Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2023.243706.2971 Volume 30, Issue 3, May 2024

Manuscript ID ZUMJ-2310-2971 (R1)
DOI 10.21608/ZUMJ.2024.270576.3175

ORIGINAL ARTICLE

Electrocardiographic Distinction of Culprit Artery in Patients with Acute Inferior ST Segment Elevation Myocardial Infarction and Multivessel Disease

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Submit Date 20-10-2023
Revise Date 27-10-2023
Accept Date 2023-10-30

ABSTRACT

Background: Myocardial infarction (MI) is leading cause of death and disability globally. The electrocardiogram (ECG) is a critical component of the diagnostic workup for individuals with suspected MI. Electrocardiograms are essential in identifying the type and location of acute myocardial infarction. This study aimed to clarify the value of ECG for identification of the culprit artery in acute inferior STEMI with multivessel coronary artery disease. **Methods:** This study included 55 patients with acute inferior STEMI who were divided into two groups based on the culprit coronary artery identification at time of primary percutaneous coronary intervention (PPCI). Electrocardiographic analysis was carried out to be compared with angiographic findings that correlate with culprit artery location (either right coronary artery (RCA) or left circumflex (LCX). Group I: Included 43 patients, (culprit was RCA), Group II: Included 12 patients, (culprit was LCX). **Results:** After comparing the findings in electrocardiographic leads, it was evident that the degree of ST segment deviation in leads II and V6 was statistically significantly higher in LCX group VS RCA group (1.9±0.8mm VS1.5±1.1mm, p-value=0.049 and 1.1±1.0VS -0.4±1.0 p-value <0.001 respectively). Also; the degree of ST segment deviation in leads aVL was statistically significantly higher in RCA group vs LCX group (-2.4±0.9mm VS -1.3±1.4mm, p-value=0.001). **Conclusions:** In patients with acute inferior STEMI and multivessel coronary artery disease where LCX is the infarct related artery (IRA), presenting ECG is more likely to reflect less pronounced reciprocal ST depression in aVL and more marked STE in V6.

Keywords: Culprit artery; Electrocardiography; Myocardial Infarction; Multivessel Disease

INTRODUCTION

Acute myocardial infarction (AMI), sometimes known as a heart attack, is a kind of coronary heart disease (CHD) that has a high fatality rate, particularly in developed countries [1]. Even while high-income nations have shown a decline in AMI-related mortality, Low- and middle-income countries are showing a rising trend [2]. MI is frequently brought on by the coronary atherosclerotic plaque rupture, erosion, or fissuring which result in inadequate oxygen and blood supply to the heart muscle [3].

MI is classified into five categories: septal, lateral, anterior, posterior, and inferior. Furthermore, myocardial infarctions (MIs) are typically classified as either non-ST elevation MI (NSTEMI) or ST elevation MI (STEMI) based on the ST elevation on the electrocardiogram (ECG) [4].

Patients frequently have transient cardiac pacing, right ventricular infarction, and serious hemodynamic problems that can result in shock, arrhythmias, and even death when RCA is determined to be the IRA [5].

For patients with LCX blockage, the prognosis is substantially better although patients may have significant mitral regurgitation owing to papillary muscle dysfunction with significant morbidity and mortality [6]

Several algorithms and ECG changes (more than 26 criteria and three algorithms) during acute injury phase of IAMI have been implemented to identify the culprit coronary artery (either RCA or LCX) with varying accuracies [7].

Aim of the work

The aim of this work was to study the value of ECG for identification of culprit artery in acute inferior STEMI with multivessel coronary artery disease

METHODS

This comparative, prospective, cross-sectional study included 55 patients with acute inferior STEMI who were admitted for coronary artery primary percutaneous intervention at Cardiovascular Department, Zagazig University Hospitals and Al-Ahrar Teaching Hospital in the period between February 2022 and April 2023.

Every patient provided written informed consent, and the study was authorized by the Zagazig University Faculty of Medicine's Research Ethical Committee (ZU-IRB:5775/5-1-2020). The study was carried out according to the Ethical code of the World Medical Association (Declaration of Helsinki) for studies including humans.

Inclusion criteria: acute myocardial infarction defined as typical prolonged ischemic chest pain >20 minutes associated with ST-segment elevation in two inferior leads (II, III, and/or aVF) ≥0.1mV. with culprit artery is either RCA or LCX referral to PPCI together with classic rise of cardiac biomarkers within 12 hours of the commencement of symptoms, the index ECG was taken.

Exclusion criteria: patients with history of percutaneous coronary intervention (PCI) or preceding left bundle branch block, ventricular pacing rhythm, and coronary bypass graft operation, acute anterior STEMI, acute inferior STEMI with single coronary artery disease (CAD), AMI brought on by an interruption in coronary flow because of invasive diagnosis and treatment, or another illness other than atherosclerosis.

Patients were divided into two groups based on the culprit coronary artery identification at time of PPCI.

Group I: included 43 patients, culprit was RCA.

Group II: included 12 patients, culprit was LCX.

All patients were subjected to:

Full history taking**,** including age, gender, presenting complaint (stressing on beginning of chest pain, characteristic, frequency, severity, duration, and causative relieving factors), important related symptoms (as dyspnea), and long-term medications.

1-General examination with a focus on weight and height for determining body mass index (BMI) and body surface area (BSA). Pulse, blood pressure and heart rate (on admission). Neck veins, and lower limbs are all examined.

2-Local examination of the chest and heart with special attention to signs of chamber enlargement, additional sounds and murmurs, presence of crepitations or diminished air entry.

3-Laboratory tests: kidney function test (initial creatinine level), cardiac enzymes (peak CKMB and peak highly sensitive troponin T) and hemoglobin level.

4-12 lead Electrocardiography: ECG was done on admission. The initial heart rate and rhythm were assessed. The presence of at least 1 mm of ST segment elevation in two or more continuous inferior leads in electrocardiogram (ECG). Calculation of number of leads with ST segment elevation. Right ventricular leads (V1, V2R, V3R, V4R, V5R and V6R) to diagnose probable RV infarction. All ECG were analyzed independently.

Echocardiographic analysis: for evaluation of left ventricular ejection fraction (LVEF), dimensions, wall motion score (WMS) and wall motion score index(WMSI).

Coronary angiographic examination: The catheterization labs of Zagazig University Hospitals and Al Ahrar Hospital, were used to conduct coronary angiography.

After PPCI patients were transferred to coronary care unit (CCU) where they were medicated by attending physician as per current STEMI guidelines.

In hospital outcome was reported including recorded death, arrhythmia, heart failure, reinfarction and heart block.

Statistical analysis:

The statistical program for social science (SPSS) version 24 was used to examine the data. The statistical information was presented as mean ±SD. The frequency and proportion of the qualitative data were reported. A discrete set of numbers' mean (average) is its central value. A set of values' dispersion is measured by the standard deviation (SD). When comparing two means, the t-test for significance was used. When comparing non-parametric data, the chi-square test was used. P-values ≤ 0.05 were used to determine statistical significance.

RESULTS

Demographic data analysis showed there was no statistically significant difference between both groups apart from smoking and BMI (table 1).

Table (2) showed that there was statistically significant increase in ST segment deviation in lead II and V6 in group II when compared with group I (p-value = 0.049) and < 0.001respectively). Lead aVL also showed statistically significant ST segment deviation in group I compared with group II (p-value = 0.001), as shown also in figure 1, 2.

On comparing some of the already set criteria of differentiating culprit artery in acute inferior STEMI, there were statistically increase in STE ≥ 0.1 in V6 or > STE III, STDV3/STEIII > 1.2 and STE II ≥ STE III in LCX patients when compared with RCA patients (p-value < 0.001, 0.005 and 0.005 respectively). There was also highly statistically significant increase in STE III > STE II and STD aVL > STD I in RCA patients when compared with LCX. (p-value < 0.001, < 0.001 respectively), as shown in table 3.

For identifying LCX culprit artery in acute inferior STEMI and multivessel disease, we used some of the most used criteria to discriminate the culprit artery and measured their diagnostic performance. STE ≥ 0.1 V6 or > STE III), (STDV3/STEIII > 1.2) and (STE II ≥ STE III) had a sensitivity of 58.3%, 33.3%, 83.3%, specificity of 93%, 95.4%, 88.4%, PPV of 70%, 66.7, 66.7%, NPV of 88.9%, 83.7%, 95% and accuracy of 85.5%, 69.1%, 87.3% respectively. For identifying RCA culprit artery, STD AVL > STD I and STE III > STE II, had a sensitivity of 63.6%, 86% specificity of 90.9%, 83.3%, PPV of 96.6%, 94.8%, NPV of 38.5%, 62.5% and accuracy of 69.1%, 85.5% respectively, as shown in table 4.

Table (5) shows that there was no statistically significant difference between both groups regarding assessed echocardiographic parameters which included dimensions, systolic function, and wall motion score index.

There was no statistically significant difference between the two groups in terms of the number of diseased vessels and the left main (LM) / proximal left anterior descending (LAD), however there was a highly statistically significant difference in terms of coronary dominance, as shown in table (6).

Table (1): Basic characteristics of the studied groups

		Group I RCA (N = 43)		Group II LCX (N = 12)			
Age (years)	**Mean**	56.3 ±		55.6±		T = 0.24	0.812 NS
	±SD	8.9		10.1			
Gender	**Male**	35	81.4%	9	75%	X^2 = 0.24	0.624 NS
	Female	8	18.6%	3	25%		
Risk factors	**DM**	12	27.9%	3	25%	X^2 = 0.04	0.842 NS
	HTN	16	37.2%	8	66.7%	X^2 = 3.3	0.069 NS
	Dyslipidemia	17	39.5%	6	50%	X^2 = 0.42	0.516 NS
	F. H. IHD	13	30.2%	4	33.3%	X^2 = 0.04	0.837 NS
	Smoking	34	79.1%	6	50%	**X^2 = 3.9**	**0.046 S**
BMI (kg/m²)	**Mean**	24.3±		27.7±		**T = 3.04**	**0.004 S**
	±SD	3.2		4.5			

HTN: hypertension, DM: diabetes mellitus, BMI: body mass index, T: Independent sample T test.

Table (2): Comparison between both groups regarding ST segment deviation on electrocardiography

ST segment deviation distance		Group I (N = 43)	Group II (N = 12)	MW	P-value
II (mm)	Mean	1.5 ±	1.9 ±	169.5	0.049 S
	±SD	1.1	0.8		
aVF (mm)	Mean	1.7 ±	1.7 ±	254	0.930 NS
	±SD	1.1	1.2		
III	Mean	2.5 ±	1.9 ±	179.5	0.096 NS

(mm)	±SD	1.3	1.4		
I (mm)	Mean	-0.6 ±	-0.3 ±	183.5	0.086 NS
	±SD	0.8	0.9		
aVL (mm)	Mean	-2.4 ±	-1.3 ±	106	0.001 S
	±SD	0.9	1.4		
V1 (mm)	Mean	-0.3 ±	0.0	198	0.09 NS
	±SD	0.8	0.0		
V2 (mm)	Mean	-0.9 ±	-0.4 ±	197.5	0.181 NS
	±SD	1.1	0.7		
V3 (mm)	Mean	-0.5 ±	-0.8 ±	243	0.739 NS
	±SD	1.3	1.3		
V4 (mm)	Mean	-0.4 ±	-0.3 ±	204	0.213 NS
	±SD	1.2	1.3		
V5 (mm)	Mean	-0.4 ±	-0.1 ±	180	0.078 NS
	±SD	1.0	1.4		
V6 (mm)	Mean	-0.4 ±	1.1 ±	66.5	< 0.001 HS
	±SD	1.0	1.0		

Table (3): Comparison between both groups regarding major set ST segment deviation parameters in literature

		Group I		Group II		Test	P-value
STE ≥ 0.1 V6 or > STE III	**No**	40	93%	5	41.7%	**X^2 = 16.6**	**< 0.001 HS**
	yes	3	7%	7	58.3%		
STE ≥ 0.1 V6 or > STE III	**Mean**	1.66±		1.28±		MW = 6.5	0.383 NS
	±SD	0.5		0.4			
STDV3/STEIII > 1.2	**No**	41	95.3%	8	66.7%	**X^2 = 7.9**	**0.005 S**
	yes	2	4.7%	4	33.3%		
STDV3/STEIII > 1.2	**Mean**	2.5±		2.1±		MW = 2.5	0.533 NS
	±SD	0.7		0.6			
STE III > STE II	**No**	6	14%	10	83.3%	**X^2 = 21.9**	**< 0.001 HS**
	yes	37	86%	2	16.7%		
STD AVL > STD I	**No**	15	34.9%	11	91.7%	**X^2 = 12.1**	**< 0.001 HS**
	yes	28	65.1%	1	8.3%		
STE II ≥ STE III	**No**	37	86%	2	16.7%	**X^2 = 21.9**	**< 0.001 HS**
	yes	6	14%	10	83.3%		

STE:ST segment elevation, STD:ST segment depression

Table (4): Sensitivity, specificity, positive, negative predictive values, and accuracy of ECG criteria for predicting culprit artery in acute inferior STEMI

(n = 55)	**STE ≥ 0.1 V6 or > STE III (LCX culprit)**	**STDV3/STEIII > 1.2 (LCX culprit)**	**STE III > STE II (RCA culprit)**	**STD AVL > STD I (RCA culprit)**	**STE II ≥ STE III (LCX culprit)**
True positive	7 (12.7%)	4 (7.3%)	37 (67.3%)	28 (50.9%)	10 (18.2%)
False positive	3 (5.5%)	2 (3.6%)	2 (3.6%)	1 (1.8%)	5 (9.1%)
True negative	40 (72.7%)	41 (74.5%)	10 (18.2%)	10 (18.2%)	38 (69.1%)
False negative	5 (9.1%)	8 (14.5%)	6 (10.9%)	16 (29.1%)	2 (3.6%)
Sensitivity	58.3%	33.3%	86%	63.6%	83.3%
Specificity	93%	95.4%	83.3%	90.9%	88.4%

Positive predictive value	70%	66.7%	94.8%	96.6%	66.7%
Negative predictive value	88.9%	83.7%	62.5%	38.5%	95%
Accuracy	85.5%	69.1%	85.5%	69.1%	87.3%

Table (5): Comparison between both groups regarding echocardiographic data

		Affected vessels		MW	P-value
		RCA (N = 42)	LCX (N = 12)		
WMS	**Mean**	21.3 ±	20.2±	184	0.123 NS
	±SD	2.5	1.3		
WMSI	**Mean**	1.2 ±	1.2 ±	184	0.123 NS
	±SD	0.1	0.1		
LVESD mm	**Mean**	34.3 ±	34.0±	247	0.822 NS
	±SD	7.3	3.7		
LVEDD mm	**Mean**	50.8 ±	50.9±	232	0.595 NS
	±SD	9.8	3.7		
EF %	**Mean**	55.6 ±	58.4±	189.5	0.162 NS
	±SD	6.9	5.4		

MW: Mann Whitney U test, WMS: wall motion score, WMSI: wall motion score index, LVEDD: left ventricle end diastole dimensions, LVESD: left ventricle end systole dimension, EF: ejection fraction

Table (6): Comparison between both groups regarding coronary angiographic data

		Group I (N = 43)		Group II (N = 12)			
Affected vessels	**2 vessels**	28	65.1%	9	75%	$X^2 = 0.41$	0.519 NS
	3 vessels	15	34.9%	3	25%		
Coronary dominance	**Right**	35	81.4%	0	0%	**$X^2 = 27.1$**	**< 0.001 HS**
	Left	2	4.7%	4	33.3%		
	Codominant	6	14%	8	66.7%		
LM/proximal LAD	**No**	29	67.4%	8	66.7%	$X^2 = 0.003$	0.960 NS
	Yes	14	32.6%	4	33.3%		

LM: left main coronary artery, LAD: left anterior descending artery

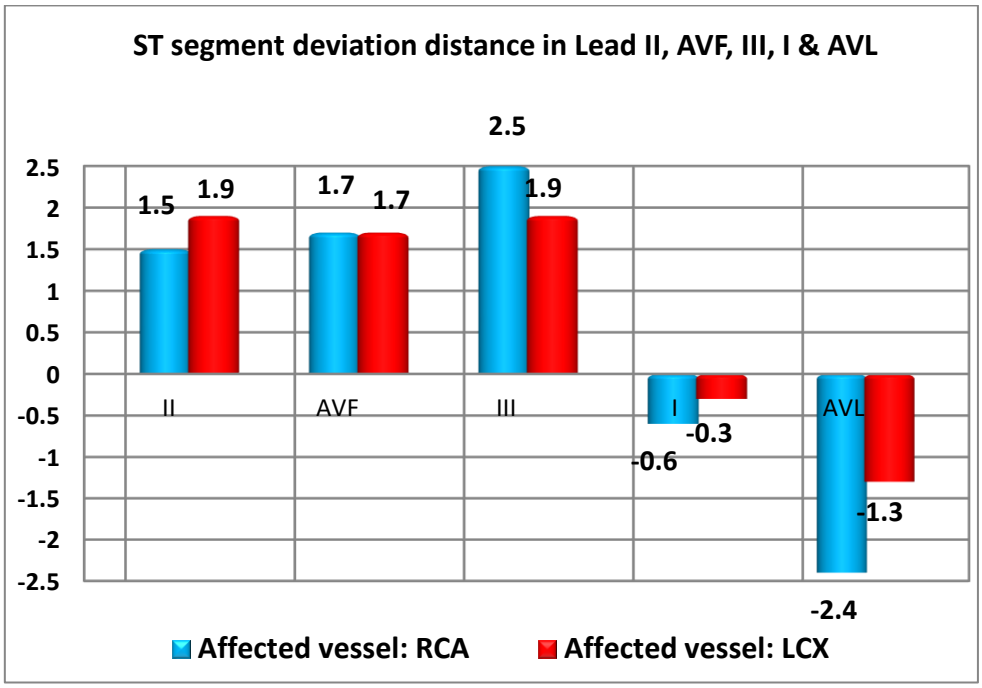

Figure (1): Comparison between both groups regarding ST Segment deviation in lead II, III, AVF, I& AVL.

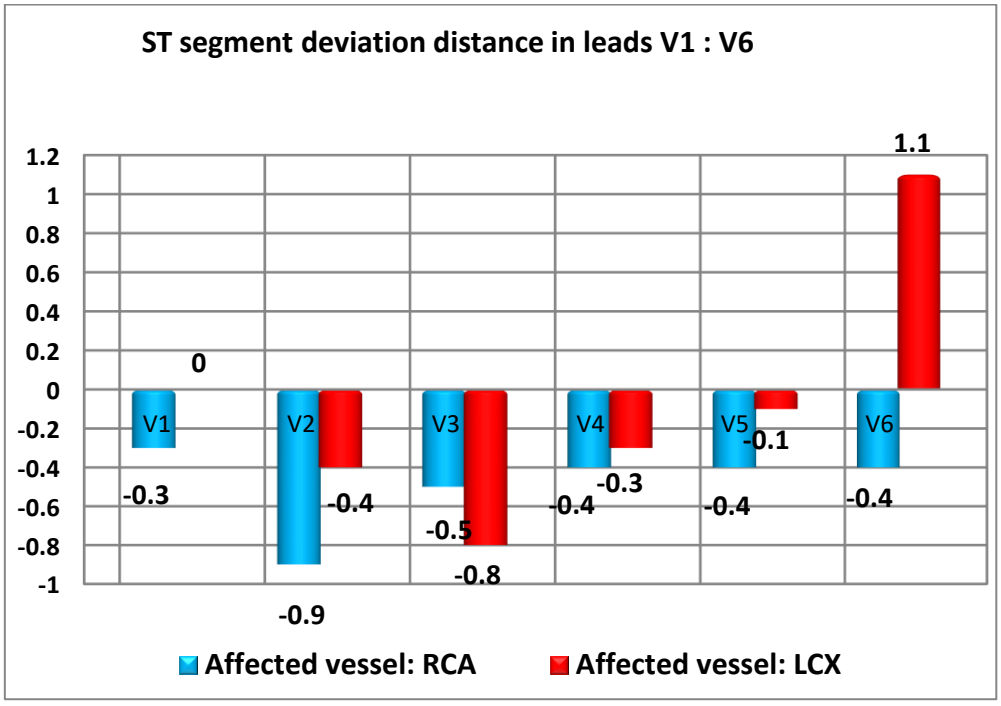

Figure (2): Comparison between both groups regarding ST segment deviation in lead V1-V6.

DISCUSSION

Acute coronary syndrome still poses a major health problem worldwide with many complications causing both morbidity and mortality [8].

Acute coronary artery blockage in patients with multivessel coronary artery disease (CAD) may be linked to distant ischemia, which could alter the pattern of ST-segment alterations. In addition, some cases may show persistent complete blockage of one main coronary artery in the epicardial and acute thrombotic occlusion in another one. Rapid identification of the culprit (IRA) artery is of crucial importance to ensure timely reperfusion as can be suggested from 12 lead surface ECG [9].

Early recognition of whether the culprit artery is the RCA or the LCX may facilitate management and, in some instances, may allow complications to be avoided [10]. For example, if the presenting ECG suggests RCA culprit artery occlusion, treating physician may try to avoid use of beta blockers before reperfusion to avoid possible brady-arrhythmic complications of early AV block. On the other hand, attending physician may choose to restrict early volume expansion in case of significant mitral regurgitation in cases of LCX culprit arteries.

In our study, there was a statistically significant (p-value = 0.046) increase in smoking percentage in RCA patients (34 %, 79.1%) compared to LCX patients (6 %, 50%) and a statistically significant (p-value = 0.004) increase in BMI in LCX patients (27.7 ±4.5) compared to RCA patients (24.3 ± 3.2). However, no statistically significant change (p-value = 0.383) was discovered between RCA and LCX patients as regard age, gender, DM, HTN, dyslipidemia and family history of IHD. This was in agreement with **Li et al.,** [1] who studied electrocardiographic changes in RCA cases versus LCX cases and found no statistical significance among studied demographic and risk factors data, while statistical significance for smoking and BMI could be attributed to small sample size in our study.

There was no statistically significant difference (p-value > 0.05) between the two groups in our investigation with relation to ST segment deviation distance in the following leads (AVF, III, I, V1, V2, V3, V4 and V5). However Statistically significant (p-value = 0.049) increased for lead II ST segment deviation in LCX patients (1.9 ± 0.8) when compared with RCA patients (1.5 ± 1.1). Highly statistically significant difference (p-value < 0.001) of increased lead V6 ST segment deviation in LCX patients (1.1 ± 1.0) when compared with RCA patients (- 0.4 ± 1.0) and statistically significant difference (p-value = 0.001) of increased lead aVL ST segment deviation in RCA patients (-2.4 ± 0.9) when compared with LCX patients (-1.3 ± 1.4).

This was in disagreement with the study of **Li et al.,** [1], among 240 patients of inferior MI according to IRA in using coronary angiography, it was determined that LCX was the IRA in 63 patients and that RCA was the cause of AIMI in 177 others. The analysis results showed that there were clear differences between the two groups in terms of ST-segment deviation, I, III–II, III, AVL, AVF, AVL-I, V1, and V6. ECG ST deviation, II, AVR, V2, V3, V4, and V5, however, revealed values that were comparable across the two groups (all P>.05) this can be explained by the fact that they included single vessel disease only.

We found that there was a strong statistically significant (p-value < 0.001) increased percentage of positive STE ≥ 0.1 V6 or > STE III in LCX patients (7 patients, 58.3%) when compared with RCA patients (3 patients, 7%), and no statistically significant difference (p-value = 0.383) between RCA and LCX patients as regard STE ≥ 0.1 V6 or > STE III, with a sensitivity of 58.3%, specificity 93%, PPV 70%, NPV 88.9% and accuracy of 85.5%. This result was consistent with a study by **Gulizia et al** [11], which indicated that whereas the LCX illness in ST elevation in III≤V6 (n-12; 71%) (p-0.0001) was substantially greater than the RCA in lead III>V6 (n-37; 82%) (p=0.0001). With awareness, precision, optimism and negative predictive values were 90%, 63%, 84%, 75% and 63%, 90%, 75%, 84% respectively.

In our study, there was statistically significant (p-value = 0.005) increased percentage of STDV3/STEIII > 1.2 in LCX patients (4 patients, 33.3%) when compared with RCA patients (2 patients, 4.7%) and no statistically significant difference (p-value = 0.533) between RCA and LCX patients as regard STDV3/STEIII > 1.2 distance, with sensitivity 33.3%, specificity 95.4%, PPV 66.7%, NPV 83.7% and accuracy 69.1%. These results agreed with study of **Vives-Borrás et al.,** [12] who concluded that STDV3/STEIII > 1.2 had the sensitivity of 35%, specificity of 95% and accuracy of 66%.

We found that there was a strong statistically significant (p-value < 0.001) increased percentage of STE III > STE II in RCA patients (37 patients, 86%) in contrast to two patients, 16.7% of LCX patients, and statistically significant (p-value = 0.002) increased ratio of STE III > STE II in RCA patients (1.5 ± 0.8) when compared with LCX patients (0.9 ± 0.5).These results was in agreement with study of **Vives-Borrás et al** [13] who showed that STE in III>II with sensitivity of 92%, specificity of 46%,accuracyof 70% for prediction of RCA occlusion.

The main limitation of this study is the small number of patients especially in the LCX group. This could have biased our results. We did not consider the intra-observer variability in our ECG analysis. This could have affected the accuracy of our statistical analysis. In our patients and methods, we excluded patients with previous coronary events or revascularization. Future studies should include those patients to reflect performance of ECG discriminatory power in actual everyday Cath lab scenarios.

Future multicenter larger studies are recommended with the aim to recruit larger sample size to better reflect accurate statistics. Inter- and intra- observer variability should be considered and safeguarded as a biasing factor. Unifying time from onset of chest pain to first ECG (defined as the presenting ECG) is crucial in ST shifts analysis. It is known that ST deviation evolves over hours following coronary artery occlusion, this could exclude a major biasing factor in electrocardiographic analysis.

CONCLUSIONS

In patients with multivessel coronary artery disease and acute inferior STEMI where LCX is the IRA, presenting ECG is more likely to reflect less pronounced reciprocal ST depression in aVL and more marked STE in V6. Previously set ECG criteria of discrimination of IRA yielded variable sensitivity, specificity, and accuracy.

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To Cite :

Aly Ahmed, A., Al –Zayat, A., Abdelsamei, M., Allam, M., Mohamed, M. Electrocardiographic Distinction of Culprit Artery in Patients with Acute Inferior ST Segment Elevation Myocardial Infarction and Multivessel Disease. Zagazig University Medical Journal, 2024; (927-934): -. doi: 10.21608/zumj.2023.243706.2971