culprit LCx 53.9% had significantly higher incidence. ST-elevation acute coronary syndrome (STE-ACS) was significantly more prevalent in culprit LAD 76.7%. NSTE-ACS was significantly more prevalent in culprit LCx 56.5% and RCA 55.6%.

Conclusion: LAD tends to carry more than one culprit lesion, more to be proximal. Risk factors responsible for instability and sheer stress (uncontrolled DM, uncontrolled hypertension, heavy smoking) were more prevalent between patients with LCx as a culprit followed by RCA in Egyptian; this may throw the light on the need for aggressive control of these risk factors to reduce vulnerability in these patients.

Introduction

Atherosclerotic coronary artery disease (CAD) has an early onset, a long latent period with heterogeneity in its phenotypic and clinical expression. It represents the main cause of death among all cardiovascular disease [1].

There is a constant interaction between blood flow and arterial geometry. While the blood circulates through the vasculature with every cardiac cycle, the vessel geometry alters the hemodynamic characteristics; consequently, changes in blood flow affect the morphology and function of arteries, resulting in the production of a complex hemodynamic pattern [2,3].

High-probability zones of coronary atherosclerosis may exist along the coronary tree [4,5]. As the coronary arteries are tethered to the surface of the beating heart, they undergo cyclic longitudinal deformations by axial bending (flexion) and stretching, cyclic flexion may fatigue and weaken the plaque on long-term, while a sudden accentuated longitudinal flexion may trigger plaque disruption [5,6].

Atheromatous plaque rupture with superimposed thrombosis is the main step in the pathogenesis of acute coronary syndromes (ACS) [1]. Which coronary vessel is the culprit and where is the vulnerability within the different clinical presentations of CAD- is unclear? Various studies have found inconsistent variabilities in the distribution of culprit lesions among coronary arteries in patients presenting with ACS. The proximal and mid portions of the coronary arteries have been identified as the most frequent sites of plaque rupture with a higher incidence in the proximal portion of the left anterior descending artery (LAD) [7,8] and an under presentation of the left circumflex coronary artery (LCx) in STEMI [9,10].

We aimed to determine the nature of distribution of significant coronary lesions among our patients.

Methods

This retrospective observational study included 529 patients, for whom coronary angiography was done for suspected or proved CAD. Patients with heart failure, severe renal impairment with eGFR<30 m ml/min, advanced degree of valve lesion, previous bypass graft or PCI with stenting, atrial fibrillations, malignancy or chronic inflammatory disease were excluded from the study.

Approval was obtained for performing the study from the Ethical Committee of the Faculty of medicine, Zagazig University, Egypt.

As the rule before all coronary angiography, after giving a written informed consent, all patients were subjected to the following:

1. **Full history taking:** specifically history of typical ischemic chest pain, demographic data and risk factors of CAD.
2. **Thorough clinical examination**, **body mass index (BMI)**: calculation was of special concern.
3. **Electrocardiographic examination:** Standard 12-lead surface ECG was done for every patient. It was analyzed by 2 different physicians to diagnose STE-ACS and NSTE-ACS, according to the guide lines [11].

As our concern was type of presentation and coronary lesion distribution, left ventricular ejection fraction, and wall-motion score index were not included in our study.

4. **Laboratory investigations:** Mainly for high sensitivity troponin.
5. **Coronary angiography:** Coronary angiography was performed using the Judkin's method, following the puncture of the femoral artery or via a radial artery approach. The severity of coronary atherosclerotic lesions was evaluated from at least 3 projections in all the patients for the visual classification of the coronary artery map in accordance with the guidelines of the American College of Cardiology/American Heart Association [12]. In ACS, we relied upon electrocardiograms (ECG) [11] or echocardiographic identification of the myocardial area at risk [13] to conclude that the "culprit" artery was the artery with at least one lesion that needed acute intervention.

In other patients, the culprit lesions were identified and treated by the operator on-site depending on the extent and severity of narrowing as well as the findings of stress tests (ECG, echocardiographic, or nuclear) or fractional flow reserve measured. All these data were recorded in the data base of the cath. Lab from which we collected.

Significant stenosis was defined as a diameter stenosis of 70% or greater.

Coronary artery territories and its segments [14]:

- *Left main*: From the ostium of the LCA through bifurcation into left anterior descending and left circumflex branches.
- *LAD proximal:* Proximal to and including first major septal branch.
- *LAD mid:* LAD immediately distal to origin of first septal branch and extending to the point where LAD forms an angle (RAO view). If this angle is not identifiable this segment ends at one half the distance from the first septal to the apex of the heart.
- *LAD distal:* it is the segment distal to the described mid segment.
- *Left circumflex artery (LCx) proximal:* Main stem of circumflex from its origin from the left main and including origin of first obtuse marginal branch.
- *Mid LCx:* it is the mid part of the LCx and of the proximal 1-2 cm of obtuse marginal branches. Distal LCx: The stem of the circumflex distal to the origin of the most distal obtuse marginal branch, and running along the posterior atrioventricular groove.
- *RCA proximal:* From the ostium to one half the distances to the acute margin of the heart.
- *RCA mid:* From the end of first segment to acute margin of heart.
- *RCA distal:* From the acute margin of the heart to the origin of the posterior descending artery.

We divided our patients into 3 groups according to the coronary artery disease and culprit lesion:

LAD group: 305 patients with significant LAD lesion.

LCx group: 148 patients with significant LCx lesion.

RCA group: 181 patients with significant RCA lesion.

Statistical Analysis

All data were analyzed using SPSS software statistical package for social science version 19 (SPSS, Inc. Chicago, IL, USA). Results were presented as mean value ± SD for continuous variables and as frequency (%) for categorical variables. Data was tested for normality using the Kolmogorov-Smirnov test. Means were compared using ANOVA, Student t-test or Mann-Whitney test according to the number of groups in comparison. Categorical data were compared using chi-squared test. P value was set at <0.05 for significant results & <0.001 for highly significant results.

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Table 1: Demographic data of the studied groups.

Variables	LAD 305 (57.7%)	LCx 148 (28%)	RCA 181 (34.2%)	P
Age (yrs)	55.7 ±10.6	57.1±9.9	57.7±9.9	>0.05
Gender: males females	245 (80.6%)^^ 59 (19.4%)	112 (76.2%)$ 35 (23.8%)	145 (80.1%) 36 (19.9%)	>0.5
Hyperchol	176 (57.7%)	85 (57.4%)	138 (63.3%)	>0.05
Hypertension	165 (54.1%)	101 (68.2%)*	117(64.6%)^	*<0.05 LAD#Lcx ^<0.05 LAD#RCA
Smokers	136 (44.7%)	84 (56.8%) *	95 (52.5%)	*<0.05 LAD#Lcx
DM	128 (42%)	75 (50.7%)*	88 (48.6%)	*<0.05 LAD#Lcx
FH-CAD	90 (29.5%)	56 (37.6%)	59 (32.9%)	>0.05
BMI	28.3±4.2	28.3± 4.2	28.7±3.8	>0.05

**<0.001 LAD#LCx, *<0.05 LAD#LCx, $<0.05 RCA#LCx, $$<0.001 RCA#LCx, ^<0.05 LAD# RCA, ^^<0.001 LAD# RCA

Results

166 (31.4%) of our patients had single vessel disease, 111 (21%) had 2 vessel disease, 83 (15.7%) with 3 vessel disease and 169 (31.9%) had non-significant lesion.

Demographic data of the patients are in Table 1, with significantly higher incidence of significant LAD lesion 305((57.5%), followed by RAC 181 (34.2%), while LCx showed the least incidence148 (27.9%).

LCx and RCA groups had older age (57.1±9.9& 57.7±9.9) compared to those in LAD group (55.7 ±10.6), with no significant difference; p>0.05.

Hypertension was significantly more prevalent in LCx 101 (68.2%) and RCA 117 (64.6%) groups compared to LAD group 156 (54.1%), P<0.05.

Significantly higher incidence of smoking in LCx 84 (56.8) group compared to LAD136 (44.4%) group, P<0.05.

Diabetes mellitus was significantly more prevalent in LCx group 75 (50.7%) compared to LAD group128 (42%), P<0.05.

Table 2 showed the angiographic characters of the groups:

Between single vessel disease, LAD was significantly more prevalent 41%, followed by RCA 27.1% (P<0.05) and LCx 13.3% (P<0.001).

52.4% of LAD lesions were proximal compared to 42.8% in LCx and 36.7% in RCA, P<0.05 to all.

Incidence of mid lesion was significantly more in RCA 50% compared to LCx42.9% P<0.05.

Distal lesion was significantly more prevalent in RCA 20.4 % compared to LCx 11.9 % (P<0.05) and LAD 9.5% (P<0.001).

Incidence of ectataic lesion was significantly higher in RCA 6.1% and LCx 7% compared to LAD 3.2%, P<0.05.

Incidence of multiple lesions was significantly more prevalent in LAD vessel 22.1% followed by LCx 7.2% and RCA2%, P<0.001.

Tables 3& 4 showed the demographic and angiographic characters of the culprit vessels:

Significantly higher incidence of culprit LAD lesion 27.6% compared to RCA 7% and LCx 5.9 % between all patients (p<0.001 to all).

LAD with significant lesion was significantly more prone to be the culprit vessel 47.5%, compared to LCx 21.1% and RCA 20.4%(p<0.001).

Patients with culprit LAD and those with culprit RCA showed significantly more male predominance (88.4% and 88.6% respectively) compared to those with LCx 77.4% culprit, (P<0.05).

Table 2: Angiographic characters.

Variables	LAD 305 (57.7%)	Lcx 148 (28%)	RCA 181(34.2%)	P
As a Culprit vessel	47.5% of LAD**,^^ & 27.6%of all pats.	21.1 % of Lcx& 5.9% of all pats	20.4% of RCA& 7% of all pats	**<0.001 LAD#Lcx , ^^<0.001 LAD# RCA
SVD	41%**,^	13.3%	27.1%$	**<0.001 LAD# Lcx, ^<0.001 LAD# RCA, $<0.05 RCA#Lcx
Proximal lesion	51.4%*,^	42.8%	36.7%	*<0.05 LAD#Lcx ^<0.05LAD# RCA
Mid lesion	44.4%	42.9%	50%$	$<0.05 RCA#Lcx
Distal lesion	9.5%	11.9%	20.4%^^,$	$<0.05 RCA#Lcx , ^^<0.001 LAD# RCA
Ectatatic	3.2%	7%*	6.1^	*<0.05 LAD#Lcx , ^<0.05 LAD# RCA
multiple	22.1 %**,^^	7.2%	2%	**<0.001 LAD#Lcx , ^^<0.001 LAD# RCA

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Table 3: Culprit vessels.

Variables	LAD	Lcx	RCA	P
	146\529 (27.6%)	31\529 (5.9%)	37\529 (7%)	
Age	52± 9.5	54± 8.9	53.3±10.1	>0.05
Gender : males	88.4%*	0.774	88.6%$	
females	0.116	0.226	0.114	
Hypercholesterolemia	0.644	0.613	76.6%$,^	
Hypertension	0.411	80.6%**,$$	62.2%^	
Smokers	0.425	71%**	70.3%^^	
DM	0.356	58.1%**,$	43.2%^	
FH-CAD	0.315	54.8%**,$	40.5%^	
BMI	28.5 ±4.5	29.5±2.6	28.8±2.5	>0.05
STE-ACS	76.7% **,^^	0.435	0.444	
NSTE-ACS	0.233	56.5%**	55.6%^^	
Troponin level	6.1±7.2**,^^	0.01±0	0.17±0.2 $	

Hypercholesterolemia was significantly more prevalent between those with culprit RCA 76.4% compared to those with LCx 61.3 % and LAD 64.4%, P<0.05.

Incidence of hypertension was significantly more in culprit LCx 80.6% followed by RCA 62.2% (p<0.05) compared to LAD 41.1%, P<0.001.

Incidence of smoking was significantly more in culprit LCx 71% and RCA 70.3% compared to LAD 42.5%, P<0.001.

Incidence of diabetes mellitus was significantly more in culprit LCx 58.1% compared to RCA 43.2 % (p<0.05) and LAD 35.6% (P<0.001), and showed significant differences in culprit RCA compared to LAD (P<0.05) .

Family history of CAD was significantly more in culprit LCx 54.8 % compared to RCA 43.2 % (P<0.05) and LAD 31.5% (P<0.001), and showed significant differences in culprit RCA compared to LAD(P<0.05) .

Proximal culprit LAD 63.5% and RCA 55.6% had significantly higher incidence compared to LCx38.5%, p<0.05.

Mid culprit LCx 53.9% had significantly higher incidence compared to RCA 38.9% and LAD 36.2%, p<0.05.

Distal culprit LCx 7.7% had significantly higher incidence compared to RCA 3%, p<0.05.

STE-ACS was significantly more prevalent in culprit LAD 76.7% compared to RCA 44.4% and LCx 43.5%., P<0.001

Table 4: Coronary characteristics of the Culprit vessel.

Variables	LAD	Lcx	RCA	P
SVD	84.9%**	0.611	79.2%$	
Proximal lesion	63.5%*	0.385	55.6%$	
Mid lesion	0.362	53.9%*,^	0.389	
Distal lesion	0.03	7.7%*,$	0	
Multiple	12.1%*,$	0.077	0.056	

NSTE-ACS was significantly more prevalent in culprit LCx 56.5% and RCA 55.6% compared to LAD 23.3%, P<0.001.

Troponin level was significantly higher in those with culprit LAD 6.1±7.2 vs, 0.17±0.2 in RCA and 0.01 in LCx, P<0.001.

Discussion

Atherosclerosis is a complex, involving endothelial cells, smooth muscle cells and arterial extracellular matrix macromolecules, which undergo a phenotypic switch from their normal physiological function to a pathological one under the effect of local flow hemodynamics and inflammatory mechanisms [15].

The identification and control of coronary risk factors, such as diabetes, smoking, arterial hypertension and hyperlipidemia, is the cornerstone of preventative strategies [1].

Local and systemic factors coexist to initiate atherosclerosis and allow its progress; atherosclerotic lesions tend to form at certain arterial segments despite the systemic effect of risk factors on all arteries.

Several studies have now validated the low shear stress hypothesis of atherosclerosis [15,16]. Low/oscillatory endothelial shear stress is an important key factor in atherosclerosis initiation and progression as it is implicated in the critical switch from normal physiological to a pathological one. Laminar flow and unidirectional high shear stress are athero-protective [17].

These observations highlight the importance of local hemodynamic conditions in susceptible individuals with CAD risk factors.

There were no difference regarding age and gender among all groups with significant coronary lesion. There were male predominance in all groups regarding presence of culprit lesions, in agreement with Ozaki et al, [10] with fewer females in LAD and RCA compared to LCx while Halim SA , et al [16] found fewer women among patients with a culprit LCx compared with those with a culprit LAD or RCA as he studied those with NSTE-ACS only.

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LAD lesion was more prevalent, it tends to carry multiple lesions mainly proximal that were more prone to be sites of culprit lesions and to be presented as STE-ACS than the other vessels, in agreement with Katritsis et al, [18] and with Wang et al, [19].

RCA significant lesions were more frequent in the mid segments followed by the proximal one, but the proximal segment was more prone to be the site of culprit lesion.

LCx was the least vessel to carry significant lesion which were equally distributed between proximal and mid segments, the mid segments were more prone to have culprit lesions this was in agreement with Katritsis DG, et al, [18] while Wang et al, [19] detected that LCx thrombosis was more frequent in the proximal segment. Culprit LCx presented in most cases as NSTE-ACS, these results were in correlation with Antoni ML et al, [9], From et al, [20] and Ghanim, et al, [21] this could be due to lesser LCx longitudinal strain that makes the plaque less prone to rupture, with lesser transmural ischemia.

The LAD, most of the RCA, and the mid to distal LCx and its branches pass along the LV longitudinal axis. The proximal-to-mid LCx, pass in the atrioventricular groove along the circumferential axis of the base of the heart. This anatomy can explain why there is a higher shear stress on the proximal LAD and RCA with predominance of the culprit lesions in their proximal segments and a lower incidence of the proximal LCx as a site of culprit lesion due to higher RCA and LAD systolic shortening [8].

In our studytroponin level was significantly higher in those with culprit LAD than RCA and was the least in LCx, this was in disagreement with Halim SA, et al, study [16] and Antoni ML et al, [9] who demonstrated that patients with a culprit lesion in the LAD and LCx had significantly higher-peak cardiac enzymes compared with patients with culprit lesions in the RCA this discrepancy may be due to their selection of patients with NSTE-ACS only. In our study, LCx as a culprit had a higher incidence of NSTE-ACS which incorporated those with unstable angina who had no enzyme elevation. LAD as an artery (more prone to carry culprit lesion) supplies a larger area of the heart, with a higher incidence of STE-ACS with a higher enzyme release.

Study Limitations

Our analysis depended on the two-dimensional coronary angiogram, that lakes good assessment of spatial anatomic relationships and not study the characters of the vulnerable plaques. We did not study the TIMI flow or the SYNTAX score.

Conclusion

LAD tends to carry more than one culprit lesion more to be proximal. Risk factors responsible for instability and sheer stress (uncontrolled DM, uncontrolled hypertension, heavy smoking) were more prevalent between patients with LCx as a culprit followed by RCA in Egyptian; this may draw the attention for aggressive control of these risk factors to reduce vulnerability in these patients.

Recommendations

Further studies of human coronary anatomy using IVUS, as well as in-depth analysis of coronary hemodynamics with study of the coronary flow reserve, TIMI and myocardial blush, are necessary for characterization and identification of coronary lesions.

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