



## Original Article

# Assessment of validity of the ‘Culprit Score’ for predicting the culprit lesion in patients with acute inferior wall myocardial infarction

Abhisekh Mohanty<sup>a,\*</sup>, R.K. Saran<sup>b</sup><sup>a</sup> Department of Cardiology, Continental Hospitals, Hyderabad, India<sup>b</sup> Department of Cardiology, King George Medical University, Lucknow, India

## ARTICLE INFO

## Article history:

Received 2 January 2016

Accepted 13 April 2016

Available online 4 May 2016

## Keywords:

Acute inferior wall myocardial infarction

Infarct-related artery

Electrocardiographic criteria

Culprit Score

## ABSTRACT

**Introduction:** Many electrocardiographic criteria have been developed to determine the infarct-related artery in acute inferior wall myocardial infarction. The aim of this study was to test the commonly used criteria and devise a simplified score to further improve the diagnostic accuracy.

**Materials and methods:** From 2011 to 2013, 100 patients with acute inferior wall myocardial infarction were recruited for electrocardiographic and angiographic analyses.

**Results:** The mean age of the patients was  $65 \pm 12$  years with 74% of patients being male. In our study population, significantly more ST-segment depression was seen in lead aVL and ST elevation in lead III in those with right coronary artery (RCA) occlusions. In left circumflex artery (LCX) occlusions, significantly more ST depression was seen in leads V1–3 (most significantly in lead V2) and ST elevation in lead II. In addition, more prominent ST depression was seen in lead aVL and ST elevation in V1 in proximal RCA occlusions. Based on the findings, we devised a score named Culprit Score, which was defined as  $[(II - V2/III + V1 - aVL)]$ . The sensitivity and specificity of Culprit Score  $\leq 0.5$  to predict proximal RCA occlusions;  $0.5$  to  $\leq 1.5$  to predict distal RCA occlusions; and score  $> 1.5$  to predict LCX occlusions were 85% and 85%; 80% and 86%; and 80% and 94%, respectively. Similarly, the negative predictive value was more than 80%.

**Conclusion:** The Culprit Score was found to have high specificity and negative predictive value to identify the infarct-related artery in inferior wall myocardial infarction.

© 2016 Cardiological Society of India. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## 1. Introduction

Early identification of the culprit artery in patients with symptoms of acute myocardial infarction (MI) could reduce the time to reperfusion and permit a better risk stratification. Many algorithms have been proposed to predict the infarct-related artery (IRA) based on electrocardiogram (ECG).

If specific ECG patterns can be recognized, it will be possible to determine the culprit coronary artery and the size of the ventricular area that is jeopardized. In inferior wall MI the culprit vessel is either the right coronary artery (RCA) or the left circumflex coronary artery (LCX). Rarely, occlusion of Type I ‘wrap-around’ distal LAD can also give rise to inferior wall MI. In patients with inferior wall MI who have right ventricular infarction, the culprit artery virtually always is the RCA, and these

patients are at increased risk for death, shock, and arrhythmias like atrioventricular block.<sup>1,2</sup> Early recognition of the culprit artery may help us in anticipating the possible complications and planning the line of management.

Inferior wall myocardial infarction classically manifests as ST elevations (STE) in inferior leads, together with ST changes in other leads that may represent either concomitant ischemia of other areas or simply reciprocal changes. However, the ECG changes at any point of time are the result of the positive and negative contributions of an infinity of variously directed vectors representing the direction of electric forces. Due to these ‘cancellation’ effects, the relation between ECG changes and the location and extent of myocardial injury is very complex, which is detrimental to the proper identification of the culprit artery by means of ECG parameters.

There are a few reported electrocardiographic criteria capable of identifying the infarct-related artery with high accuracy.<sup>3–9</sup> These criteria are simple, focusing on one or two leads, and user-friendly. However, the same prediction accuracy may not be reproducible in other populations because of heterogeneity in

\* Corresponding author at: Duplex No. 21, Suraj Vihar, Bargada Brit Colony, Brahmeshwar Patna, Bhubaneswar, Odisha 751018, India. Tel.: +91 9666121115.  
E-mail address: [abhisekh.mhnt@gmail.com](mailto:abhisekh.mhnt@gmail.com) (A. Mohanty).

patients' baseline characteristics and variations in the individual's coronary anatomy. Analysis of multiple electrocardiographic leads may instead plausibly minimize the errors and improve the diagnostic accuracy.

The purpose of this descriptive study was to devise a new and more accurate ECG algorithm to identify the culprit lesion in inferior wall MI by taking into consideration the reciprocal changes.

## 2. Materials and methods

In this prospective study, we enrolled patients admitted in the Emergency Department (ED) of King George Medical University (Lucknow, India) between January 2012 and January 2014 because of acute inferior wall STEMI. The diagnosis of acute inferior wall STEMI, was made on the basis of standard 12-lead ECG showing at least 1 mm ST elevation in at least 2 of the inferior leads (II, III, and aVF). They subsequently underwent coronary angiography during their hospitalization. ST changes were measured at 80 ms from J points, and the TP segment was the isoelectric line of the ECG. We excluded multivessel coronary artery disease, left bundle branch block, left ventricular hypertrophy, prior history of myocardial infarction and cases of inferior wall STEMI due to occlusion of "wrap-around" (Type I) left anterior descending (LAD) at distal parts.

The admission ECG of patients of ST-elevation inferior myocardial infarction was recorded and subsequently was correlated with the angiography.

The study consisted of 2 parts. In the first part, the magnitude of ST displacement was compared between LCX and RCA occlusion in order to find out the leads that have discriminative value. The sensitivity, specificity, and the predictive values of the commonly used algorithms to localize the infarct-related artery in inferior wall myocardial infarction were determined. In the second part, we will try to devise an algorithm whose accuracy will be compared with the most specific and sensitive ECG findings based on the results of the first part.

Angiographic findings were evaluated by an independent investigator blinded to patients' clinical and electrocardiographic data. The infarct-related lesion was identified either by (i) total occlusion or a significant stenosis ( $\geq 70\%$  of diameter narrowing) of the LCX or RCA or their branches, or (ii) angiographic evidence of an intraluminal thrombus. The diameter stenosis in non-thrombus containing lesions was measured by quantitative coronary angiography (QCA). Proximal RCA was defined as the coronary segment that lay proximal to the first RV branch based on the American College of Cardiology/American Heart Association guidelines.

### 2.1. Statistical analysis

We reported continuous variables in terms of means  $\pm$  standard deviation and for discrete variables, frequencies and percentages. We compared means of continuous variables with independent samples *t*-test. Correlations of discrete variables were measured by chi-square test of independence. *P* value  $< 0.05$  was considered statistically significant. Data analysis was performed using the SPSS version 13.0 statistical package (Chicago, IL). For all criteria, sensitivity, specificity, and predictive values were calculated.

## 3. Results

There were altogether 100 patients who met the inclusion criteria and were included within the study period. All patients were of Indian ethnicity, the mean age was  $65 \pm 12$  years with male predominance (74%). The baseline characteristics of the patients have been shown in Table 1.

**Table 1**  
Baseline features of the patients ( $n = 100$ ).<sup>a</sup>

Characteristics	Group 1 (RCA) $n = 74$	Group 2 (LCX) $n = 26$
Age	$65 \pm 12$	$63 \pm 10$
Male gender	56 (75.6)	18 (69.2)
Hypertension	35 (47.2)	14 (53.8)
Diabetes mellitus	34 (45.9)	14 (53.8)
Dyslipidemia	10 (13.5)	6 (23)
Smoking	62 (83.7)	17 (65.3)
Family history of coronary artery disease	3 (4)	2 (7.6)

<sup>a</sup> Data in this table are presented as mean  $\pm$  SD or number (%).

In the study population, the number of patients who underwent primary angioplasty was 28 and the number of patients receiving fibrinolytic therapy as the primary modality of treatment was 63.

In our study population, the number of patients with RCA lesion was 74, of which 40 had proximal RCA lesion and 34 had distal RCA lesion. Rest of the 26 patients had circumflex artery lesion. In the comparison of LCX and RCA occlusions, the former group had significantly more ST depression in lead V1–V3, and ST elevation in lead V6, whereas the latter group had more ST depression in lead I and aVL (Table 2). While comparing the groups with proximal and distal RCA occlusions, it was found that the former group had more ST depression in lead I and more ST elevation in lead V1 (Table 3). Based on these results, we devised a score called 'the Culprit Score'. It was defined as  $(II - V2)/(III + V1 - aVL)$ . If the denominator was  $\leq 0$  and the numerator was  $> 0$ , then it was considered to determine LCX artery involvement and the Culprit Score was arbitrarily considered to be 5. The mean Culprit Scores in proximal and distal RCA, and LCX lesions have been listed in Table 4. The mean score in

**Table 2**  
Comparison of ST segment displacements between right coronary artery (RCA) and left circumflex artery (LCX) occlusions.

Leads	RCA occlusion	LCX occlusion	<i>P</i> value
I	$-2.55 \pm 1.39$	$-1.61 \pm 0.40$	0.04
II	$2.20 \pm 1.63$	$2.82 \pm 1.48$	0.15
III	$3.97 \pm 1.91$	$3.04 \pm 1.49$	0.07
aVR	$3.27 \pm 0.76$	$2.93 \pm 1.47$	0.45
aVL	$-1.27 \pm 0.83$	$-0.21 \pm 1.07$	$< 0.01$
aVF	$1.36 \pm 0.83$	$1.59 \pm 1.02$	0.54
V1	$0.08 \pm 1.12$	$-1.93 \pm 1.64$	$< 0.01$
V2	$-0.85 \pm 2.30$	$-3.53 \pm 2.84$	$< 0.01$
V3	$-1.16 \pm 2.13$	$-3.57 \pm 2.56$	$< 0.01$
V4	$-0.84 \pm 2.09$	$-1.79 \pm 2.34$	0.18
V5	$-0.35 \pm 1.62$	$0.21 \pm 1.91$	0.32
V6	$0.10 \pm 1.34$	$1.21 \pm 1.50$	0.02

**Table 3**  
Comparison of ST segment displacement between proximal and distal right coronary artery (RCA) occlusions.

Leads	Proximal RCA occlusion	Distal RCA occlusion	<i>P</i> value
I	$-1.57 \pm 0.86$	$-0.83 \pm 0.57$	$< 0.01$
II	$2.47 \pm 1.58$	$2.54 \pm 1.74$	0.88
III	$4.00 \pm 1.88$	$3.67 \pm 1.96$	0.51
aVR	$3.29 \pm 1.72$	$3.25 \pm 1.87$	0.93
aVL	$-2.71 \pm 1.45$	$-2.31 \pm 1.30$	0.25
aVF	$-0.18 \pm 0.79$	$-0.62 \pm 0.83$	0.04
V1	$0.51 \pm 0.98$	$-0.56 \pm 1.02$	$< 0.01$
V2	$-1.00 \pm 2.35$	$-1.40 \pm 1.79$	0.44
V3	$-0.73 \pm 2.67$	$-1.04 \pm 1.64$	0.56
V4	$-0.84 \pm 2.42$	$-0.83 \pm 1.53$	0.98
V5	$-0.46 \pm 1.72$	$-0.19 \pm 1.47$	0.51
V6	$-0.07 \pm 1.28$	$0.35 \pm 1.41$	0.24

**Table 4**  
Distribution of Culprit Score according to the infarct locations.

Culprit Score	Proximal RCA (n = 44)	Distal RCA (n = 30)	LCX (n = 26)
Total score	0.55 ± 0.53	0.89 ± 0.61	4.23 ± 3.94
Culprit Score ≤ 0.5	34	4	2
0.5 < Culprit Score ≤ 1.5	8	24	3
Culprit Score > 1.5	2	2	21

proximal RCA occlusions was significantly lower than distal RCA occlusions ( $P = 0.02$ ). Similarly the score was found to be significantly lower when RCA is the culprit vessel as compared to LCX ( $P < 0.001$ ).

The Culprit Score was used in this study to not only accurately localize the infarct-related artery as RCA or LCX but also proximal or distal RCA lesions. It was found that most patients with proximal RCA involvement had Culprit Score  $\leq 0.5$ , those with distal RCA involvement had score between 0.5 and 1.5, and those with LCX artery involvement had score  $> 1.5$  (Table 4).

The accuracy of Culprit Score in identifying the infarct-related artery is shown in Table 5. In our study we found that the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of the Culprit Score of  $\leq 0.5$  determining proximal RCA lesions were 85%, 85%, 77%, and 90%, respectively.

Similarly, the corresponding values for Culprit Score of 0.5–1.5 for distal RCA lesions were 80%, 86%, 68%, and 92%, respectively. When we calculated the Culprit Score for LCX artery lesions the corresponding values were 80%, 94%, 84%, and 93%, respectively.

We also compared the accuracy of Culprit Score with the other commonly used ECG criteria for infarct-related artery localization. The Culprit Score was found to be more accurate in terms of specificity and negative predictive value.

#### 4. Discussion

Inferior wall MI due to RCA occlusion frequently presents with STE in leads II, III, and aVF, with reciprocal ST-segment depression (STD) in leads I and aVL. The ECG changes in LCX occlusions are highly variable. Approximately 30–50% of patients present with STE, usually in the inferior leads II, III, and aVF. Others show STD in leads V1–V4, or occasionally a tall R wave in lead V1. In up to 38% of patients, there is no discernible STE.<sup>10</sup>

To understand the ECG changes in acute inferior wall MI, we need to understand the anatomy of coronary arteries. In most cases, the RCA terminates into a posterior descending artery and a few posterolateral branches to supply the inferior myocardium and the inferior part of the inferoposterior wall, respectively, whereas the LCX gives off a number of obtuse marginal branches to supply the posterior part of the inferoposterior as well as posterolateral 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. The axes of leads III and aVF are directed inferiorly, and therefore, STE is more prominently seen in these leads in RCA occlusions. On the other hand, since the leads I and aVL are oriented laterally, STD are less prominent in these leads in LCX artery occlusions.<sup>8</sup> Precordial leads (V1–3) STD is common in inferior myocardial infarction, and explained either by reciprocal changes or various degrees of inferoposterior wall ischemia.<sup>11,12</sup> The injury current vectors between anterior and posterior walls are more strongly opposed than that between anterior and inferior walls. Therefore, LCX occlusions produce more prominent precordial STD than distal or proximal RCA occlusions. Proximal RCA occlusions are known to be associated with STE in V1 due to right ventricular infarction.<sup>13,14</sup> The degree of STE has been found to be significantly more in proximal as compared to distal RCA occlusions.

In reality, there are great variations in coronary anatomy amongst individuals, such as the relative size of vessels and the degree of dominance. Acute occlusion in a segment of a coronary artery may not produce the “expected” changes in a particular lead because of other anatomical opposing factors. By combining several leads, the error caused by variations in a single lead may be plausibly minimized.

The construction of Culprit Score [ $II - V2/III + V1 - aVL$ ] was based on the ratio of II/III suggested by Chia et al. and Herz et al.<sup>8</sup> It was chosen as the backbone of the score because they are positive numbers by definition of inferior ST elevation myocardial infarction. The leads that are found to be of discriminative value (differentiate between RCA and LCX occlusion and between proximal and distal RCA occlusion) are either added to or subtracted from the numerator and/or the denominator. The subtraction of lead V2 from lead II in the numerator exaggerates the influence of LCX occlusion; whereas the addition of lead V1 to and subtraction of lead aVL from lead III in the denominator increase the effect of proximal RCA occlusion and reduce that of distal RCA occlusion.

Previous studies have already described various ECG signs to identify the infarct-related artery. For instance, (1) ST-segment depression in lead I  $\geq 0.5$  mm, (2) ratio of ST-segment elevation in II/III  $< 1$ , (3) ratio of ST-segment depression in V3/ST-segment elevation in III  $< 1.2$ , (4) ST-segment elevation in V4R and (5) ST-segment depression in aVL  $> I$  predicts RCA occlusion.<sup>3–8</sup> By contrast (1) ST-segment isoelectric or elevation in lead I, (2) ratio of ST-segment elevation in II/III  $> 1$  and (3) ratio of ST-segment depression in V3/ST-segment elevation in III  $> 1.2$  predicts LCX occlusion.

We tested the accuracy of some of the commonly used criteria in our study population. We found that the criteria STE in III  $> II$  determining RCA lesion had a sensitivity and positive predictive value of 81% and 84%, respectively, but the specificity and negative predictive value were only 56% and 58%, respectively.

Similarly the sensitivity and positive predictive value of the criterion that STD in V3/STE III  $< 0.5$  determines proximal RCA

**Table 5**  
Accuracy of Culprit Score.

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Culprit Score $\leq 0.5$ predicts proximal RCA occlusion	85	85	77	90
0.5 < Culprit Score $\leq 1.5$ predicts distal RCA occlusion	80	86	68	92
Culprit Score $> 1.5$ predicts LCX occlusion	80	94	84	93
III $\geq II$ predicts RCA occlusion	81	56	84	58
ST $\downarrow$ V3/ST $\uparrow$ III $< 0.5$ predicts proximal RCA occlusion	86	56	79	59
0.5 $\leq$ ST $\downarrow$ V3/ST $\uparrow$ III $\leq 1.2$ predicts distal RCA occlusion	80	68	78	61
ST $\downarrow$ V3/ST $\uparrow$ III $> 1.2$ predicts LCX occlusion	86	63	80	65
ST $\downarrow$ in aVL $> I$ predicts RCA occlusion	79	70	81	70
Sum of ST $\downarrow$ in V1–3/ST $\uparrow$ in inferior leads $< 1$ predicts RCA occlusion	90	45	83	51

lesion were 86% and 79%, respectively. But the specificity and negative predictive value of this criterion were again found to be low (56% and 59%, respectively). When we analyzed the same ratio with value between 0.5 and 1.2 for distal RCA and value  $>1.2$  for LCX lesion, we found almost similar results (Table 5).

The criteria of  $aVL > I$  to localize RCA lesion was analyzed in our study population. We found a sensitivity and positive predictive value of 79% and 81%, and specificity and negative predictive value of 70%.

Similarly the other criteria were also tested (Table 5). Based on our analysis, we can say that the criteria were sensitive but most of them had a low specificity and a negative predictive value.

Hence, we needed a better electrocardiographic criteria to differentiate between the LCX artery and RCA lesions. So our trial was designed in such a way that in the first phase we analyzed the ECG leads with maximum ST-segment elevation or depression. Then we devised a score called the Culprit Score taking into account leads with maximum ST-segment elevation and those with maximum reciprocal ST segment depression.

In our study we found that the sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of the Culprit Score of  $\leq 0.5$  determining proximal RCA lesion were 85%, 85%, 77%, and 90%, respectively (Table 5).

Similarly the corresponding values for Culprit Score of 0.5–1.5 for distal RCA lesions were 80%, 86%, 68%, and 92%, respectively.

When we calculated the Culprit Score for LCX artery lesion the corresponding values were 80%, 94%, 84%, and 93%, respectively.

Based on these results, we can say that the culprit has a sensitivity and a positive predictive value almost similar to the previously described criteria; but when we analyzed the specificity and the negative predictive value of this score, they were  $>85\%$  for lesions in proximal RCA, distal RCA, or LCX artery. Hence, the 'Culprit Score' can be useful before doing angiography to accurately predict the location of the culprit lesion in acute inferior wall myocardial infarction.

## 5. Conclusions

In our study, we found that all the commonly used electrocardiographic criteria had a low specificity and negative predictive value to differentiate between LCX artery and RCA lesions in acute inferior wall myocardial infarction. Most of the studies carried out so far had considered only one ECG criteria. Based on ST-segment changes, we devised a new score which we called the 'Culprit Score'. It was found to have a comparatively higher specificity and predictive value for localizing both RCA and LCX lesions as compared to the previously used criteria.

## 5.1. Study limitations

Obviously, inferior STEMI is not always limited to just one vessel occlusion and in one site; thus, this study is less helpful to determine the exact value of the examined criteria in patients with multi-vessel involvement. Small number of patients, especially patients with LCx occlusion, is another limitation in these kinds of studies.

## Conflicts of interest

The authors have none to declare.

## References

1. Braat SH, de Zwaan C, Brugada P, Coenegracht JM, Wellens HJ. Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of developing atrioventricular nodal conduction disturbances. *Am Heart J*. 1984;107:1183–1187.
2. Zehender M, Kasper W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. *N Engl J Med*. 1993;328:981–988.
3. Prieto-Solis JA, Martin-Duran R, San Jose JM. A new electrocardiographic approach to identify the coronary artery responsible for acute inferior myocardial infarction: a study in 100 patients. *Rev Port Cardiol*. 1998;17(February):133–142.
4. 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;51:281–284.
5. Prieto-Solis JA. Diagnostic value of the arithmetic sum of the ST segment of inferior and V2 leads, II + V2, III + V2 and aVF + V2 in identifying the artery responsible for inferior acute myocardial infarction. *Angiology*. 1995;46:885–894.
6. Kosuge M, Kimura K, Ishikawa T, et al. New electrocardiographic criteria for predicting the site of coronary artery occlusion in inferior wall acute myocardial infarction. *Am J Cardiol*. 1998;82:1318–1322.
7. Jim MH, On-On Chan A, Wong CP, et al. Clinical implications of precordial ST-segment elevation in acute inferoposterior myocardial infarction caused by proximal right coronary artery occlusion. *Clin Cardiol*. 2007;30:331–335.
8. 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 Cardiol*. 1997;80:1343–1345.
9. Fiol M, Cygankiewicz I, Carrillo A, et al. Value of electrocardiographic algorithm based on "ups and downs" of ST in assessment of a culprit artery in evolving inferior wall acute myocardial infarction. *Am J Cardiol*. 2004;94:709–714.
10. O'Keefe Jr JH, Sayed-Taha K, Gibson W, et al. Do patients with left circumflex coronary artery-related acute myocardial infarction without ST-segment elevation benefit from reperfusion therapy. *Am J Cardiol*. 1995;75:718–720.
11. Wasserman AG, Ross AM, Bogaty D, Richardson DW, Hutchinson RG, Rios JC. Anterior ST segment depression during acute inferior myocardial infarction: evidence for the reciprocal change theory. *Am Heart J*. 1983;106:516–520.
12. Wong CK, Freedman SB, Bautovich G, Bailey BP, Bernstein L, Kelly DT. Mechanism and significance of precordial ST-segment depression during inferior wall acute myocardial infarction associated with severe narrowing of the dominant right coronary artery. *Am J Cardiol*. 1993;71:1025–1030.
13. Khan ZU, Chou TC. Right ventricular infarction mimicking acute anteroseptal left ventricular infarction. *Am Heart J*. 1996;132:1089–1093.
14. Porter A, Herz I, Strasberg B. Isolated right ventricular infarction presenting as anterior wall myocardial infarction on electrocardiography. *Clin Cardiol*. 1997;20:971–973.