

Evaluation of Different ECG Parameters to Predict the Culprit Artery and Site of occlusion in patient with Acute Inferior Wall Myocardial Infarction

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Abstract: Background: Acute myocardial infarction is still a major public health problem despite the impressive stride in diagnosis and management. Furthermore, the early and accurate diagnosis with the proper management of this major event is an important challenge. The determination of infarct related artery in acute inferior myocardial infarction is extremely important for the prediction of potential complication and predicting the probable site of occlusion is worthwhile because proximal occlusions are likely to cause greater myocardial damage. **Aim of Work:** The aim of the present study is to evaluate the use of different ECG criteria to evaluate the culprit artery and site of occlusion in patients with acute inferior wall myocardial infarction. **Methodology:** The present history was conducted on 100 patients presenting to Al Azhar University Hospitals by acute inferior myocardial infarction. All patients were subjected to: thorough history taking, full clinical examination, surface ECG, serial cardiac enzymes, echocardiography and coronary angiography. Studied ECG criteria included ST segment depression in lead aVR > 1mv, ST segment elevation in lead III more than lead II, ST segment depression in lead I > 0.05 mv, ST segment elevation in lead V4R > 1mv, rhythm and heart rate. **Results:** This present study confirmed the utility of ST segment depression in lead aVR for predicting LCX occlusion and association with larger infarct size, A higher ST segment elevation in lead III than in lead II and ST segment depression in lead I > 0.05mm are markers for right coronary artery related acute inferior wall myocardial infarction, The amplitude of ST segment elevation and the proximity of culprit lesion along the infarct related right coronary artery was found to be significantly related, ST segment elevation in lead V4R \geq 1mm as a criterion for RCA involvement has excellent specificity and PPV however low sensitivity and NPV were found and ST segment elevation in lead V4R \geq 1mm was also assessed as a criterion for proximal RCA involvement vs. mid to distal RCA involvement in cases of inferior wall MI. **Conclusion:** It is possible to predict the culprit coronary artery in inferior wall acute myocardial infarction by using the readily obtainable measures on the admission electrocardiography. **Recommendations:** We recommend analysis of electrocardiography in patients with myocardial infarction to predict the culprit coronary artery and its proximity, for more proximal occlusions are likely to cause greater myocardial damage and early intervention is recommended. [Mamdouh Helmy Eltahan, Ali Ali Ramzy, Ashraf Mohamed Anwar, Ashraf Al-Amir Abd Elfattah and Omar Samir Sedik. **Evaluation of Different ECG Parameters to Predict the Culprit Artery and Site of occlusion in patient with Acute Inferior Wall Myocardial Infarction.** *N Y Sci J* 2016;9(12):132-139]. ISSN 1554-0200 (print); ISSN 2375-723X (online). <http://www.sciencepub.net/newyork>. 23. doi:[10.7537/marsnys091216.23](https://doi.org/10.7537/marsnys091216.23).

Keywords: Evaluation; Different ECG Parameter; Predict; Culprit Artery; occlusion; patient; Acute Inferior Wall Myocardial Infarction

1. Introduction:

Acute myocardial infarction (AMI) is a common disease with serious consequences in mortality, morbidity and cost to the society. Coronary atherosclerosis plays a pivotal part as the underlying substrate in many patients (Boersma et al., 2003). Acute myocardial infarction has always been a potential health problem due to the life-threatening complications (Correale et al., 1999). Myocardial infarction results from prolonged myocardial ischemia precipitated in most cases by an occlusive coronary thrombus at the site of a pre-existing atherosclerotic

plaque. More rarely, infarction may result from prolonged vasospasm, inadequate myocardial blood flow or excessive myocardial demand. Very rarely myocardial infarction may be caused by embolic coronary occlusion, vasculitis, coronary artery dissection or aortitis. Certain risk factors predispose to AMI which are categorized as modifiable (smoking, hypertension, dyslipidemia, obesity, physical inactivity and diabetes) and non-modifiable (age, sex and family history of heart disease) (Iqbal et al., 2008). The electrocardiogram (ECG) is being used for decades reliable and inexpensive tool to diagnose

acute myocardial infarction in the patient with chest pain. More recently, an emphasis has been made on ECG features that allow better identification of the coronary occlusion site and thereby better estimation of the size of the area at risk, which is important for the preferred type of reperfusion. On average the left anterior descending coronary artery supplies 50% of blood flow and the right coronary artery (RCA) and circumflex (Cx) coronary artery each supply.

2. Patients and Methods:

This study is a prospective study which was conducted on 100 patients with first time inferior STEMI who were admitted to Al Azhar University Hospitals from January 2016 till October 2016.

Inclusion criteria:

The present study included 100 patients presenting by first attack inferior wall STEMI. Inferior wall STEMI was defined as typical rise and/or fall of biochemical markers of myocardial necrosis with Ischemic symptoms and ST-segment elevation of more than or equal to 1 mm in two or more inferior leads (II, III and aVF) (Thygesen et al., 2012). The presence of right ventricular infarction in association with inferior MI will be defined by ST-segment elevation more than or equal to 1 mm in leads v4R through v6R (Erhardt et al., 2000). All patients underwent coronary angiography either as a part of primary percutaneous coronary intervention or elective coronary angiography during their hospital stay after thrombolytic therapy.

Exclusion criteria:

History of previous myocardial infarction, History of previous PCI or coronary artery bypass graft (CABG) surgery and Absence of conditions precluding the evaluation of ST-segment. Changes on the ECG: (bundle branch block, ventricular pacing, ventricular hypertrophy and pericarditis).

Methods:

All the patients underwent detailed clinical evaluation that included:

1-Complete history taking

Full history was taken from the patients regarding the age, sex and risk factors of CAD including: smoking, hypertension, diabetes mellitus, dyslipidemia and family history of atherosclerosis.

ECG examination:

Standard 12-lead electrocardiogram was performed for all patients on admission. The ECG was recorded at a paper speed of 25mm/second at a calibration of 1 mv equals 10 mm. ST segment deviation was measured 80 milliseconds from the J point with the naked eye, using the TP segment as isoelectric line unless tachycardia caused fusion of the T and P waves, in which case the PR segment was used (Pelter et al., 1996). The following ECG

criteria were studied for detection of the culprit artery in cases of inferior MI: -ST-segment depression in lead avR ≥ 0.1 mv, ST-segment elevation in lead III more than lead II, ST-segment depression in lead I ≥ 0.05 mv, ST-segment elevation in lead v4R ≥ 1 mm and Rhythm and heart rate.

The following ECG criteria were studied for detection of the site of occlusion within RCA in cases of inferior MI caused by RCA occlusion:

Sum of ST elevations in inferior leads (II, III and avF) was compared between proximal, mid and distal right coronary artery patients and ST-segment elevation in lead v4R ≥ 1 mm was compared between proximal and mid to distal right coronary artery.

Coronary Angiography:

All patients undergone coronary angiography either as a part of primary percutaneous coronary intervention or elective coronary angiography during their hospital stay after thrombolytic therapy. The culprit artery was determined from angiographic characteristics of occlusion (occlusion due to thrombus formation or ulceration with decreased contrast density). Coronary artery stenosis of more than 70% was defined as obstructive and multi vessel coronary artery disease was defined as having two or more coronary arteries with obstructive lesions (Mill et al., 2003).

Laboratory analysis:

The cardiac enzymes total CPK and CK-MB were estimated regarding their peak level on hospital admission and at 6 hour intervals during the first 24 hours then daily until discharge.

Echocardiography:

The following items were assessed: -Left ventricular ejection fraction by using **modified Simpson method** This technique requires recording an apical, four and two chamber views from which the endocardial border is outlined in end diastole and end-systole (Lang et al., 2006)., Quantification of wall motion abnormalities involves generation of **wall motion score index**. This methodology involves describing the wall motion characteristics of each of the predefined segments as being normal, hypokinetic, akinetic, dyskinetic, or aneurysmal. A numerical score, typically 1 to 5, is then applied to each of these segments respectively, and the total score is divided by the number of segments. A ventricle with completely normal wall motion would have a score index of 1.0 (total score divided by the number of segments) (Cerqueira et al., 2002) and Right ventricular examination: Assessment of right ventricular functions by TAPSE (Tricuspid annulus plane systolic excursion) is a measure of RV longitudinal function. TAPSE < 16 mm indicates RV systolic dysfunction. It is measured from the tricuspid lateral annulus. (Kaul et al., 2004).

3. Results:

This study was conducted on 100 patients with first time inferior STEMI Using ST segment depression ≥ 1 mm in aVR as a criterion for LCX as the culprit artery in cases of inferior wall MI showed sensitivity and specificity of 60% and 81% with PPV and NPV 44 % and 89% respectively. The sensitivity and specificity of ST segment elevation in lead III greater than lead II as a criterion of RCA occlusion were 78% and 60% respectively. PPV and NPV of this criterion were 88% and 41% respectively. The use of ST segment depression in lead I > 0.05 mm as a

criterion of RCA occlusion had been also studied. The sensitivity, specificity, PPV and NPV of this criterion were 71%, 65%, 89% and 36% respectively. In the present study, the mean heart rate was significantly lower in RCA cases (57.11 ± 8.36 b/m) than LCX cases (75 ± 14.23 b/m) (P value < 0.05).

I- Demographic data:

Mean age of patients involved in the study was 51.3 ± 10.2 years, maximum age of 77 years and minimum of 29 years. 79% of the studied cases were males.

Table (1): Distribution of the studied cases as regards demographic data:

	Mean \pm SD	Range
Age (yrs)	51.3\pm10.2	29-77
Gender	No	%
Male	79	79%
Female	21	21%

II-Clinical risk factors:**Table (2): Distribution of clinical risk factors among the studied cases.**

Risk factors	No	%
DM		
Non diabetic	48	48%
IDDM	23	23%
NIDDM	29	29%
HTN	58	58%
FH	40	40%
Dyslipidema	48	48%
Smoking		
Non smoker	34	34%
Smoker	56	56%
Ex-smoker	10	10%

III- Angiographic data:

Table (3): Angiographic finding among the studied cases.

Angiographic finding	No	%
Culprit artery:		
RCA	79	79%
LCX	20	20%
LAD	-	-
Not Identified	1	1%
Dominance		
Right dominance	67	67%
left /co dominance	33	33%
MVD	25	25%
TIMI 0	82	82%

V- Echo Data**Table (4): Echo findings for the studied cases in relation to ST segment depression in lead avR**

	ST-segment segment depression in avR (no= 28)	Non ST-segment depression in avR (no=72)	t. test	P. value
WMSI	1.20±0.24	1.24±0.22	0.865	>0.05
EF	56.16±5.22	55.14±5.88	0.902	>0.05
TAPSE	2.13±0.32	2.04±0.28	1.413	>0.05

VI- ECG Data:**1-Heart Rate:****Table (5): Heart Rate of the studied cases in relation to the culprit artery.**

ECG DATA	LCX(n=20)	RCA(n=79)	t. test	P. value
Heart Rate	75±14.23	57.11±8.36	4.663	<0.05

2- Rhythm:**Table (7): Rhythm abnormalities of the studied cases in relation to the culprit artery.**

Rhythm	All patients	LCX (n=20)	RCA (n=79)	X ²	P. value
Sinus Rhythm	87(87.8%)	18(90%)	69(87.3%)	0.639	>0.05
Sinus brady-cardia	24(24.2%)	3(15%)	21(26.6%)	4.220	<0.05
AF	4(4.04%)	2(10%)	2(6.3%)	1.639	>0.05
1st degree HB	-	-	-	-	-
2nd degree HB	2(2.02%)	-	2(2.5%)	0.852	>0.05
3rd degree HB	6 (6.06%)	-	6(5.1%)	0.552	>0.05

3- ST segment changes in various leads:

Table (6): ST segment changes in various leads in relation to the culprit artery

ST segment change	LCX(n=20)	RCA(n=79)	X2	P-value
Elevation in lead III>II	8 (40%)	62 (78%)	7.75	<0.001
Depression in lead I> 0.05mv	7 (35%)	56 (70%)	6.39	<0.001
Depression in lead avR ≥0.1mv	12 (60%)	15(19%)	4.336	<0.05
Elevation in lead V4R ≥1mm	0	29(36.7%)	6.332	<0.001

Table (8): Sensitivity, specificity and predictive values of various ECG criteria in detection of Culprit artery in inferior wall MI

ST segment change	Sensitivity	specificity	PPV	NPV
Elevation in lead III>II (RCA)	78%	60%	88%	41%
Depression in lead I> 0.05mv (RCA)	71%	65%	89%	36%
Depression in lead avR >0.1mv (LCX)	60%	81%	44%	89%
Elevation in lead V4R >1mm(RCA)	37 %	100%	100%	40%

B-Study of ECG data for detection of the site of occlusion within The RCA:

Table (9): Relation between total ST elevations in inferior leads and site of lesion within RCA.

Site of occlusion	Sum of ST elevations in inferior leads		
	Mean	Std. Deviation	N
Proximal RCA	8.51	4.44	30
Mid RCA	5.95	3.06	23
Distal RCA	5.00	2.77	26
f.test	4.336		
p. value	<0.001		

2- ST segment elevation in lead V4R ≥ 1mm:

Table (10): ST elevation in lead V4R≥1mm in proximal and mid to distal RCA.

	Proximal RCA(n=30)	Mid to distal RCA(n=49)	X2	p. value
ST elevation in lead V4R≥1mm	24(80%)	5(10.2%)	9.336	<0.001

4. Discussion:

In acute myocardial infarction, the first direct diagnosis is made using the surface electrocardiogram (ECG). The information yielded by the ECG provides the physician with an initial analysis of the patient's condition. It gives the physician directly the location of the infarct, its extent and basically the artery that has been occluded (Engelen et al., 1999). Various ECG criteria have been suggested to predict the culprit artery in acute inferior wall MI based on analysis of ST segment elevation and depression in different leads (Verouden et al., 2009). In the present study, the importance of ST segment depression in lead aVR was pointed out as a sign of LCx occlusion and infarct size in cases of acute inferior wall MI. Evaluation of other different ECG criteria to detect culprit artery and site of occlusion in cases of inferior wall MI have been also evaluated. In the present study, the RCA was much more likely than the LCX to be the culprit artery in patients with acute inferior wall MI. Among 100 patients with acute inferior wall MI, the culprit artery was in the RCA in 79% of patients and in the LCX in 20% of patients, with ratio of 3.95:1. Rarely, acute inferior MI may result from occlusion of the recurrent LAD branch which is the terminal portion of a "wraparound" LAD, but this was not the case in any of the present study patients (Correale et al., 1999 and Birnbaum 1999). Chia et al., (2000) concluded that the RCA supplies blood mainly to the posterior part of the septum and the inferior part of the infero posterior wall of the myocardium, whereas the LCx supplies blood mainly to the posterior part of the infero posterior wall and the posterior part of the lateral wall. As a result, the vector of injury current is directed more to the right and inferior in RCA occlusions and more to the left and posterior in LCX occlusions. This minor difference in vector direction forms the basis of electrocardiographic differentiation between LCX and RCA occlusions in cases of acute inferior wall MI. Regarding detection of the culprit artery in cases of acute inferior wall STEMI, Various ECG criteria have been studied. In the present study, ST depression in lead aVR $\geq 1\text{mm}$ was found in 60 % of patients who had LCX as a culprit artery and in 19 % of patients who had RCA as a culprit artery. When ST segment depression $\geq 1\text{mm}$ in aVR was taken as a criterion for LCX involvement in cases of acute inferior wall STEMI, sensitivity and specificity were 60% and 81% respectively. Positive and negative predictive values were 44 % and 89% respectively. The current of injury with RCA occlusion is more or less perpendicular to the axis of lead aVR, whereas the current of injury resulting from occlusion of the LCX has a mean vector that forms an obtuse angle with the axis of aVR.

Therefore, significant ST segment depression in aVR is more likely to occur with LCX occlusion.

Regarding ST segment elevation in lead III greater than lead II as a criterion of RCA occlusion in cases of acute inferior wall MI. The sensitivity, specificity, PPV and NPV of this criterion for prediction RCA occlusion were (78%), (60%), (88%) and (41%) respectively. Standard lead III is oriented to the right inferior segment where as lead II is oriented principally to the left inferior segment and also tend to be oriented to inferior region of left lateral and superior wall of left ventricle. Consequently lead III is influenced by RCA related acute inferior wall MI where as lead II is more influenced by LCX related acute inferior wall MI (Foil et al., 2004).

Regarding ST segment depression in lead I $> 0.05\text{mm}$ as a criterion of RCA occlusion in cases of acute inferior wall MI, The sensitivity, specificity, PPV and NPV of this criterion for prediction RCA occlusion were (71%), (65%), (89%) and (36%) respectively. Lead I is more oriented to lateral segments of the left ventricle. In cases of acute inferior wall MI due to RCA occlusion, ST depression in lead I represents reciprocal change for the area supplied by the RCA. LCX occlusion is more affecting high posterolateral and apical segments of the left ventricle. In cases of acute inferior wall MI due to LCX occlusion, ST depression in lead I is less prominent and even ST elevation can appear (Jim et al., 2012).

ST segment elevation in lead V4R $\geq 1\text{mm}$ as a criterion for RCA involvement in cases of inferior wall MI showed excellent specificity 100% and PPV 100 % but low sensitivity and NPV of 37 % and 40 % respectively were found.

In the present study, the mean heart rate was statistically significantly lower in RCA cases (57.11 ± 8.36 b/m) than LCX cases (75 ± 14.23 b/m) (P value < 0.05). This can be attributed to higher incidence of sinus bradycardia among RCA cases as compared to LCX cases. 21 of the RCA cases (26.6%) had sinus bradycardia, while in LCX cases only 3 cases (15%) had sinus bradycardia (P value < 0.05). There was a trend towards a higher incidence of CHB and 2:1 block among RCA cases as compared to LCX cases, where CHB and 2:1 block were observed in 5.1% and 2.5% of RCA cases respectively, while neither CHB nor 2:1 block were observed in LCX cases. However this difference did not reach a statistical significance (P value > 0.05). This may be attributed to a relatively small number of LCX cases. Transient sinus bradycardia and AV block are common following inferior infarction, usually related to occlusion of the sinoatrial artery and AV nodal artery as well as reflex vagal activation. Stimulation of cardiac sensory receptors with vagal afferents enhances

parasympathetic activity producing bradycardia, vasodilatation and hypotension. This phenomenon is known as the Bezold–Jarish reflex (**Kawasaki et al., 2009**) in 60% of individuals, the sinoatrial artery originates from proximal RCA and sinus arrest is likely to occur in proximal RCA occlusions. Also, the atrioventricular (AV) node artery originates from proximal RCA in 86% of individuals (**Trappe, 2010**).

Regarding detection of the site of occlusion within the RCA in cases of acute inferior wall STEMI caused by RCA occlusion, different ECG criteria have been studied. Sum of ST elevations in inferior leads (II, III and avF) was compared between proximal, mid and distal right coronary artery patients. ST-segment elevation in lead v4R ≥ 1 mm was also compared between proximal and mid to distal right coronary artery patients. Greater total ST segment elevation was observed in patients with proximal RCA occlusion as compared to those with mid and distal RCA occlusions, Patients with proximal RCA occlusion showed a mean ST segment elevation of (8.51 \pm 4.44mm), with mid RCA occlusion (5.95 \pm 3.06 mm) and (5.00 \pm 2.77) with distal RCA occlusion. The total ST elevation showed decreasing trends with lesion progression from proximal to distal location within the RCA (P value<0.001).

In the present study, ST segment elevation in lead V4R ≥ 1 mm was found in 80% patients who had proximal RCA involvement and in 10.2% of patients who had mid to distal RCA involvement in cases of inferior wall MI. This difference was statistically highly significant (P value <0.001). ST segment elevation in lead V4R ≥ 1 mm as a criterion for proximal RCA involvement vs. mid to distal RCA involvement in cases of inferior wall MI showed sensitivity, specificity, PPV and NPV for prediction RCA occlusion of (80%), (90%), (83%) and (88%) respectively.

Conclusion:

It is possible to predict the culprit coronary artery in inferior wall acute myocardial infarction by using the readily obtainable measures on the admission electrocardiography. The present study confirmed the utility of ST segment depression in lead aVR for predicting LCX occlusion and association with larger infarct size. A higher ST segment elevation in lead III than in lead II and ST segment depression in lead I > 0.05mm are markers for right coronary artery related acute inferior wall myocardial infarction. The amplitude of ST segment elevation and the proximity of culprit lesion along the infarct related right coronary artery was found to be significantly related. ST segment elevation in lead V4R ≥ 1 mm as a criterion for RCA involvement has excellent specificity and PPV however low sensitivity and NPV

were found. ST segment elevation in lead V4R ≥ 1 mm was also assessed as a criterion for proximal RCA involvement vs. mid to distal RCA involvement in cases of inferior wall MI.

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