

The Diagnostic Value of 12-Lead Electrocardiogram in Predicting Infarct-Related Artery and Right Ventricular Involvement in Acute Inferior Myocardial Infarction

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Background: The aim of the present study was to investigate the predictive value of presentation and 24-hour electrocardiograms in defining the infarct-related artery (IRA), its lesion segment, and the right ventricular involvement in acute inferior myocardial infarction (MI).

Methods: One hundred forty-nine patients with acute inferior MI were included. Infarct-related artery, its lesion segment, and the validity of new ECG criteria for the diagnosis of right ventricular MI (RVMI) were investigated by means of criteria obtained from admission and 24-hour ECGs.

Results: The presence of ST-segment elevation in lead III > lead II criterion (Criterion 1) and ST-segment depression in lead I > lead aVL criterion (Criterion 2) from admission ECG defined the right coronary artery (RCA) as IRA with a sensitivity of 64% and a specificity of 100%. These two criteria also defined the proximal or mid lesions in RCA as culprit lesions (sensitivity of 99%, specificity of 96%). Absence of these two criteria indicated Cx as IRA with a sensitivity of 50% and a specificity of 97%. The depth of Q wave in lead III > lead II criterion (Criterion 3) had no value for discrimination of IRA, but the width of Q wave in lead III > lead II criterion (Criterion 4) supported the RCA to be IRA with a sensitivity of 60% and a specificity of 61% (Criteria 3 and 4 were obtained from 24-hour ECGs). The finding of Criterion 1 plus Criterion 5 (ST elevation in V₁ but no ST elevation in V₂) on admission ECG had a sensitivity of 63% and a specificity of 99% in the diagnosis of RVMI.

Conclusion: We concluded that 12-lead ECG is a cheap, easy, and readily obtainable diagnostic approach in discrimination of IRA and its culprit lesion segment. However, despite high specificity, due to moderate degree sensitivity, its value for the diagnosis of RVMI is questionable.

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Despite recent advances in expensive and complex diagnostic and treatment modalities, electrocardiography (ECG) remains a simple and cheap diagnostic tool in clinical cardiology. In parallel to the

advances in other methods, more information has been attempted from surface electrocardiographic findings. There are several studies that investigated the ECG findings in acute inferior wall myocardial

infarction (MI). Some of these studies were designed to differentiate the right coronary artery (RCA) or the circumflex artery (Cx) as the infarct-related artery (IRA),¹⁻⁸ whereas others suggested new criteria for the diagnosis of right ventricular MI (RVMI).^{2,7-11} However, the results are contradictory.

The aim of the present study was to investigate the predictive value of presentations and 24-hour electrocardiograms in defining the IRA, lesion segment of IRA, and the RVMI in the setting of acute inferior wall MI.

METHODS

Subjects

The records of 361 patients admitted to the Coronary Care Unit in our institution between January 1994 and August 1999 and underwent coronary angiography were analyzed; 149 who satisfied the inclusion and exclusion criteria were selected. The inclusion criteria were (1) typical chest pain with a duration of > 30 minutes but < 6 hours from the onset; (2) minimum 0.1 mV ST-segment elevation in at least two of the inferior leads (leads II, III, and AVF); (3) increase in serum creatinine kinase myocardial band (CK-MB) at least two times the upper limit of normal. Patients with the history of remote MI, bypass surgery or angioplasty, as well as those with ECG findings of left ventricular hypertrophy, intraventricular conduction defect, bundle branch block, ventricular or pacemaker rhythm were excluded.

Presentation and 24-hour electrocardiograms and coronary angiographic findings were evaluated. After the definition of certain ECG criteria; their predictive value for IRA, lesion segment of IRA, and the presence of RVMI were tested.

Electrocardiographic Examinations

ST segment changes were evaluated 60 ms after J point with the TP segment taken as the baseline and changes > 0.1 mV were considered to be significant. The following criteria were evaluated from baseline (Criteria 1-2, 5) and 24-hour (Criteria 3-4) ECGs.

Criterion 1: ST-segment elevation in lead III > lead II

Criterion 2: ST-segment depression in lead aVL > lead I

Criterion 3: Depth of Q wave in lead III > lead II

Criterion 4: Width of Q wave in lead III > lead II

Criterion 5: The presence of greater than 0.5 mV ST-segment elevation in lead V₁ associated with the absence of ST-segment elevation in lead V₂.

These hypotheses were tested with the use of the criteria described above:

Hypothesis 1: The presence of Criterion 1 and Criterion 2 supports the RCA as IRA, discards the Cx to be IRA. In addition it points to the RCA proximal and mid lesions.

Hypothesis 2: The absence of Criterion 1 and Criterion 2 supports the Cx as IRA and discards the RCA to be IRA.

Hypothesis 3: The presence of Criterion 3 supports the RCA as IRA.

Hypothesis 4: The presence of Criterion 4 supports the RCA as IRA.

Hypothesis 5: The presence of both Criterion 1 and Criterion 5 supports the diagnosis of RVMI.

The presence of greater than 1 mm ST-segment elevation in lead V_{4R} was considered to be diagnostic for the RVMI.⁸⁻⁹ Patients with this finding in their ECG were grouped as RVMI group and diagnostic value of Hypothesis 5 was tested in these patients.

Coronary Angiographic Examinations

All patients included in the study had undergone coronary angiography with standard techniques. The coronary angiography films were retrospectively evaluated and the patients were divided into two groups, as RCA Group or Cx Group, depending on the IRA. The IRA and its lesion segment were determined according to the following findings:

Total or subtotal occlusion in the coronary artery perfusing the left ventricular segment with contractile dysfunction;

Coronary lesion with fresh thrombus, rupture, dissection or ulceration.

Patients with significant lesions both in RCA and Cx arteries in whom IRA could not be identified were excluded from the study.

Left ventricular end-diastolic pressure (LVEDP)

values were noted as measures of left ventricular contractility. The occlusion site was recorded as proximal, mid, and distal depending on the branching points of the first right ventricular and acute marginal vessels for RCA. For the Cx artery, the first and second marginal branches were used as markers to make this differentiation.

Statistical Analysis

The Statistical Package for the Social Science (SPSS 9.1 version for Windows) was used for statistical analyses. Values were expressed as mean \pm SD and percentage when appropriate. For the measured parameters, statistical analyses for between-group differences were performed with the use of independent sample *t*-test. If the variables did not satisfy the criteria for parametric tests, the Mann-Whitney U test was used for statistical analysis. Prevalence data were compared by group with Pearson chi-square analysis or Fisher's exact chi-square test. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) measures were calculated to test the defined hypotheses. Statistical significance was set as $P < 0.05$.

RESULTS

Out of 149 patients included in the study (126 male, 23 female; mean age 55 ± 9), 64 (43%) were hypertensive, 42 (28%) had diabetes mellitus, and 43 (29%) had a history of hyperlipidemia (Table 1). One-hundred nineteen (80%) patients received thrombolytic therapy in the first 6 hours of chest pain.

Coronary Angiographic Findings

Coronary angiography was performed 13 ± 6 days (range 4-38 days) after an acute episode of MI. Based on coronary angiography, 123 (83%) patients were enrolled into the RCA Group and 26 (17%) into the Cx Group. The detailed anatomical distribution of the lesion segment of IRA was as follows: Proximal-RCA in 77 patients (52%), mid-RCA in 30 patients (20%), distal-RCA in 16 patients (11%), proximal-Cx in 18 patients (12%), and mid-Cx in 8 patients (5%).

The clinical characteristics of the patients in RCA and Cx Groups were similar except for the history of smoking which was more prevalent in the Cx group ($P = 0.036$) (Table 1).

The LVEDP was significantly higher in the RCA group compared to Cx Group (17 ± 6 mmHg vs 13 ± 5 mmHg, $P < 0.001$).

Determination of the Infarct-Related Artery

Hypotheses 1, 2, 3, and 4 were evaluated to determine the IRA.

Hypothesis 1 and hypothesis 2: The presence of Criteria 1 and 2 in RCA and Cx Groups are given in Table 2 with the results of statistical analysis summarized in Table 3. The presence of Criterion 1 had a sensitivity of 93% and a specificity of 62% for predicting RCA as IRA. The addition of Criterion 2 to Criterion 1 (hypothesis 1) increased specificity to 100% but had lower sensitivity (64%). The absence of these two criteria (hypothesis 2) predicted Cx as IRA with a sensitivity of 50% and a specificity of 97%.

As the second part of hypothesis 1, the presence

Table 1. Clinical Characteristics of Subjects in Study

	RCA Group	Cx Group	Total
Number of patients	123	26	149
Age (years)	55 ± 11	55 ± 9	55 ± 9
Male sex (n, %)	102 (83)	24 (92)	126 (85)
Family history (n, %)	71 (58)	14 (54)	85 (57)
Hypertension (n, %)	56 (46)	8 (31)	64 (43)
Diabetes mellitus (n, %)	36 (29)	6 (23)	42 (28)
Hyperlipidemia (n, %)	35 (28)	8 (31)	43 (29)
Smoking* (n, %)	90 (73)	24 (92)	114 (77)
Thrombolytic therapy (n, %)	95 (77)	24 (92)	119 (80)

* $P = 0.036$ (between RCA and Cx Groups). All other comparisons were nonsignificant. RCA = right coronary artery; Cx = circumflex artery.

Table 2. The Presence of Criteria 1, 2, 3, and 4 in Right Coronary Artery and Circumflex Groups (n, %)

	RCA Group (n = 123)	Cx Group (n = 26)	Total (n = 149)	P Value
Criterion 1+	114 (93)	10 (38)	124 (83)	< 0.0001
Criterion 2+	84 (68)	3 (12)	87 (58)	< 0.0001
Criteria 1+, 2+	79 (64)	0 (0)	79 (53)	< 0.0001
Criteria 1-, 2-	4 (3)	13 (50)	17 (11)	< 0.0001
Criterion 3+	113 (92)	22 (85)	135 (91)	NS
Criterion 4+	74 (60)	10 (38)	84 (56)	< 0.043

NS = nonsignificant; RCA = right coronary artery; Cx = circumflex artery.

of Criteria 1 and 2 were also evaluated to determine the lesion segment of RCA (Table 4), with the statistical analysis outlined in Table 5. The presence of these two criteria had a sensitivity of 99% and a specificity of 96% for predicting proximal and mid RCA lesions as IRA.

Hypothesis 3 and hypothesis 4: The presence of Criterion 3 (Hypothesis 3) and Criterion 4 (Hypothesis 4) in RCA and Cx groups are given in Table 2 with results of statistical analysis summarized in Table 3. Hypothesis 3 had a sensitivity of 92% but a very low specificity (16%) in determining RCA as IRA, whereas the sensitivity and specificity values of Hypothesis 4 were 60 and 61%, respectively.

Diagnosis of Right Ventricular Myocardial Infarction

A total of 27 patients had RVMI according to the presence of at least 1 mm ST-segment elevation in V4R; all were in the RCA Group (26 proximal-RCA and 1 mid-RCA). Of these 27 patients, Criterion 1

was present in 26 (96%) and Criterion 5 was present in 18 (67%) patients. Seventeen (63%) had both Criterion 1 and Criterion 5 (Hypothesis 5). The results of statistical analysis for Criterion 1, Criterion 2, and hypothesis 5 are summarized in Table 6. The presence of only Criterion 1 had a high sensitivity (96%), but low specificity (28%). The presence of only Criterion 5 had lower sensitivity (67%), but a very high specificity (95%). Accordingly, hypothesis 5 had a sensitivity of 63% and a specificity of 99% for the diagnosis of RVMI.

DISCUSSION

A greater majority of our patients (83%) had acute inferior MI due to RCA lesions. The baseline clinical characteristics of the patients included in the RCA and Cx Groups were similar except for the history of smoking. The increased LVEDP in the RCA Group compared to Cx Group can be explained with the RCA perfusing a larger myocardial area than the Cx artery.¹²

There are many studies in the literature which investigated several ECG criteria to define RCA or Cx as IRA in the setting of acute inferior MI.¹⁻⁸ In the present study, we evaluated presentation and 24-hour ECGs to define IRA, with the Criteria 1, 2, and 5 defined from presentation and Criteria 3 and 4 from 24-hour ECGs.

The presence of Criterion 1 or Criterion 2 was significantly higher in the RCA Group compared to Cx Group ($P < 0.0001$). More strikingly, the presence of both of these two criteria together was found only in the RCA Group and in none of the patients in the Cx Group ($P < 0.0001$). So Cx lesion was excluded as IRA in our study. Our findings are consistent with the studies of Herz et al.⁶ and Zimetbaum et al.⁷

The sensitivity and PPV of Criterion 1 was high

Table 3. Predictive Value of Hypotheses 1, 2, 3, and 4 for Defining Right Coronary Artery as Infarct-Related Artery (%)

	SENS	SPES	PPV	NPV
Criterion 1+	93	62	93	64
Criterion 2+	68	89	97	37
Criteria 1+, 2+	64	100	100	37
Criteria 1-, 2-*	3	50	24	10
Criteria 1-, 2-**	50	97	77	90
Criterion 3+	92	16	83	28
Criterion 4+	60	61	88	25

Hypothesis 1: Criteria 1+, 2+; Hypothesis 2: Criteria 1-, 2-; Hypothesis 3: Criterion 3+; Hypothesis 4: Criterion 4+.
* Predicting RCA as IRA; ** predicting Cx as IRA; SENS = sensitivity; SPES = specificity; PPV = positive predictive value; NPV = negative predictive value.

Table 4. Presence of Criteria 1 and 2 in Right Coronary Artery Segments (n, %)

	RCA-proximal (n = 77)	RCA-mid (n = 30)	RCA-distal (n = 16)	Total (n = 123)
Criterion 1+	75 (97)	26 (87)	13 (81)	114 (93)
Criterion 2+	62 (81)	16 (53)	6 (38)	84 (68)
Criteria 1+, 2+	62 (81)	14 (47)	3 (19)	79 (64)
Criteria 1-, 2-	2 (3)	2 (7)	0 (0)	4 (3)

RCA = right coronary artery.

in defining RCA as IRA (both were 93%), whereas the specificity and NPV were not that high (62 and 64%). On the other hand, Criterion 2 had higher specificity for defining RCA as IRA (89%), but it had low sensitivity (68%). The use of both of these criteria (hypothesis 1), on the other hand, had 100% specificity and 100% PPV for RCA lesions. The findings of Herz et al.,⁶ who investigated the same criteria, were parallel to our results. In that study, patients lacking these two criteria were in the Cx Group, but in our study four patients were in the RCA Group. So, our findings can only provide support but not completely agree with the idea of 'The absence of these two criteria exclude RCA lesions.' However, it should be kept in mind that the study group of Herz included only 66 RCA and 17 Cx lesions, whereas our study population consisted of 123 RCA and 26 Cx lesions. Although the ratio of IRAs was nearly the same, our study population was considerably larger.

Related to the lesion localization of the RCA, Criterion 1 had a high sensitivity (97%), but a low specificity (32%); however Criterion 2 had slightly

lower sensitivity (81%), but considerably higher specificity (65%) in defining proximal RCA lesions. The presence of both Criteria 1 and 2 did not further increase the sensitivity than the sensitivity of the presence of only Criterion 2. This finding was due to the presence of Criterion 1 in all patients with proximal RCA lesion in whom the Criterion 2 was already positive. The presence of both of these criteria had high sensitivity, specificity, PPV and NPV for proximal RCA lesions (81, 76, 78, and 79%, respectively). For proximal or mid lesions the presence of Criteria 1 and 2 together increased the sensitivity, specificity, PPV and NPV (99, 96, 96, and 99%, respectively) providing further support for the second part of Hypothesis 1.

What could be the logic behind the definitions of Criteria 1 and 2? Lead III directs to right inferior and lead II directs to left inferior and left inferolateral parts of left ventricle.⁵⁻⁶ Lead aVL represents the high lateral part of left ventricle and is a true reciprocal of inferior leads.⁶ The circumflex artery perfuses posterolateral and inferoapical regions of the left ventricle (the area between anterolateral and posteromedial papillary muscles). All other regions of the inferior wall, right ventricle, and inferior part of interventricular septum are perfused by RCA.¹² Therefore the primary changes (ST segment elevations) in RCA occlusions are seen in lead III with reciprocal changes (ST segment depressions)

Table 5. Predictive Value of Criteria 1 and 2 for Right Coronary Artery Segments (Proximal and Mid) as Infarct Related Artery (Second Part of Hypothesis 1) (%)

	SENS	SPES	PPV	NPV
Criterion 1+*	97	32	60	92
Criterion 1+**	94	45	81	76
Criterion 2+*	81	65	71	76
Criterion 2+**	73	79	90	93
Criteria 1+, 2+*	81	76	78	79
Criteria 1+, 2+**	99	96	96	99
Criteria 1-, 2-*	3	79	12	43
Criteria 1-, 2-**	5	82	24	45

* For proximal RCA lesions, ** for proximal + mid RCA lesions. SENS = sensitivity; SPES = specificity; PPV = positive predictive value; NPV = negative predictive value.

Table 6. Statistical Results for Presence of Criterion 1, Criterion 5, and Test of Hypothesis 5 (Criterion 1+, 5+)

	SENS	SPES	PPV	NPV
Criterion 1+	96	28	23	97
Criterion 5+	67	95	75	86
Criterion 1+, 5+	63	99	94	92

SENS = sensitivity; SPES = specificity; PPV = positive predictive value; NPV = negative predictive value.

seen in leads I and aVL (being more prominent in aVL). Circumflex artery lesions, on the other hand, affects posterolateral and apical regions; so we do not observe reciprocal changes in leads I and aVL. We might even see ST-segment elevations in these leads.^{3,6} However, the primary changes for Cx occlusions (ST segment elevations) are more prominent in lead II.⁶ In summary, for RCA lesions the primary changes in lead III and the reciprocal changes in lead aVL are prominent, whereas in Cx lesion the primary changes in lead II gains importance.

Two theories have been proposed to explain the formation of pathological Q waves.¹³ One possible explanation is that the infarct area is electrically inert and the negative electrical flow of the opposite wall causes a negative deflection (Q wave) in the leads of the infarct region. According to the second theory, Q wave develops due to the formation of a second vector with the same magnitude but opposite in direction from the infarct area. Hypothesis 3 (depth of Q wave in lead III > lead II) and hypothesis 4 (width of Q wave in lead III > lead II) describe a new method for defining IRA after the resolution acute phase of MI and the formation of pathological Q waves. These hypotheses can be prolonged further than 24 hours, based on the persistence of pathological Q-waves for years after acute MI.¹³ But according to the results of our study, the hypothesis 4 weakly supported the RCA as IRA (sensitivity 60% and specificity 61%). However, hypothesis 3 had no diagnostic value for IRA identification because of very low specificity (16%). These findings can be explained with the presence of living and necrotic myocardial segments in the same myocardial area and the heterogeneity of electrophysiological changes of that region.¹²

The most commonly accepted criterion for the diagnosis of RVMI is the ST-segment elevation in lead V4R.⁸⁻⁹ However, several other criteria have also been suggested:

1. Transient ST-segment elevation in lead V₁
2. ST-segment elevation in lead V₁ and ST-segment depression in lead V₂
3. ST-segment elevation in lead III > lead II
4. ST-segment elevation in leads V₁₋₅ together with minimal ST-segment elevation in inferior leads. ST-segment elevation in lead V₁ decreases towards V₆.
5. ST-segment depression in lead V₂ is smaller

than the 50% of the ST-segment elevation in lead aVF.

In our study the predictive value of the presence of Criterion 1 (ST segment elevation in lead III > lead II) and Criterion 5 (ST segment elevation in lead V₁, but no ST-segment elevation in lead V₂) at presentation ECGs were tested for the diagnosis of RVMI. The presence of only Criterion 1 had a high sensitivity (96%), but low specificity (28%). The presence of only Criterion 5 had lower sensitivity (67%) but a very high specificity (95%). In the presence of both of these criteria (Hypothesis 5), sensitivity is similar to Criterion 5 (63%), but specificity further increases to 99% with high PPV and NPV (94 and 92%, respectively). The accuracy of Hypothesis 5 was also tested by Zimetbaum et al.,⁷ but differing from our study they compared the ECG findings with echocardiography for the diagnosis RVMI. Hypothesis 5 was true in 50% of their study population, and in 40% of them the IRA was mid-RCA. They explained the incidence of mid-RCA lesion higher than expected to be due to the use of echocardiography for RVMI diagnosis. In our study, a majority of the patients with RVMI diagnosis had proximal RCA lesions. This might be related to the use of ST-segment elevation criterion in lead V4R for RVMI diagnosis. The finding of a higher incidence of proximal RCA lesion in patients with RVMI in our study is consistent with the results of Braat et al.⁸ According to our results, despite high specificity the clinical usefulness of hypothesis 5 is denied due to relatively lower sensitivity. So we recommend the conventional criteria using V4R for diagnosis of RVMI.

CONCLUSION

In acute inferior MI, 12-lead ECG at presentation is very important in defining IRA and lesion segment of IRA. The presence of ST-segment elevation in lead III > lead II and ST-segment depression in lead aVL > lead I criteria support the RCA as IRA, points to the proximal and mid-RCA lesions, and excludes the Cx as IRA. The absence of both of these two criteria, on the other hand, defines the Cx as IRA. Q wave width in lead III > lead II obtained at ECGs after the first 24 hours of acute MI poorly supports the RCA as IRA. However, the depth of Q wave in lead III > lead II has no diagnostic value.

The findings of ST-segment elevation in lead

III > lead II and the presence of ST-segment elevation in lead V₁ (without ST elevation in lead V₂) has high specificity but relatively lower sensitivity for RVMI diagnosis. Therefore its value is questionable compared to ST segment elevation in lead V4R criterion in RVMI.

REFERENCES

1. Sclarovsky S, Topaz O, Rechavia E, et al. Ischemic ST-segment depression in V2-V3 as the presenting electrocardiographic feature of posterolateral wall myocardial infarction. *Am Heart J* 1987;113:1085-1090.
2. Geft IL, Shah PK, Rodriguez L, et al. ST elevation in lead V1 to V5 may be caused by right coronary artery occlusion and acute right ventricular infarction. *Am J Cardiol* 1984;53:991-996.
3. Huey BL, Beller GA, Kaiser DL, et al. A comprehensive analysis of myocardial infarction due to left circumflex artery occlusion: Comparison with infarction due to right coronary artery and left anterior descending artery occlusion. *J Am Coll Cardiol* 1988;12:1156-1166.
4. Birnbaum Y, Sclarovsky S, Mager A, et al. ST segment depression in aVL: A sensitive marker for acute inferior myocardial infarction. *Eur Heart J* 1993;14:4-7.
5. Hasdai D, Birnbaum Y, Herz I, et al. ST segment depression in lateral limb leads in inferior wall acute myocardial infarction. Implication regarding the culprit artery and the site of obstruction. *Eur Heart J* 1995;16:1549-1553.
6. Herz I, Assali AR, Adler Y, et al. 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.
7. Zimetbaum PJ, Krishnan S, Gold A, et al. Usefulness of ST segment elevation in lead III exceeding that of lead II for identifying the location of the totally occluded coronary artery in inferior wall myocardial infarction. *Am J Cardiol* 1998;82:918-919.
8. Braat SH, Brugada P, Den Dulk K, et al. Value of V4R for recognition of the infarct coronary in acute inferior myocardial infarction. *Am J Cardiol* 1984;63:1538-1541.
9. Robalino BD, Whitlow PL, Underwood DA, et al. Electrocardiographic manifestations of right ventricular infarction. *Am J Cardiol* 1989;118:138-144.
10. Lew AS, Laramee P, Shah PK, et al. Ratio of ST segment depression in lead V₂ to ST segment elevation in lead aVF in evolving inferior acute myocardial infarction: An aid to the early recognition of right ventricular ischemia. *Am J Cardiol* 1986;57:1047-1051.
11. Isner JM. Right ventricular myocardial infarction. *JAMA* 1988;259:712-718.
12. Waller BF, Schlant RC. Anatomy of the heart. Chapter In Alexander RW, Schlant RC, Fuster V (eds.): *Hurst's The Heart Arteries and Veins*. 9th Edition. New York McGraw-Hill Book Company 1998, pp. 53-54.
13. Fish C. Electrocardiography. Chapter in Braunwald E (ed.): *Heart Disease A Textbook of Cardiovascular Medicine*. 5th Edition,. Philadelphia, WB Saunders Company 1997, pp. 107-136.