International Journal of Cardiology Sciences

ISSN Print: 2664-9020
ISSN Online: 2664-9039
Impact Factor: RJIF 5.42
IJCS 2024; 6(1): 01-07
www.cardiologyjournals.net
Received: 01-11-2023
Accepted: 07-12-2023

Ahmed Mohamed El Sayed
Resident of Cardiology, Cardiovascular Medicine Department, Faculty of Medicine, Tanta University, Tanta, Egypt

Aliaa Elsayed Shaban
Lecturer of Cardiology, Cardiovascular Medicine Department, Faculty of Medicine, Tanta University, Tanta, Egypt

Ehab Abd Ellatif Elgendy
Assistant Professor of Cardiology, Cardiovascular Medicine Department, Faculty of Medicine, Tanta University, Tanta, Egypt

Ayman Mohammed El Saeid
Professor of Cardiology, Cardiovascular Medicine Department, Faculty of Medicine, Tanta University, Tanta, Egypt

Corresponding Author:
Ahmed Mohamed El Sayed
Resident of Cardiology, Cardiovascular Medicine Department, Faculty of Medicine, Tanta University, Tanta, Egypt

Correlation between St segment elevation in the 12-lead ECG and the culprit coronary artery in patients with acute stemi undergoing primary PCI

Ahmed Mohamed El Sayed, Aliaa Elsayed Shaban, Ehab Abd Ellatif Elgendy and Ayman Mohammed El Saeid

DOI: https://doi.org/10.33545/26649020.2024.v6.i1a.42

Abstract
Background: Use of the Electrocardiogram (ECG) is necessary for early assessment of people believed to have acute coronary syndrome (ACS). This study sought to determine whether there was any relationship between ST segment elevation in the 12-lead ECG and the culprit coronary artery in patients with acute ST-segment elevation myocardial infarction (STEMI) having primary PCI.
Methods: This cohort study was carried out on 100 patients with STEMI experiencing primary PCI. 12-lead ECG and Echocardiography were performed to all patients.
Results: ECG findings in different types of myocardial infarctions are significant predictors of culprit artery described by angiogram. The ECG findings of LAD had a sensitivity of 92.45%, specificity of 87.23%, PPV of 89.1% and NPV of 91.1%. The ECG findings of RCA had a sensitivity of 73.3%, specificity of 97.1%, PPV of 91.7% and NPV of 89.5%. The ECG findings of LCx had a sensitivity of 58.8%, specificity of 86.7%, PPV of 47.6% and NPV of 91.1%.
Conclusions: The ECG findings of RCA, LAD, and LCx were significant predictors of RCA, LAD, and LCx culprit artery by angiogram.

Keywords: 12-Lead ECG, culprit coronary artery, STEMI, primary PCI

Introduction

According to the most recent recommendations, the electrocardiogram (ECG) is currently the most important diagnostic tool for the treatment of acute coronary syndromes, with the initial ECG and clinical symptoms serving as a precursor to the subsequent urgent steps [1].

The ECG from the acute period, especially in ST-segment elevation Acute Coronary Syndrome (ACS), contains crucial details regarding the location and size of the area at risk, assisting in the choice of the most effective therapy for the specific patient [2]. To improve clinical decision-making and customise reperfusion therapy, the use of the ECG to identify the culprit artery and even the location of the culprit lesion inside the infarct-related artery could provide clinically valuable information [3].

Coronary artery disease (CAD) primarily describes coronary atherosclerotic disease, which causes severe coronary artery constriction and results in insufficient blood flow to the heart muscle (myocardium). Unstable angina, non-ST-segment elevation myocardial infarction, and ST-segment elevation myocardial infarction (STEMI) are among the acute presentations of CAD that are included in ACSs [4].

In the majority of STEMI instances, a thrombus (a blood clot) that formed on a coronary atherosclerotic plaque causes an epicardial coronary artery to become completely blocked, leading to transmural myocardial ischaemia. Patients experiencing chest discomfort and sustained ST-segment elevation in two or more physically contiguous ECG leads are thought to have STEMI [5].

Patients are divided into one of three groups based on their 12-lead ECG results: Those who have a new bundle branch block or an elevated ST segment (suspicious for acute injury and a possible candidate for acute reperfusion therapy with thrombolytics or primary PCI), those who have an ECG that is deemed to be non-diagnostic or normal and those who exhibit ST segment depression or T wave inversion (suspicious for ischemia) [6].

Acute myocardial infarction cannot be ruled out by a normal ECG [6]. Pre-hospital 12-lead ECG interpretation has been proven to be critical for the management of patients with suspected STEMI [7]. Early detection increases the likelihood of a positive outcome and shortens the time it takes to perform percutaneous coronary intervention (PCI) or fibrinolysis to quickly restore blood flow [8].

This study sought to determine the relationship between ST segment elevation in the 12 leads of the ECG and the responsible coronary artery in patients with acute STEMI receiving primary PCI.

Patients and Methods

One hundred patients with STEMI who were receiving primary PCI and were between the ages of 50 and 68 were included in this cohort study.

After receiving approval from Tanta University Hospitals' Ethical Committee, the study was carried out. All patients provided written, fully informed consent.

Exclusion criteria were complete left BBB, ventricular paced rhythm, LVH in ECG, previous history of MI, previous cardiac surgery, coronary angiography not revolving significant occlusion and significant stenosis in more than one coronary artery.

All patients underwent a comprehensive history review, physical examination, general and cardiac examination, 12-lead ECG, and baseline venous blood sampling for (Random blood glucose, hemoglobin, serum creatinine and GFR), ccoronary angiography, primary PCI, echocardiography (ECG), assess systolic, diastolic, and valvular functions.

Electrocardiogram (ECG)

The standard test for measuring the heart's electrical function. A 12-lead ECG typically has electrodes implanted on the left arm, right leg, and both legs. Another set of electrodes is positioned on the left and right sides of the sternum, between the fourth and fifth ribs. Between these two electrodes on the fourth intercostal space, one electrode is placed [9].

The mid-clavicular line, a hypothetical reference line that extends downward from the midpoint of the clavicle, is the location of the eighth electrode, which is positioned between the fifth and sixth ribs. The ninth electrode is positioned in the anterior axillary line, or the hypothetical reference line that runs southward from the place where the collarbone and arm meet, which is in line horizontally with the eighth electrode.

The tenth and eleventh electrodes are positioned horizontally on the same line as the eighth and ninth electrodes, but this electrode is orientated with the midaxillary line, which is the imaginary line drawn straight down from the patient's armpit [9]. Two observers independently performed the measurements on both healthy volunteers and diabetes patients (C. F. M. and D. V.).

Echocardiography: Performed to all patients and control group the ECG was recorded simultaneously. Electronic routine At end-expiratory apnea, grayscale two-dimensional cine loops from three consecutive heartbeats were recorded using typical parasternal long-axis view, three apical views, and depths of 12–14 cm at mean frame rates of 67±8 frames/sec.

The sector width was adjusted to maximize frame rate and enable full cardiac imaging. According to the most recent American Society of Echocardiography/European Association of Cardiovascular Imaging recommendations, standard LV measures were taken [10]. The left atrial volume and left ventricular ejection fraction (LVEF) were computed using the modified biplane Simpson's method utilizing the apical 2- and 4-chamber views at the ventricular end-systole, and then the results were normalized to body surface area. With obesity into account, LV mass was linked to both body surface area and height 2.7.

Utilizing a pulsed-wave doppler placed at the LV outflow tract, the LV stroke volume was calculated using the velocity-time integral. Utilizing pulsed-wave Doppler recording from the apical four-chamber view, the early diastolic (E) and atrial wave velocities as well as the E-wave deceleration time were determined. Early diastolic velocity (E') determined from the septal mitral annulus using spectral pulsed-wave doppler was calculated, and the LV filling pressure was estimated utilizing the E/E' ratio [11].

Statistical analysis

The SPSS v26 statistical analysis program was utilized (IBM Inc., Chicago, IL, USA). Histograms and Shapiro-Wilks test were employed to determine the normality of data distribution. The mean and standard deviation were employed to depict quantitative parametric data (SD). Interquartile range and median were employed to depict non-parametric quantitative data (IQR). Qualitative variables were reported as frequency and percentage (%).

Results

The study's participants were between the ages of 50 and 68. 61 (61.0%) of the studied patients were males and 39 (39.0%) were females. The average value of BMI of the studied patients was 26.83±3.19 kg/m^2. Regarding the clinical data of the studied patients, the average value of SBP was 131.12±12.73, DBP was 75.21±9.93, and pulse rate was 78.60±8.33. Regarding the laboratory data of the studied patients, the average value of Hb was 13.07±1.24, for PLT was 215.78±53.42, for WBCs was 6.16±1.28, for S.cr was 0.969±0.18, for AST was 19.53±4.28, for ALT was 22.60±7.83, for TC was 195.07±27.21, for TGS was 140.88±22.49, for LDL was 117.29±8.72, and for HDL was 49.39±6.54. The troponin level was normal in 34 (34.0%) patients and was elevated in 66 (66.0%) patients. Regarding the risk factors of the studied patients, 35 (35.0%) patients had HTN, 22 (22.0%) patients had DM, 19 (19.0%) patients had dyslipidaemia, 33 (33.0%) patients were smokers, and 12 (12.0%) patients had family history of dyslipidaemia. Table 1.

Table 1: Demographic, clinical, laboratory data and risk factors among the studied groups

Patients group (n=100)		
Demographic data		
Age (Years)		59.23±4.92
Sex	Male	61 (61.0%)
	Female	39 (39.0%)

BMI		26.83±3.19
Clinical data		
SBP		131.12±12.73
DBP		75.21±9.93
Pulse		78.60±8.33
Laboratory data		
Hb		13.07±1.24
PLT		215.78±53.42
WBCs		6.16±1.28
S.cr		0.969±0.18
AST		19.53±4.28
ALT		22.60±7.83
TC		195.07±27.21
TGS		140.88±22.49
LDL		117.29±8.72
HDL		49.39±6.54
Troponin	Normal	34 (34.0%)
	Elevated	66 (66.0%)
Risk factors		
HTN		35 (35.0%)
DM		22 (22.0%)
Dyslipidemia		19 (19.0%)
Smoking		33 (33.0%)
Family history		12 (12.0%)

Data are shown as mean ± SD or frequency (%). BMI: Body mass index. SBP: Systolic blood pressure, DBP: Diastolic blood pressure. Hb: Hemoglobin, PLT: Platelets, WBCs: White blood cells, S.cr: Serum creatinine, AST: Aspartate transaminase, ALT: Alanine transaminase, TC: Total cholesterol, TGS: Triglycerides, LDL: Low density lipoprotein, HDL: High density lipoprotein. HTN: Hypertension, DM: Diabetes mellitus.

Regarding the type of infarction by ECG, it was anterior MI in 26 (26.0%) patients, inferior MI in 26 (26.0%) patients, anterolateral MI in 19 (19.0%) patients, inferolateral MI in 14 (14.0%) patients, and Antero septal MI in 15 (15.0%) patients. The most common sites of infarction in the studied patients were V3 and V4 leads each in 60 (60%) patients followed by V5 in 41 (41%) patients. The least common sites of infarction were I and aVL each in 15 (15%) patients. According to ECG findings the lesion was in LAD artery in 55 (55%) patients, in RCA in 24 (24%) patients, and in LCx in 21 (21%) patients. Table 2

Table 2: ECG among the studied group

Patients group (n=100)		
ECG		
Type of Infarction	Anterior MI	26 (26.0%)
	Inferior MI	26 (26.0%)
	Anterolateral MI	19 (19.0%)
	Inferolateral MI	14 (14.0%)
	Antero septal MI	15 (15.0%)
Size of Infarction (mm)		5 (3-6)
Site of infarction	I	15 (15%)
	II	40 (40%)
	III	40 (40%)
	aVF	40 (40%)
	aVL	15 (15%)
	V1	19 (19%)
	V2	31 (31%)
	V3	60 (60%)
	V4	60 (60%)
	V5	41 (41%)
	V6	29 (29%)
Correlation	LAD	55 (55%)
	RCA	24 (24%)
	LCx	21 (21%)

Data are presented as frequency (%) or median (IQR). ECG: Electrocardiogram, MI: Myocardial infarction, LAD: Left anterior descending, RCA: Right coronary artery, LCx: Circumflex.

The culprit artery by angiogram was LAD in 53 (53%) patients of them 23 (43.4%) patients were proximal, and 30 (56.6%) patients were distal, was RCA in 30 (30%) patients of them 23 (76.7%) patients were proximal, and 7 (23.3%) patients were distal, and was LCx in 17 (17%) patients of them 6 (35.3%) patients were proximal and 11 (64.7%) were distal. The mean value of EF was 53.30±10.321%. Table 3

Table 3: Angiographic results among the studied group

Angiography	Patients group (n=100)
LAD	53 (53%)
Proximal	23 (43.4%)
Distal	30 (56.6%)
RCA	30 (30%)
Proximal	23 (76.7%)
Distal	7 (23.3%)
LCx	17 (17%)
Proximal	6 (35.3%)
Distal	11 (64.7%)
EF%	53.30±10.321

Data are presented as frequency (%). LAD: Left anterior descending, RCA: Right coronary artery, LCx: Circumflex, EF: Ejection fraction

There was no significant variation in demographic data, risk factors and laboratory investigations between patients with different types of STEMI. Table 4

Table 4: Association between types of STEMI and demographic data, risk factors

		Anterior (n=26)	Inferior (n=26)	Anterolateral (n=19)	Inferolateral (n=14)	Antero septal (n=15)	P value
Demographic data							
Age (Years)		58.65±5.4	59.03±4.9	58.68±4.7	58.78±5.01	61.7±3.8	0.36
Sex	Male	9 (34.6)	13 (50)	7 (36.8%)	5 (35.7%)	5 (33.3)	0.76
	Female	17 (65.4)	13 (50)	12 (63.2%)	9 (64.3%)	10 (66.7)	
BMI		27.48±3.1	26.92±2.8	26.45±3.20	26.29±3.90	26.54±3.4	0.76
Risk factors							
HTN		12 (46.2%)	11 (42.3%)	4 (21.1%)	2 (14.3%)	6 (40.0%)	0.17
DM		4 (15.4%)	7 (26.9%)	3 (15.8%)	6 (42.9%)	2 (13.3%)	0.24
Dyslipidemia		3 (11.5%)	7 (26.9%)	4 (21.1%)	1 (7.1%)	4 (26.7%)	0.43
Smoking		8 (30.8%)	7 (26.9%)	6 (31.6%)	5 (35.7%)	7 (46.7%)	0.76
Family history		6 (23.1%)	2 (7.7%)	1 (5.3%)	2 (14.3%)	1 (6.7%)	0.32
Laboratory investigations							
Hb		12.74±1.3	12.91±1.2	13.27±1.24	13.16±1.44	13.61±0.91	0.24
PLT		229.69±64.1	206.08±48.5	228.7±58.1	202.14±24.5	204.73±51.5	0.26
WBCs		6.29±1.3	6.05±1.5	5.81±1.16	6.85±1.29	5.95±0.68	0.18
S.cr		0.99±0.16	0.98±0.23	0.96±0.17	0.85±0.07	1.02±0.19	0.11
AST		19.96±4.5	19.19±4.6	19.63±3.75	19.14±4.8	19.6±4.04	0.97
ALT		23.5±8.6	21.42±5.8	22.47±7.47	22.28±10.3	23.5±8.04	0.88
TC		198.15±29.5	191.08±28.04	197.11±20.7	196±30.2	193.2±28.4	0.90
TGS		145.73±25.2	137.65±19.5	137.79±21.2	146±22.84	137.2±24.2	0.53
LDL		117.92±9.5	115.38±7.73	118.32±9.44	116.9±10.5	118.5±6.35	0.75
HDL		47.07±6.2	52.11±4.9	49.52±7.11	48.28±7.3	49.53±7.2	0.08
Troponin	Normal	8 (30.8)	10 (38.5)	9 (47.4%)	2 (14.3%)	5(33.3)	0.37
	Elevated	18 (69.2)	16 (61.5)	10 (52.6%)	12 (85.7%)	10(66.7)	

Data are displayed as mean±SD or frequency (%). BMI: Body mass index. HTN: Hypertension, DM: Diabetes mellitus. Hb: Hemoglobin, PLT: Platelets, WBCs: White blood cells, S.cr: Serum creatinine, AST: Aspartate transaminase, ALT: Alanine transaminase, TC: Total cholesterol, TGS: Triglycerides, LDL: Low density lipoprotein, HDL: High density lipoprotein.

LAD culprit lesion by angiogram was significantly greater in patients with anterior MI in contrast to inferior, anterolateral, and inferolateral MI, and was significantly greater in anteroseptal MI in contrast to inferior and inferolateral MI, and was significantly greater in inferolateral and anterolateral MI in contrast to inferior MI. RCA culprit lesion by angiogram was significantly greater in patients with inferior and inferolateral MI in contrast to patients with anterior, anterolateral and anteroseptal MI. RCA culprit lesion by angiogram was significantly greater in patients with anterolateral MI in contrast to patients with anterior MI. EF was significantly less in patients with inferolateral MI in contrast to patients with anterior, inferior, anterolateral, and anteroseptal MI. There was no significant variation in EF between patients with anterior, inferior, anterolateral, and anteroseptal MI. Table 5

Table 5: Association between types of STEMI and angiographic result

Angiography	Anterior (n=26)	Inferior (n=26)	Anterolateral (n=19)	Inferolateral (n=14)	Antero septal (n=15)	P value
LAD	26 (100) a	0 (0) b	11 (57.9) c, d	2 (14.3) b, d	14 (93.3) a, c	<0.001*
RCA	0 (0) a	21 (80.8) b	0 (0) a	9 (64.3) b	0 (0) a	
LCX	0 (0) a	5 (19.2) a, b	8 (42.1) b	3 (21.4) a, b	1 (6.7) a, b	
EF%	53.38±10.03a	56.46±7.01a	57.15±12.04 a	41.28±7.6 b	54.0±7.8 a	P≤.001*

Data are shown as mean ± SD. LAD: Left anterior descending, RCA: Right coronary artery, LCx: Circumflex. EF: Ejection fraction. abcd: Similar letters indicate significant difference between groups, *significant $p \leq 0.05$

ECG findings in different types of myocardial infarctions are significant predictors of culprit artery defined by angiogram. Findings of LAD were significant predictors of LAD culprit artery by angiogram (AUC: 0.898, p<0.001). Findings of RCA were significant predictors of RCA culprit artery by angiogram (AUC: 0.852, p<0.001). The ECG findings of LCx were significant predictors of LCx culprit artery by angiogram (AUC: 0.728, p<0.001). Figure 1.

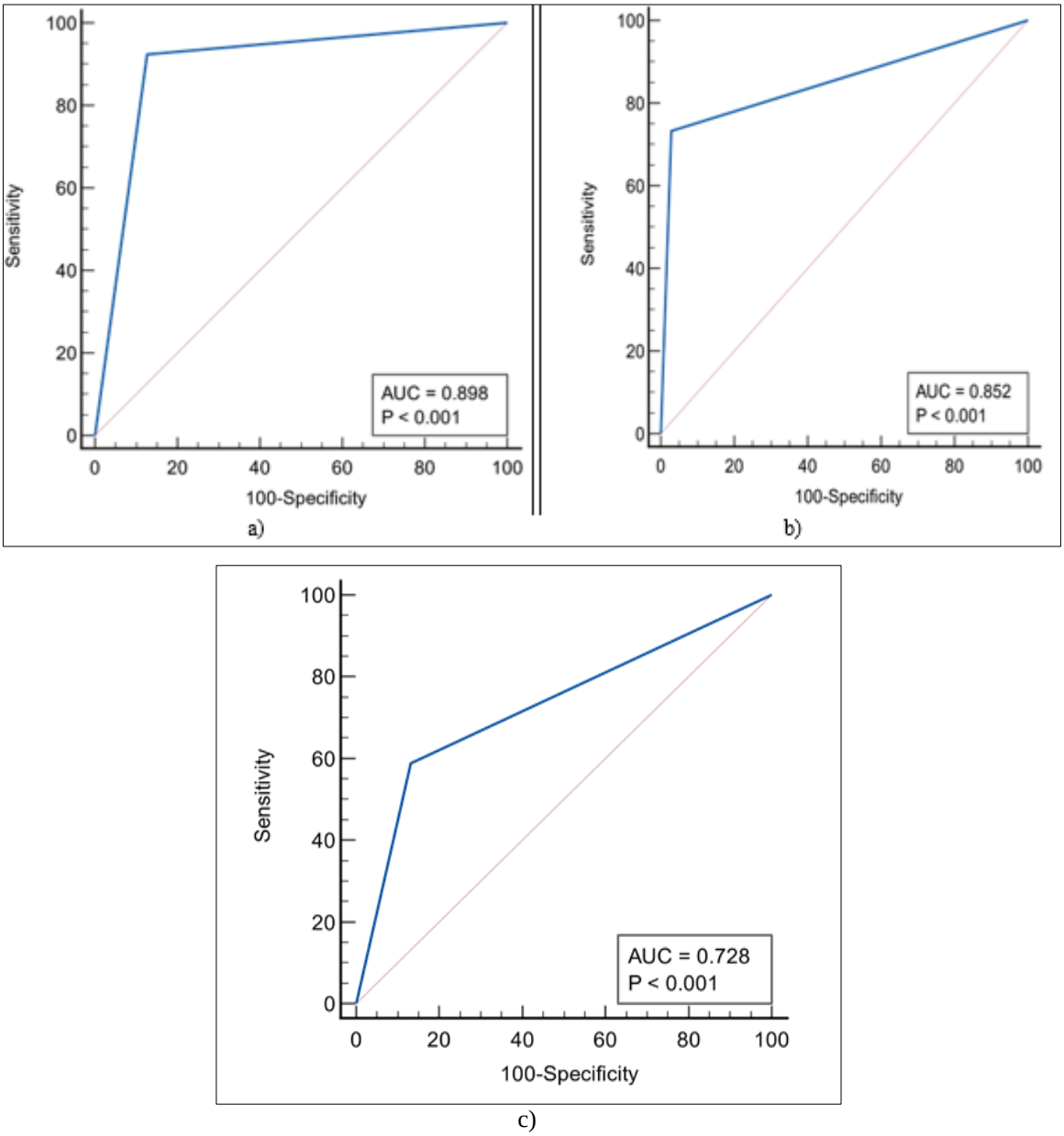

Fig 1: ROC curve of ECG findings of (A): LAD, (B): RCA and (C): LCx to predict LAD, RCA and LCx culprit artery defined by angiogram

Discussion

Use of the ECG is necessary for early examination of people believed to have acute coronary syndrome (ACS) [12]. The most recent AHA/ACC and ESC recommendations state that the ECG from the acute phase, particularly in ST-segment elevation ACS, provides critical information on the location and size of area at risk [13]. The most appropriate therapy for each patient is selected using this information. The ECG is the only reliable diagnostic tool available in an emergency because serum markers don't start to climb until necrosis has already occurred [14].

Our study was carried out on 100 patients, 61 were males and 39 were females with a mean age of 59.23 ± 4.92 years. Kamal *et al* [15] conducted a similar study the mean age was 53.34 ± 10.54 years which is lower than our study participants, but the male gender is predominant as our study. The second group included patients with inferior STEMI, 40 (80%) male and 10 female (20%) patients. The mean age was 55.7 ± 12.5 years which is lower than our study with predominance of male gender as well.

Regarding the type of infarction by ECG, our study reported that it was anterior MI in 26 (26.0%) patients, inferior MI in 26 (26.0%) patients, anterolateral MI in 19 (19.0%) patients, inferolateral MI in 14 (14.0%) patients, and Antero septal MI in 15 (15.0%) patients. Contrasted with our study, Kamal *et al* and Joseph *et al* [15, 16].reported their findings on patients with anterior and inferior wall STEMI only.

In our study, the most common risk factor discovered in 35% of patients was hypertension, which was followed by smoking (33%), diabetes (22%), and dyslipidemia (19%). Contrary to our research, Joseph *et al*. [16] found that the top four risk factors for diabetes mellitus, each accounting for 39.8% of cases, were smoking, hypertension, and dyslipidemia. According to the INTERHEART-South Asia

study, abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, low fruit and vegetable consumption, and inactivity are the eight coronary risk factors that account for 89% of all acute MI cases in Indians [17, 18]. In contrast to our findings and concordant with Joseph *et al*., Kiani *et al*. [16, 19] discovered that diabetes, hyperlipidemia, and hypertension were the most prevalent risk factors. Regarding the laboratory data there was no significant variation in laboratory investigations between patients with different types of STEMI. Contrary to our results kamal *et al*. [15] reported the creatine kinase, creatine kinase-myoglobin, and troponin values only of the RCA and LCX patients. Contrast to our finding's troponin levels were elevated in all participants while in our study troponin was elevated in 66% only. Like our findings there was no statistically significant variation between ECG findings and the laboratory investigations in kamal *et al*. findings [15].

Our study showed that ECG findings in different types of myocardial infarctions are significant predictors of culprit artery defined by angiogram. This is consistent with Kamal *et al*. [15] as this study reported that there was a statistically significant relation found in the analysis of the relation between the ECG criteria and the angiographic.

Regarding relation between culprit artery and the location of STEMI, our study showed the following: LAD culprit lesion by angiogram was significantly greater in patients with anterior MI in contrast to inferior, anterolateral, and inferolateral MI, and was significantly greater in anteroseptal MI in contrast to inferior and inferolateral MI, and was significantly greater in inferolateral and anterolateral MI in contrast to inferior MI. RCA culprit lesion by angiogram was significantly greater in patients with inferior and inferolateral MI in contrast to patients with anterior, anterolateral and anteroseptal MI. RCA culprit lesion by angiogram was significantly greater in patients with anterolateral MI in contrast to patients with anterior MI. RCA was more common than LCX in inferior STEMI. Like our study, Kamal *et al*. Chia *et al*. and Herz *et al*. [15, 20, 21] stated that the RCA is more common that LCX in patients with inferior STEMI.

Similar to our study, Gaude *et al* [22] reported that LAD is the most common culprit artery in anterior wall STEMI. This was consistent with Engelen *et al* [23], and Ghosh *et al* [24] Regarding inferior wall STEMI, Gaude *et al* [22] reported that the culprit artery can be RCA or LCX as observed by our findings as well. This is in accordance with Gupta *et al*, Kosuge *et al* and Verouden *et al* [25, 27].

Regarding the relation between ejection fraction and location of STEMI, our study showed that EF was significantly less in patients with inferolateral MI in contrast to patients with anterior, inferior, anterolateral, and anteroseptal MI. There was no significant variation in EF between patients with anterior, inferior, anterolateral, and anteroseptal MI. Different to our findings, Kiron *et al*. [28] showed that Patients who experienced an anterior MI had a statistically poorer LVEF than those who experienced an inferior or inferoposterior MI (P = 0.04).

Regarding the accuracy of different ECG findings to determine culprit artery utilizing angiogram our study reported ECG findings in different types of myocardial infarctions are significant predictors of culprit artery defined by angiogram. The ECG findings of LAD were significant predictors of LAD culprit artery by angiogram (AUC: 0.898, $p<0.001$). It had a sensitivity of 92.45%, specificity of 87.23%, PPV of 89.1% and NPV of 91.1%. The ECG findings of RCA were significant predictors of RCA culprit artery by angiogram (AUC: 0.852, $p<0.001$). It had a sensitivity of 73.3%, specificity of 97.1%, PPV of 91.7% and NPV of 89.5%.

The ECG findings of LCx were significant predictors of LCx culprit artery by angiogram (AUC: 0.728, $p<0.001$). It had a sensitivity of 58.8%, specificity of 86.7%, PPV of 47.6% and NPV of 91.1%. Kamal *et al* [15] reported the following: group A (anterior STEMI) the sensitivity of the ECG criteria in predicting the culprit artery was 76.92% and its specificity was 100%, with 100% PPV and 92.5% NPV. However, in group B (inferior STEMI), the sensitivity and specificity of the ECG criteria in predicting the culprit artery were 100 and 76.92%, respectively, with 92.5% PPV and 100% NPV.

Limitations: No correlation between different leads of ECG and angiography, no correlation with the type of culprit artery whether proximal or distal, single center study as studies in different centers can give different results and small sample size.

Conclusion

LAD culprit lesion by angiogram was significantly greater in patients with anterior MI. RCA culprit lesion by angiogram was significantly greater in patients with inferior and inferolateral MI and LCX culprit lesion by angiogram was significantly greater in patients with anterolateral. The ECG findings of RCA, LAD, and LCx were significant predictors of RCA, LAD, and LCx culprit artery by angiogram.

Financial support and sponsorship: Nil

Conflict of Interest: Nil

References

1. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, *et al*. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2018;39:119-77.
2. Vales L, Kanei Y, Schweitzer P. Electrocardiographic predictors of culprit artery in acute inferior ST elevation myocardial infarction. J Electrocardiol. 2011;44:31-5.
3. Kiani R, Sanati HR, Abdi S, Shakerian F, Firoozi A, Zahedmehr A, *et al*. Comparison of the clinical and morphologic characteristics of culprit lesions in unstable angina and non-ST-elevation myocardial infarction. Heart Asia. 2012;4:32-6.
4. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, *et al*. Fourth universal definition of myocardial infarction. J Am Coll Cardiol. 2018;72:2231-64.
5. Vogel B, Claessen BE, Arnold SV, Chan D, Cohen DJ, Giannitsis E, *et al*. ST-segment elevation myocardial infarction. Nat Rev Dis Primers. 2019;5:39-44.
6. Tanguay A, Lebon J, Lau L, Hébert D, Bégin F. Detection of STEMI using prehospital serial 12-lead

electrocardiograms. Prehosp Emerg Care. 2018;22:419-26.
7. Mortensen MB, Dzaye O, Steffensen FH, Bøtker HE, Jensen JM, Rønnow SNP, *et al*. Impact of plaque burden versus stenosis on ischemic events in patients with coronary atherosclerosis. J Am Coll Cardiol. 2020;76:2803-13.
8. Ducas RA, Labos C, Allen D, Golian M, Jeyaraman M, Lys J, *et al*. Association of pre-hospital ECG administration with clinical outcomes in ST-segment myocardial infarction: a systematic review and meta-analysis. Can J Cardiol. 2016;32:1531-41.
9. Khan GM. A new electrode placement method for obtaining 12-lead ECGs. Open Heart. 2015;2:226-50.
10. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, *et al*. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28:14-9.
11. Ommen SR, Nishimura RA, Appleton CP, Miller FA, Oh JK, Redfield MM, *et al*. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. Circulation. 2000;102:1788-94.
12. Kamphuis VP, Wagner GS, Pahlm O, Man S, Olson CW, Bacharova L, *et al*. Comparison of model-based and expert-rule based electrocardiographic identification of the culprit artery in patients with acute coronary syndrome. J Electrocardiol. 2015;48:483-9.
13. Tierala I, Nikus KC, Sclarovsky S, Syvänne M, Eskola M. Predicting the culprit artery in acute ST-elevation myocardial infarction and introducing a new algorithm to predict infarct-related artery in inferior ST-elevation myocardial infarction: correlation with coronary anatomy in the HAAMU Trial. J Electrocardiol. 2009;42:120-7.
14. van der Laarse A, Cobbaert CM, Gorgels AP, Swenne CA. Will future troponin measurement overrule the ECG as the primary diagnostic tool in patients with acute coronary syndrome? J Electrocardiol. 2013;46:312-7.
15. Kamal A, Soltan G, Ali M. Electrocardiographic prediction of culprit artery in acute ST-segment elevation myocardial infarction. Menoufia Med J. 2018;31:1463-9.
16. Joseph J, Menon J. The sensitivity and specificity of electrocardiogram in localizing the culprit artery with angiographic correlation in Indian patients with acute ST-segment elevation myocardial infarction. J Indian Coll Cardiol. 2021;11:70-81.
17. Guha S, Sethi R, Ray S, Bahl VK, Shanmugasundaram S, Kerkar P, *et al*. Cardiological Society of India: Position statement for the management of ST elevation myocardial infarction in India. Indian Heart J. 2017;69:63-97.
18. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, *et al*. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364:937-52.
19. Kiani F, Hesabi N, Arbabisarjou A. Assessment of risk factors in patients with myocardial infarction. Glob J Health Sci. 2015;8:255-62.
20. Chia BL, Yip JWL, Tan HC, Lim YT. Usefulness of ST elevation II/III ratio and ST deviation in lead I for identifying the culprit artery in inferior wall acute myocardial infarction. Am J Card. 2000;86:341-3.
21. Herz I, Assali AR, Adler Y, Solodky A, Sclarovsky S. New electrocardiographic criteria for predicting either the right or left circumflex artery as the culprit coronary artery in inferior wall acute myocardial infarction. Am J Card. 1997;80:1343-5.
22. Gaude R, Gautam D, Jain D, Singh G, Das P, Choudhury A, *et al*. Study of coronary angiographic correlation with electrocardiography in patients of acute coronary syndrome-ST-elevation myocardial infarction. Heart India. 2018;6:115-22.
23. Engelen DJ, Gorgels AP, Cheriex EC, De Muinck ED, Ophuis AJ, Dassen WR, *et al*. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. J Am Coll Cardiol. 1999;34:389-95.
24. Ghosh B, Indurkar M, Jain MK. ECG: A simple noninvasive tool to localize culprit vessel occlusion site in acute stemi. Heart India. 2013;5:20-9.
25. Gupta A, Lokhandwala YY, Kerkar PG, Vora AM. Electrocardiographic differentiation between right coronary and left circumflex coronary arterial occlusion in isolated inferior wall myocardial infarction. Indian Heart J. 1999;51:281-4.
26. Kosuge M, Kimura K, Ishikawa T, Hongo Y, Mochida Y, Sugiyama M, *et al*. New electrocardiographic criteria for predicting the site of coronary artery occlusion in inferior wall acute myocardial infarction. Am J Cardiol. 1998;82:1318-22.
27. Verouden NJ, Barwari K, Koch KT, Henriques JP, Baan J, van der Schaaf RJ, *et al*. Distinguishing the right coronary artery from the left circumflex coronary artery as the infarct-related artery in patients undergoing primary percutaneous coronary intervention for acute inferior myocardial infarction. Europace. 2009;11:1517-21.
28. Kiron V, George PV. Correlation of cumulative ST elevation with left ventricular ejection fraction and 30-day outcome in patients with ST elevation myocardial infarction. J Postgrad Med. 2019;65:146-51.

How to Cite This Article
Sayed AME, Shaban AE, Elgendy EAE, Saeid AME. Correlation between St Segment elevation in the 12-lead ECG and the culprit coronary artery in patients with acute stemi undergoing primary PCI. International Journal of Cardiology Sciences 2024; 6(1): 01-07.