

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

Omar Samir Sedik, Ashraf Mohammed Anwar*, Ashraf Al-Amir Abd Elfattah, Ali Ali Ramzy, Mamdouh Helmy Eltahan

Department of Cardiology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt

Email address:

ashrafanwar2000@hotmail.com (A. M. Anwar)

*Corresponding author

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Abstract: The determination of infarct related artery in acute inferior myocardial infarction (MI) is extremely important. The present study aimed to evaluate the use of different ECG criteria to predict the culprit artery and site of occlusion in patients with acute inferior wall MI. The study conducted 100 patients (51.3±10.2 yrs, 79% males) presented by acute inferior MI. All patients were subjected to surface 12-lead ECG. Four ECG criteria were analyzed for prediction of culprit artery; ST segment depression in lead aVR >1 mV, ST segment elevation in lead III more than lead II, ST segment depression in lead I >0.05 mV and ST segment elevation in lead V₄R > 1mV. The sum of ST segment elevation in lead II, III and aVF and ST segment elevation in lead V₄R > 1mV were analyzed to predict the site of occlusion. Patients were divided into 2 groups based on the angiographic definition of the culprit artery: Group I included 79 patients (79%) with RCA lesion and Group II included 20 patients (20%) with LCX lesion. Only 1 patient (1%) was excluded because he had normal coronary angiography. In Group I, the ST segment elevation in lead III greater than lead II and ST segment depression in lead I > 0.05 mm had a comparable sensitivity (78% and 71% respectively) and specificity (60% and 65% respectively) for RCA as the culprit artery. The ST segment elevation ≥ 1mm in V₄R had very low sensitivity (37%) and highest specificity (100%). In Group II, ST segment depression ≥ 1mm in aVR was the best criteria for LCX as the culprit artery with sensitivity of 60% and specificity 81%. The sum of ST segment elevation in lead II, III and aVF was higher in proximal RCA (8.51±4.44mm) than both mid RCA (5.95 ± 3.06 mm) and distal RCA (5.00 ± 2.77 mm) (P value <0.001). The study concluded that it is possible to predict the culprit coronary artery in acute inferior wall MI by using the readily obtainable measures on the admission ECG.

Keywords: ECG, ACUTE Inferior Myocardial Infarction, Culprit Artery

1. Introduction

Acute myocardial infarction (MI) is a common disease with serious consequences in mortality, morbidity and cost to the society [1]. Inferior wall MI is generally considered as being low risk, compared with anterior wall MI. The reported in hospital mortality rate ranges from 11% in the pre-thrombolytic era to 3.5%–9% in the thrombolytic era which is about half that of anterior wall MI [2]. In ST elevation MI, identifying the culprit artery on presenting electrocardiogram (ECG) can lead to earlier risk stratification and better guide

therapy for reperfusion [3]. ECG is being used as reliable and inexpensive tool to diagnose acute MI in the patient with chest pain. 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. The closer the occlusion site to the origin of the coronary artery the larger the ischemic area and the greater the necessity of a rapid reperfusion attempt [4]. The culprit artery of ST elevation MI is nearly always the left anterior descending artery (LAD), but inferior STEMI can be caused by an occlusion of either the right coronary artery (RCA) or left

circumflex (LCX) artery. Various ECG criteria have been suggested to predict the culprit artery based on analysis of ST segment elevation and ST-segment depression in different leads. ST-segment depression in lead aVR has been suggested as a predictor of LCX artery involvement [5]. ECG findings used to predict culprit artery in inferior MI are less useful in patients with dominant LCX infarction. Few studies have correlated aVR depression with the relative dominance of the RCA and LCX [6]. The study aim was to use different ECG criteria to detect the accuracy of each in the detection of culprit artery and site of occlusion in patients with acute inferior wall MI.

2. Patients and Methods

This study is a prospective study which was conducted 100 patients (mean age: 51.3 ± 10.2 years, 79% were males) with the first attack of inferior ST elevation MI who were admitted to Al Azhar University Hospitals from January 2016 till October 2016. Inferior wall ST elevation MI 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) [7]. The presence of associated right ventricular infarction with inferior MI was defined by ST-segment elevation more than or equal to 1 mm in leads V₄R through V₆R [8]. Patient with history of previous MI, percutaneous intervention or coronary artery bypass graft were excluded. Patients with ECG changes led to inaccurate interpretation of ST segments e.g. bundle branch block, ventricular pacing, ventricular hypertrophy and pericarditis were excluded also. All patients underwent the following:

Complete history regarding the age, sex and risk factors of coronary artery disease.

Laboratory analysis: The peak level of creatinine kinase (CK) and CK-MB were estimated on hospital admission and at 6 hour intervals during the first 24 hours then daily until discharge.

Standard 12-lead ECG: ECG was performed on admission at a paper speed of 25mm/second and a calibration of 1 mv equals 10 mm. ST segment deviation was measured 80 milliseconds from the J point with the, 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 [9]. In addition to heart rate and rhythm, 4 ECG criteria were analyzed: 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 and ST- segment elevation in lead V₄R ≥ 1 mm for the prediction of the culprit artery. Two ECG criteria were used for the prediction of the site of

occlusion; ST- segment elevation in lead V₄R ≥ 1 mm and sum of ST elevations in inferior leads (II, III and aVF).

2-Dimensional Echocardiography: The following measurements were assessed: Left ventricular ejection fraction (LV-EF) using modified biplane Simpson method [10], quantification of wall motion score index (WMSI) [11] and tricuspid annulus plane systolic excursion (TAPSE) as a measure of RV longitudinal function [12].

Coronary Angiography: All patients underwent coronary angiography either as a part of primary percutaneous coronary intervention or elective coronary angiography during hospital stay after thrombolytic therapy. The culprit artery was determined angiographically by thrombus occlusion or ulceration with decreased contrast density. Coronary artery stenosis was defined as $> 70\%$ obstruction [13].

Statistical analysis: Statistical analysis was conducted using SPSS V.16 and expressed as mean \pm SD. Chi-square test for comparison between two groups as regards qualitative data. Analysis of variance (ANOVA) tests was used for comparison among different times in the same group in quantitative data.

3. Results

Distribution of risk factors and angiographic data are displayed in Table 1.

Table 1. Distribution of risk factors and angiographic data.

Variable	No (%)
<i>Risk factors of CAD</i>	
Diabetes Mellitus	48 (48%)
Hypertension	58 (58%)
Smoking	56 (56%)
Hyperlipidemia	48 (48%)
Family history	40 (40%)
<i>Angiographic Data</i>	
RCA is culprit	79 (79%)
LCX is culprit	20 (20%)
Unidentified	1 (1%)
RCA dominance	67 (67%)
LCX / co dominance	33 (33%)
Multi-vessel disease	25 (25%)

Based on the angiographic definition of the culprit artery, patients were divided into 2 groups:

Group I included 79 patients (79%) with RCA lesion and

Group II included 20 patients (20%) with LCX lesion.

Only 1 patient (1%) was excluded because he had normal coronary angiography. ECG criteria, echocardiographic parameters, CK and CK-MB were analyzed in both groups (table 2)

Table 2. Comparison between both groups.

	Total N= 99	Group I N= 79	Group II N= 20	P value
ECG Criteria				
ST- segment depression in lead aVR ≥ 0.1 mm	27	15 (19%)	12 (60%)	0.03
ST -segment elevation in lead III $>$ lead II	70	62 (78%)	8 (40%)	<0.001
ST -segment depression in lead I ≥ 0.05 mm	7	0 (0%)	7 (35%)	<0.001

	Total N= 99	Group I N= 79	Group II N= 20	P value
ST- segment elevation in lead V ₄ R ≥ 1 mm	29	29 (36.7%)	0 (0%)	<0.001
Echocardiographic Data				
LV-EF (%)	55.49±3.2	55.12±5.6	55.78±4.3	NS
WMSI	1.22±0.2	1.22 ±0.5	1.23±0.1	NS
TAPSE (cm)	2.11±0.4	1.94 ±0.18	2.17 ± 0.25	NS
Peak CK (U/L)	2143.6±600.4	2184.9±601.8	2100.4±518.7	NS
Peak CK-MB (ng/ml)	183.5±50.2	183.3±51.1	183.9±49.1	NS

ECG Analysis: The underlying heart rhythm was sinus in 88 patients (88%), atrial fibrillation in 4 patients (4%), 2nd degree heart block in 2 patients (2%) and complete heart block in 6 patients (6%). Group I showed slower heart rate than group II (57.1 ± 8.4 bpm vs. 75 ± 14.2 bpm; $p = 0.02$). All patients (8 patients) with 2nd degree and complete heart block were in Group I.

Echocardiographic Parameters, CK and CK-MB:

All echocardiographic parameters (LVEF, WMSI and TAPSE), CK and CK-MB showed no difference between both groups.

ECG criteria and culprit artery:

As shown in table 2, both groups were compared regarding the presence or absence of the 4 ECG criteria and the

accuracy of each criterion for the detection of the culprit artery was analyzed.

In Group I, the ST segment elevation in lead III greater than lead II and ST segment depression in lead I > 0.05 mm had a comparable sensitivity (78% and 71% respectively) and specificity (60% and 65% respectively) for RCA as the culprit artery. The ST segment elevation ≥ 1 mm in V₄R had very low sensitivity (37%) and highest specificity (100%). ST segment depression ≥ 1 mm in aVR did not show relation.

In Group II (Figure 1), ST segment depression ≥ 1 mm in aVR was the best criteria for LCX as the culprit artery with sensitivity of 60% and specificity 81%, while other criteria did not show relation.

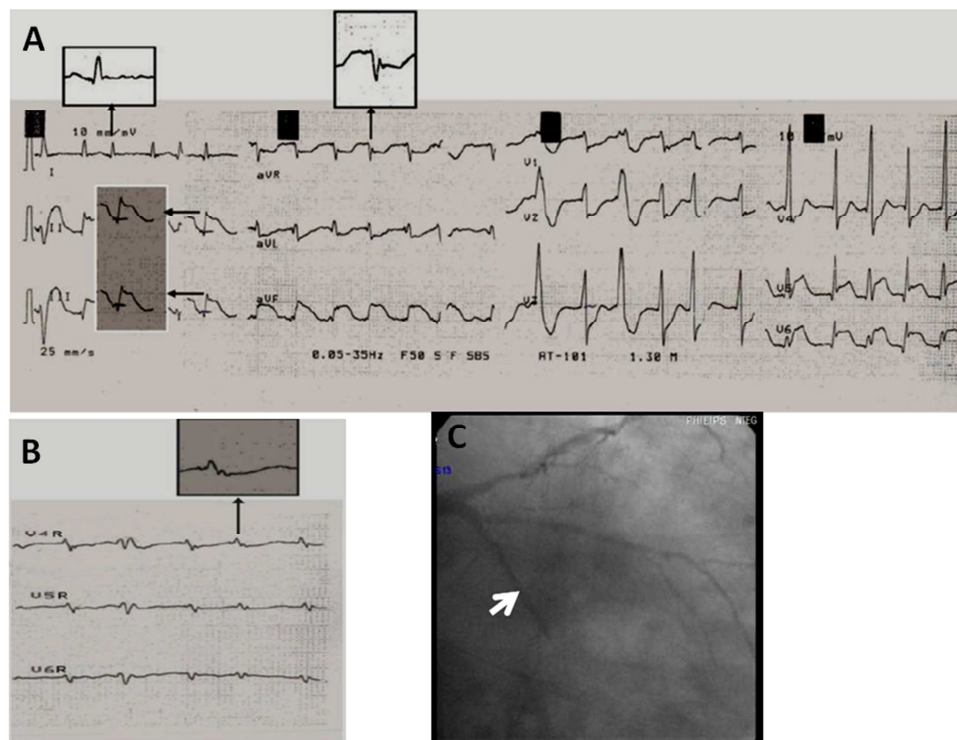


Figure 1. (A) 12 lead ECG tracings of patient in Group II showing ST segment elevation in lead II more than that in lead III, ST segment depression in lead aVR and absence of ST segment depression in lead I, (B) Right ECG leads showing absence of ST segment elevation and (C) coronary angiography showing totally occluded LCX after giving the first obtuse marginal branch.

ECG criteria and Site of occlusion:

In Group I (Figure 2), the site of RCA occlusion was proximal in 30 patients (38%), mid at 23 patients (29.1%) and distal in 26 patients (32.9%). The sum of ST segment elevation in lead II, III and aVF was higher in proximal RCA (8.51 ± 4.44 mm) than both mid RCA (5.95 ± 3.06 mm) and distal RCA (5.00 ± 2.77 mm) (P value <0.001). The sum of ST segment elevation in lead II, III and aVF was higher in

proximal RCA (8.51 ± 4.44 mm) than both mid RCA (5.95 ± 3.06 mm) and distal RCA (5.00 ± 2.77 mm). ST segment elevation in lead V₄R ≥ 1 mm was detected in 24 patients (80%) who had proximal RCA involvement and in 5 patients (10.2%) of who had mid to distal RCA involvement (P value <0.001). Therefore the sensitivity and specificity for prediction of proximal RCA occlusion of this criterion were (80%) and (90%).

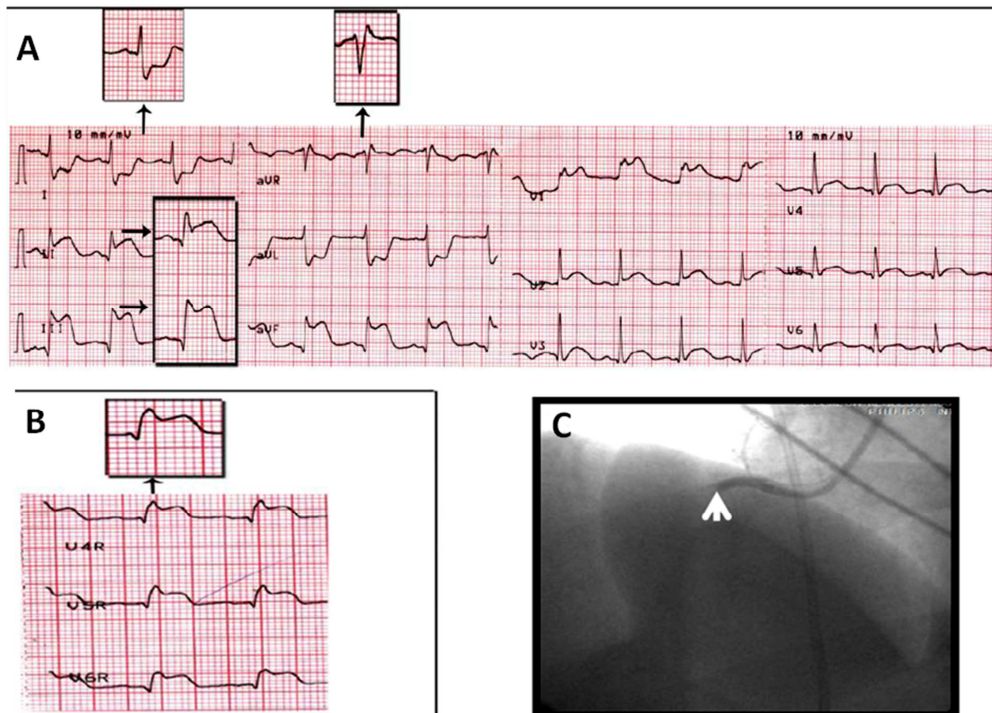


Figure 2. (A) 12 lead ECG tracings of patient in Group I showing ST segment elevation in lead III more than that in lead II, absence of ST segment depression in lead aVR and presence of ST segment depression in lead I, (B) Right ECG leads showing ST segment elevation and (C) coronary angiography showing totally occluded proximal RCA.

The sensitivity, specificity and predictive values of various ECG criteria in detection of culprit artery are listed in table 3

Table 3. Sensitivity, specificity and predictive values of various ECG criteria in detection of culprit coronary artery.

ECG Criteria	Sensitivity	Specificity	Positive predictive value	Negative predictive value
ST -segment elevation in lead III > lead II (RCA)	78%	60%	88%	41%
ST -segment depression in lead I ≥ 0.05 mm (RCA)	71%	65%	89%	36%
ST- segment depression in lead aVR ≥ 0.1 mm (LCX)	60%	81%	44%	89%
ST- segment elevation in lead V ₄ R ≥ 1 mm (RCA)	37%	100%	100%	40%

Subgroups:

Using ST segment depression ≥ 1 mm in aVR, patients in the 2 groups were reclassified into subgroups:

Group I_A: included 15 patients (19%) with ST segment depression ≥ 1 mm in aVR,

Group I_B: included 46 patients (81%) without ST segment depression ≥ 1 mm in aVR,

Group II_A included 12 patients (60%) with ST segment depression ≥ 1 mm in aVR and

Group II_B included 8 patients (40%) without ST segment depression ≥ 1 mm in aVR

Echocardiographic parameters and peak levels of CK and CK-MB were compared between the 4 subgroups (table 4).

Table 4. Echocardiographic parameters, CK and CK-MB in all subgroups.

	Group I n= 79			Group II n= 20		
	I _A (n= 15) (19%) with ST segment depression ≥ 1mm in aVR	I _B (n= 64) (81%) without ST segment depression ≥ 1mm in aVR	P	II _A (n= 12) (60%) with ST segment depression ≥ 1mm in aVR	II _B (n= 8) (40%) without ST segment depression ≥ 1mm in aVR	P
LV-EF (%)	56.16±5.22	55.12±5.63	NS	55.78±4.32	55.14±5.88	NS
WMSI	1.20 ±0.24	1.22 ±0.54	NS	1.23±0.14	1.24 ±0.22	NS
TAPSE (cm)	2.13 ± 0.32cm	1.94 ±0.18cm	NS	2.17 ± 0.25cm	2.04 ±0.28cm	NS
Peak CK (U/L)	2193.3±689.5	2176.5±514.2	NS	2110.9±534.7	2089.5±502.8	NS
CK-MB (ng/ml)	191.9±68.1	174.8±34.2	NS	188.3±59.6	179.6±48.6	NS

In Group I, the mean peak value of both CK and CK-MB was significantly higher in patients with ST segment depression in lead aVR (Group I_A) than those without ST segment depression in lead aVR (Group I_B) (2193.3 \pm 68 U/L,

191.9 \pm 68.11 ng/ml vs. 2176.5 \pm 51 U/L, 174.8 \pm 34.2 ng/ml; P value 0.02). All echocardiographic parameters showed no significant differences between both subgroups. In Group II, the mean peak value of both CK and CK-MB showed no

significant difference in both subgroups (Group II_A and Group II_B). Also, all echocardiographic parameters showed no significant differences between both subgroups.

4. Discussion

In acute myocardial infarction, the first direct diagnosis is made using the surface ECG. It gives the physician directly the location of the infarct, its extent and basically the artery that has been occluded [14]. 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 [15-18]. The present study aimed to evaluate different ECG criteria to detect culprit artery and site of occlusion in cases of inferior wall MI.

In concordant with the previous studies [19-21], our study showed RCA was the culprit artery in 79% of patients much more than LCX (20%); (Ratio of 3.95:1). The selection of the ECG criteria for the differentiation between LCX and RCA occlusions was based on anatomical distribution of myocardial segments supplied by both. 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 in cases of acute inferior wall MI [22-24].

Using ST depression in lead aVR \geq 1mm as a criterion for LCX occlusion, sensitivity and specificity were 60% and 81% respectively in our study. Previous studies [25-27] reported the same criterion with average sensitivity of 66.6% and specificity of 89.6%. This can be explained by the direction of the current of injury to the axis of lead aVR which forms right angle with RCA occlusion and an obtuse angle with LCX occlusion.

In consistent with the previous studies [19, 27], our study showed that ST segment elevation in lead III more than in lead II as a criterion of RCA occlusion had sensitivity of 78% and specificity of specificity 60%. The influence of lead III by RCA related acute inferior wall MI is due to its orientation to the right inferior segment while 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 LV [23].

Lead I is more oriented to lateral segments of the LV. 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 LV. In cases of acute inferior wall MI due to LCX occlusion, ST segment depression in lead I is less prominent and even ST elevation can appear [28]. Previous studies described ST segment depression in lead I $>$ 0.05mm for prediction RCA occlusion with an average sensitivity of 80.5% and specificity of 61% [20, 23]. Our study detected less sensitivity (71%) and comparable specificity (65%) of this criterion for RCA occlusion. Our study showed that ST segment elevation in lead V4R \geq 1mm had an excellent specificity (100%) and low sensitivity (37%) for RCA occlusion.

For the prediction of the site of occlusion within the RCA,

our study showed a higher sum of ST segment elevation in lead II, III and aVF in proximal RCA than both mid RCA and distal RCA (P value $<$ 0.01). ST segment elevation in lead V4R \geq 1mm as a criterion for proximal RCA involvement showed high both sensitivity (80%), and specificity (90%). Previous studies reported similar findings [29, 30].

5. Conclusion

In acute inferior wall MI, ST segment depression in lead aVR can be used for predicting LCX occlusion and associated with larger infarct size. For predicting RCA occlusion, ST segment elevation in lead III more than in lead II and ST segment depression in lead I $>$ 0.05mm can be used. The amplitude of ST segment elevation was significantly related the proximal RCA lesion.

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