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New Electrocardiographic Score for Predicting the Site of Coronary Artery Occlusion in Inferior Wall Acute Myocardial Infarction

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Background: An electrocardiogram (ECG) was used to determine the type of acute myocardial infarction (MI) and locate the culprit vessel. Inferior wall myocardial infarction (IWMI) patients with the right coronary artery (RCA) as the culprit vessel may have a worse clinical prognosis than the left circumflex artery (LCx). We aimed to develop a new, simple, accurate scoring system to localize the RCA.

Methods: From January 2018 to January 2020, patients were admitted to the Department of Cardiology of TEDA International Cardiovascular Hospital and the Second Hospital of Tianjin Medical University due to IWMI and coronary angiography confirmed that the infarct-related vessel was a single RCA or LCx. ECG of patients before percutaneous coronary intervention (PCI) was collected to quantitatively analyze the characteristics of ST-segment deviation in non-inferior wall leads (N-IWL) and establish the RCA score in N-IWL.

Results: 149 patients were enrolled, including 83 in the RCA group and 66 in the LCx group. Finally, ST-segment depression (ST↓) lead I, aVR, V1, and V6, and ST↓≥ 1mm in lead V4 were found to be associated with the location of the RCA. The sensitivity, specificity, and area under the curve (AUC) of the N-IWL RCA scoring system were 77.1%, 72.7%, and 0.83, respectively. The diagnostic ability of the scoring system was better than that of other algorithms and scoring systems.

Conclusion: ECG helps identify the RCA in patients with IWMI before PCI. The N-IWL RCA score may help identify the culprit vessel as the RCA in patients with IWMI.

Keywords: electrocardiogram, inferior wall myocardial infarction, percutaneous coronary intervention, right coronary artery

Introduction

According to the World Health Organization data, although the mortality rate of coronary artery disease (CAD) has decreased, it is still one of the leading causes of death worldwide, which brings heavy social and economic burdens.[1,2] Inferior wall myocardial infarction (IWMI) caused by occlusion of the right coronary artery (RCA) and left circumflex artery (LCx) accounts for 40–50% of all patients with myocardial infarction (MI), and the in-hospital mortality is between 2%-9%. IWMI patients with non-inferior ST-segment deviation have higher postoperative complications, rehospitalization rate, and one-year mortality.[3]

MI patients whose culprit vessel is the RCA rather than the LCx may have a worse clinical prognosis. The RCA occlusion, especially when the myocardium supplied by the occluded vessel involves the right ventricle, increases the incidence of hemodynamic complications accompanied by arrhythmia, hypotensive shock, and increased risk of death.[4]

Early identification of the RCA as the culprit vessel may help make treatment decisions and prevent and manage complications after percutaneous coronary intervention (PCI).[5]

The role of the electrocardiogram (ECG) ST-segment shift in pinpointing culprit vessels was recognized as early as the 1980s.[6] The typical ECG findings of IWMI are ST-segment elevation (ST↑) in inferior leads and ST-segment deviation in non-inferior wall leads (N-IWL). However, the change in ECG at any time results from the integration of infinite positive and negative vectors. The relationship between ECG changes and the location and degree of MI is very complex. It is difficult to identify the culprit vessel using ECG parameters correctly. With research development, several diagnostic criteria have been proposed to determine the RCA as the culprit.[7–11] These criteria focus on one to three clues, are simple, and have high diagnostic efficacy. However, the prediction accuracy is difficult to replicate in other people due to the heterogeneity of the enrolled population, the differences in baseline characteristics of patients, and the differences in coronary artery anatomy. Comprehensive analysis of multiple ECGs leads to a simple algorithm and score that may help reduce errors and improve diagnostic accuracy.[7,12]

This study aims to identify the correlation between the N-IWL ST-segment deviation and the location of the RCA and then design a new, simple, and highly accurate scoring system for the localization of the RCA.

Materials and Methods

Patients and Inclusion-Exclusion Criteria

This retrospective, case-control study is based on TEDA International Cardiovascular Hospital and the Second Hospital of Tianjin Medical University. From January 2018 to January 2020, 229 patients with the initially diagnosed ST↑ IWMI were enrolled. According to the exclusion criteria, 80 patients were excluded, and 149 patients were included in the final study.

Participants who met the following criteria were included in the study: (1) The diagnosis of ST↑ IWMI was based on the requirements formulated by the joint European Society of Cardiology/Alliance of American College of Cardiology/American Heart Association/World Heart Federation (ESC/ACCF/AHA/WHF) working group in 2017:[13] 1. Symptoms of myocardial ischemia ≥20 minutes; 2. New ECG changes: ST↑ ≥0.1mV in two or more leads II, III, and aVF; 3. Elevated markers of myocardial injury: troponin T or I (cTnT or cTnI) or phosphocreatine kinase isoenzyme (CK-MB) elevations more significant than 99% of the upper limit of the standard reference value. (2) The time from onset of MI to ECG examination was ≤6 hours. (3) Coronary angiography was performed within 12 hours after the onset of MI. (4) The first episode of IWMI. (5) Coronary angiography showed that the coronary artery disease (CAD) was the single vessel, namely the RCA or LCx.

One of the following conditions is excluded: (1) Double or multi-vessel disease; (2) Previous history of acute MI with coronary artery stent implantation or coronary artery bypass grafting; (3) ECG showed left ventricular hypertrophy or left or right bundle branch block; (4) Diseases affecting ST-segment deviation such as pacing heart rate, pericarditis or early repolarization syndrome; (5) Severe electrolyte disturbance; (6) Other heart diseases: cardiac structural changes caused by dilated cardiomyopathy, hypertrophic cardiomyopathy and heart valve lesions; (7) Incomplete ECG, clinical or coronary angiography data.

ECG Variables

ECG was performed after hospitalization or in the emergency department. The speed of paper walking was 25mm/s, and the standard voltage was 1mm=0.1mV. The T-P segment was chosen as the equipotential line, the ST↑ was represented by J point, and the ST↑ ≥0.1mV was meaningful.

The ST-segment deviation data were double-checked by two attending cardiologists who were unaware of the patient's clinical data and coronary angiography results to ensure the accuracy of the data. The superior physician was asked to coordinate the results when the two data were inconsistent.

Unaware of the patient's electrocardiographic findings, two attending cardiologists reviewed coronary angiography. The infarct-related artery was defined as follows: (1) MI-related coronary artery complete or subtotal occlusion (≥70% stenosis); (2) Acute thrombosis or ulcerative plaque rupture; (3) Patients with apparent stenosis in both RCA and LCx were excluded.

Statistical and Analysis

Continuous variables were demonstrated in mean ± standard deviation (SDs) or median (25th to 75th percentile) form and compared using *t*-tests or Wilcoxon rank-sum tests when appropriate. Categorical variables are displayed as frequencies and percentages using Fisher's exact or chi-square test, which is suitable for determining the significance of categorical variables between the two groups.

For analysis, continuous variables were transformed into simple categorical variables classified according to the degree of ST↓. Univariate logistic regression analysis was used to analyze the relationship between the degree of ST↓ in each N-IWL ECG and the location of RCA.

At the same time, the factors with P≤0.05 were selected for further multivariate analysis. If two or more factors were correlated in the same lead, we included them in the stepwise logistic regression model according to the order of ST↓, ST↓ ≥0.1mV, and ST↓ ≥0.05mV. The overall performance of the model was assessed using the C-index.

A simplified N-IWL RCA score was developed by assigning a weight corresponding to the beta coefficient, rounded to the nearest integer, associated with the degree of ST-segment deviation for each cue in the multivariate model. Patients were divided into three groups (low, medium, and high) according to the likelihood that the final culprit vessel was the RCA. We evaluated the accuracy of RCA localization in the three groups of patients. The N-IWL RCA scoring system was compared with the existing diagnostic criteria for locating the RCA. The sensitivity, specificity, positive predictive value, negative predictive value, and C-index were calculated for each ECG algorithm to find the RCA. Two-tailed P values of within 0.05 were thought statistically necessary. All statistical analyses were performed employing SPSS 27.0 and GraphPad Prism 8.0.

Results

The Demographic and ECG Baseline Characteristics

149 patients were enrolled, including 83 in the RCA group and 66 in the LCx group. The mean age of the patients was 61.72±12.84 years, and 71.8% were male. Hypertension and smoking were the most common risk factors (60.4%), followed by diabetes (25.5%) and hyperlipidemia (4.0%). 80.5% of the patients still had symptoms of myocardial ischemia at diagnosis. Patients in the RCA group had lower systolic blood pressure (76.51 vs 84.56, P<0.001), creatine kinase (334.05 vs 680.82, P=0.027) and creatine kinase isoenzyme (32.86 vs 75.45, P=0.008). There were no significant differences in other demographic data between the two groups. See Table 1.

The differences in ST-segment deviation in each lead between the RCA and LCx groups were compared. In the RCA group, the ST↓ in lead I was higher, the ST↓ in lead aVR and V1 was lower, and the ST↑ in lead V6 was lower. The two groups had no significant difference in the degree of ST segment deviation in leads II, III, aVF, aVL, and V2-V5. See Table 2.

Univariate and Multivariate Logistic Regression Analysis

We tested for univariate associations between the degree of ST↓ in N-IWL and the location of the RCA in the enrolled population. ST↓ in leads I, aVL, and V6 was associated with a higher likelihood of RCA involvement. In contrast, ST↓ in leads avR, V1, V3, and V4 was associated with a higher probability of unaffected RCA.

According to our inclusion rules, the multivariate logistic regression analysis included ST↓ in lead I, aVR, aVL, V1, V3, V6, and ST↓≥0.1mV in lead V4. In multivariate logistic regression analysis, ST↓ in leads I and V6 was associated with a greater likelihood of involvement of the RCA, and ST↓ in leads aVR and V1 and ST↓≥0.1mV in lead V4 were associated with a greater chance of no participation of the RCA. The model's C-index (the ability to assess RCA involvement) was excellent at 0.83. See Table 3.

The Score of Locating the RCA

The specific criteria for the simplified RCA score in N-IWL designed after multivariate modeling in this study were as follows: ST↓ was scored for 2 points in lead I, 1 point in lead aVR, −2 points in lead V1, −2 points in lead V4 with ST↓ ≥0.1mV, and 1 point in lead V6.

The enrolled population had scores ranging from −5 to 3. According to the score, patients were divided into three groups: low (score of −2 or less), medium (score of −2, −1, and 0), and high (score greater than 0). See Table 4 and Figure 1.

Table 1 Baseline Characteristics of the Two Groups

	ALL (N=149)	RCA (N=83)	LCX (N=66)	P value
Clinical characteristics				
Age(years)	61.72(48.88–74.56)	63.04(50.45–75.63)	60.06(47.02–73.10)	0.161
Male sex, n(%)	107(71.8%)	58(69.9%)	49(74.2%)	0.587
Hypertension, n(%)	90(60.4%)	51(61.4%)	39(59.1%)	0.866
Diabetes mellitus, n(%)	38(25.5%)	20(24.1%)	18(27.3%)	0.707
Dyslipidemia, n(%)	6(4.0%)	3(3.6%)	3(4.5%)	0.999
Current smoker, n(%)	90(60.4%)	46(55.4%)	44(66.7%)	0.181
Family history, n(%)	2(1.3%)	1(1.2%)	1(1.5%)	0.999
Have chest pain, n(%)	120(80.5%)	69(83.1%)	51(77.3%)	0.409
Killip classification	1.09(0.73–1.45)	1.05(0.83–1.27)	1.15(0.68–1.62)	0.078
Ejection fraction(%)	56.33(47.45–65.21)	56.68(48.19–64.87)	56.06(46.64–65.48)	0.712
SBP(mmHg)	130.91(106.22–155.60)	127.64(103.07–152.21)	135.02(110.62–159.42)	0.071
DBP(mmHg)	80.07(65.54–94.60)	76.51(63.58–89.44)	84.56(69.28–99.84)	<0.001
Heart rate(bpm)	72.93(56.70–89.16)	72.08(55.73–88.43)	73.98(57.85–90.28)	0.479
Laboratory parameters				
CK(U/L)	458.43(234.09–682.77)	334.05(210.34–457.76)	680.82(324.06–1037.58)	0.027
CK-MB(ng/mL)	48.14(23.55–72.73)	32.86(20.83–44.89)	75.45(38.58–112.32)	0.008
Total cholesterol(mmol/L)	4.57(3.65–5.49)	4.51(3.69–5.33)	4.68(3.61–5.75)	0.381
Triglycerides(mmol/L)	1.89(0.86–2.92)	1.88(0.46–3.30)	1.89(0.84–2.94)	0.989
HDL(mmol/L)	1.04(0.81–1.27)	1.03(0.81–1.25)	1.07(0.83–1.31)	0.413
LDL(mg/dL)	2.96(2.11–3.81)	2.92(2.18–3.66)	3.03(2.00–4.06)	0.561
Lactate dehydrogenase(U/L)	271,75(85.12–458.38)	275.85(68.37–483.33)	264.42(119.73–409.11)	0.781
FBG(mmol/L)	8.82(4.93–12.71)	8.76(5.17–12.35)	8.94(4.49–13.39)	0.838

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; CK, creatine kinase; CK-MB, creatine kinase isoenzyme; HDL, high-density lipoprotein; LDL, low-density lipoprotein; FBG, fasting blood glucose.

Table 2 ST-Segment Changes of Different Leads in Patients with Inferior Wall Myocardial Infarction

Lead	ALL (N=149)	RCA Occlusions (N=83)	LCX Occlusions (N=66)	P value
I	−0.75(−1.51–0.01)	−0.84(−1.66–0.02)	−0.58(−1.22–0.06)	0.013
II	1.78(0.58–2.98)	1.72(0.65–2.79)	1.86(0.52–3.20)	0.488
III	2.26(0.78–3.74)	2.30(0.85–3.75)	2.21(0.69–3.73)	0.715
aVR	−0.33(−1.21–0.55)	−0.14(−1.10–0.82)	−0.57(−1.28–0.14)	0.003
aVL	−1.30(−2.69–0.09)	−1.47(−2.84–0.10)	−1.28(−2.49–0.09)	0.367
aVF	1.96(0.69–3.23)	1.94(0.78–3.10)	1.98(0.57–3.39)	0.845
V1	−0.02(−0.97–0.93)	0.16(−0.67–0.99)	−0.24(−1.30–0.82)	0.011
V2	−0.87(−2.83–1.09)	−0.78(−2.70–1.14)	−0.98(−2.99–1.03)	0.543
V3	−0.72(−2.49–1.05)	−0.57(−2.10–0.96)	−0.92(−2.94–1.10)	0.224
V4	−0.32(−1.98–1.34)	−0.28(−1.62–1.06)	−0.36(−2.37–1.65)	0.771
V5	−0.06(−1.40–1.28)	−0.14(−1.38–1.10)	0.04(−1.43–1.51)	0.416
V6	0.24(−0.92–1.40)	0.03(−1.13–1.19)	0.51(−0.60–1.62)	0.011

The incidence of culprit vessels was 77.11% in the high-score group, 21.69% in the middle-score group, and 1.20% in the low-score group. Significant differences existed in the incidence of the culprit's vessels between the high, medium, and low-score groups (P<0.001). See Figure 1.

When the RCA score was used to diagnose the LCx, the incidence of culprit vessels in the high score group was 27.27%, and the incidence of culprit vessels in the non-high score group was 72.73%; the difference between the two groups was statistically significant (P<0.001). See Figure 1.

Table 3 Univariable and Multivariable Predictors of Right Coronary Artery (RCA) Involvement

Lead		Univariable Cox Regression		Multivariable Cox Regression	
		HR(95% CI)	P value	HR(95% CI)	P value
I	STD I	4.745(2.080–10.827)	<0.001	4.322(1.626–8.488)	0.003
	STD I≥0.5mm	1.591(0.797–3.177)	0.188		
	STD I≥1.0mm	1.881(0.971–3.643)	0.061		
aVR	STD aVR	0.282(0.142–0.557)	<0.001	0.426(0.186–0.974)	0.043
	STD aVR≥0.5mm	0.282(0.142–0.557)	<0.001		
	STD aVR≥1.0mm	0.377(0.172–0.824)	0.015		
aVL	STD aVF	6.541(1.778–24.060)	0.005	3.999(0.844–18.958)	0.081
	STD aVL≥0.5mm	1.232(0.557–2.722)	0.607		
	STD aVL≥1.0mm	1.241(0.622–2.474)	0.541		
V1	STD V1	0.187(0.080–0.438)	<0.001	0.330(0.120–0.909)	0.032
	STD V1≥0.5mm	0.187(0.080–0.438)	<0.001		
	STD V1≥1.0mm	0.307(0.123–0.767)	0.011		
V2	STD V2	0.916(0.477–1.759)	0.792		
	STD V2≥0.5mm	0.873(0.455–1.674)	0.682		
	STD V2≥1.0mm	0.638(0.333–1.223)	0.176		
V3	STD V3	0.899(0.866–0.933)	0.011	0.847(0.312–2.297)	0.744
	STD V3≥0.5mm	0.467(0.242–0.903)	0.024		
	STD V3≥1.0mm	0.537(0.277–1.040)	0.065		
V4	STD V4	0.515(0.264–1.006)	0.052		
	STD V4≥0.5mm	0.515(0.264–1.006)	0.052		
	STD V4≥1.0mm	0.400(0.195–0.819)	0.012	0.286(0.095–0.866)	0.027
V5	STD V5	1.018(0.513–2.019)	0.959		
	STD V5≥0.5mm	1.018(0.513–2.019)	0.959		
	STD V5≥1.0mm	0.976(0.465–2.049)	0.949		
V6	STD V6	3.514(1.571–7.860)	0.002	2.693(1.005–7.215)	0.049
	STD V6≥0.5mm	1.880(0.788–4.487)	0.155		
	STD V6≥1.0mm	2.062(0.688–6.182)	0.196		

Note: C-index = 0.83.
Abbreviation: STD, ST segment depression.

Table 4 Non-Inferior Lead Right Coronary Artery Score

Lead	Value	Points
I	Any ST↓	2
aVR	Any ST↓	−1
V1	Any ST↓	−2
V4	ST↓≥1mm	−2
V6	Any ST↓	1

Abbreviation: ST↓, ST segment depression.

Comparison of the Accuracy of RCA Localization by Different Diagnostic Criteria

Table 5 summarizes the sensitivity, specificity, positive predictive value, and negative predictive value of the N-IWL RCA score for the RCA diagnosis, compared with six previously reported single criteria and one score.
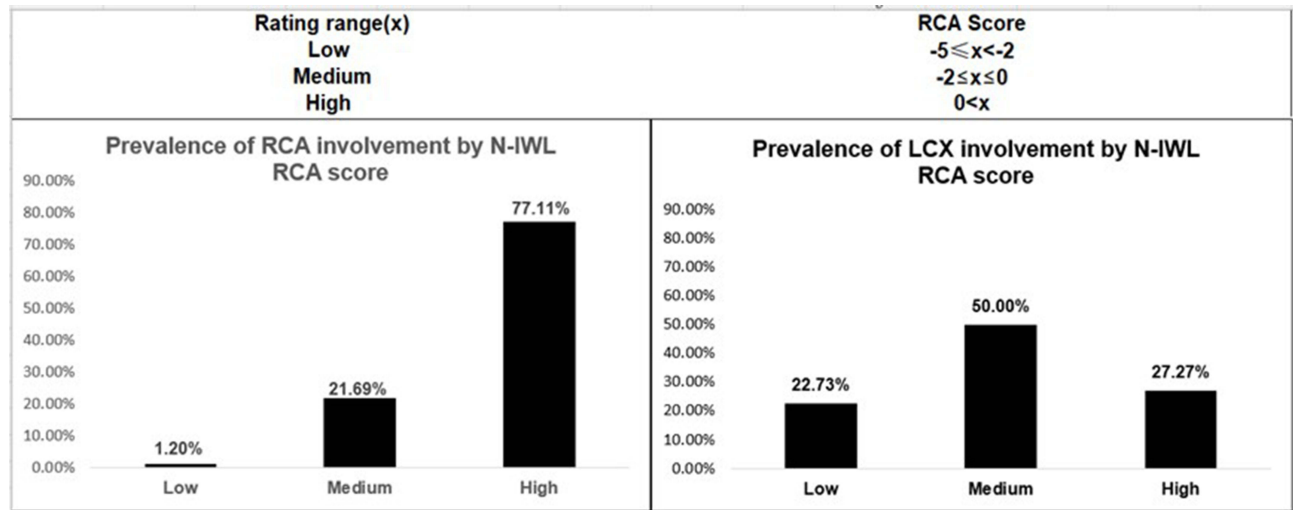
Rating range(x)	RCA Score
Low	$-5 \leqslant x < -2$
Medium	$-2 \leq x \leq 0$
High	$0 < x$

Prevalence of RCA involvement by N-IWL RCA score

Prevalence of LCX involvement by N-IWL RCA score

Figure 1 Stratification of non-inferior lead right coronary artery score and diagnostic efficacy of each stratification.

Based on these data, the diagnostic power of our N-IWL RCA score was not inferior to that of the RCA score proposed by Almansori et al (0.83 vs 0.82). It was substantially higher than the other six previously reported single criteria. See Table 5.

Discussion

Although coronary angiography is the gold standard for identifying the culprit vessel in MI, ECG can be a valuable tool for locating the culprit vessel in the first place. Distinguishing LCx from RCA involvement may also be helpful for risk stratification in patients with IWMI.[14,15] Previous studies have shown that patients with occlusion of the RCA have worse clinical prognosis than those with occlusion of the LCx in patients with MI.[16] Subsequent studies have also confirmed increased mortality and worse clinical prognosis in patients with RCA occlusion in patients with anterior MI.[17] Similarly, IWMI patients with RCA occlusion have a worse clinical prognosis than those with LCx occlusion due to right ventricular injury, hemodynamic changes, and an increased risk of conduction block.[18] At present, there is still a lack of research on the relationship between infarction vessels and long-term prognosis in patients with IWMI, and the current research is more limited to a single center, small sample study, and the level of evidence is relatively low.

IWMI is the most common type of MI in clinical practice. The culprit's vessels are mostly RCA and LCx, and the occlusion of RCA is more common. According to the coronary artery anatomy, the coronary artery is divided into left and right systems.[19] The left anterior descending artery and LCx supply the anterior septal wall, anterior wall, and

Table 5 Sensitivity, Specificity, Positive Predictive Value, and Negative Predictive Value of Different Diagnostic Criteria for Right Coronary Artery Involvement

Criteria	Sensitivity(%)	Specificity(%)	PPV(%)	NPV(%)	C-Index
N-IWL RCA score>0	77.1	72.7	78.1	71.6	0.83
Almansori et. RCA score>2	79.5	57.6	70.2	69.1	0.82
STE III>II	71.1	40.9	60.2	52.9	0.56
STD aVL>I	53.1	81.8	78.6	58.1	0.68
STD I	87.9	39.4	64.6	72.2	0.64
(sum STD V1-V3)/(sum STE inf)≤I	95.2	9.1	56.8	60.0	0.51
Arth sum ST segment: aVF+V2>0	77.1	39.4	61.5	57.8	0.58
STD V3/STE III≤1.2	92.8	15.2	57.9	62.5	0.54

Abbreviations: PPV, positive predictive value; NPV, negative predictive value; Arth Arithmetic, inf Lead II, III, and aVF; STE, ST-segment elevation; STD, ST-segment depression; N-IWL, Non-inferior wall lead.

anterior lateral wall myocardium. The RCA mainly provides the inferior wall, the right ventricle's posterior wall, and the left ventricle's posterior wall. When the culprit vessel is occluded, ischemic necrosis occurs in the corresponding blood supply area, and the electrocardiogram shows ST-segment deviation. According to the degree of ECG deviation, the location of the culprit's vessels and the degree of the lesion can be quickly judged, which can guide the risk stratification of patients, the application of reperfusion treatment strategies, and clinical prognosis management.[20,21] However, so far, most studies have only compared and analyzed the results of ECG and coronary angiography to obtain the results of sensitivity, specificity, etc. Still, no comprehensive, complete, and highly reliable diagnostic criteria exist.[22]

According to the degree of ST-segment deviation in different leads, different ECG diagnostic criteria have been proposed to locate the culprit vessel. These criteria are all based on the fact that in patients with IWMI, the injury vector is more downward than backward and more right than left in RCA occlusion.[23,24]

Recently, Mario et al through the application of precordial bipolar leads (PBL) of the new method, the analysis of the culprit vessels electrocardiogram before and after revascularization, discusses in detail the currents of injury in the right-to-left axis. In brief: (1) There is a cornerstone point for ischemic change in the QRS loop during LAD occlusion, and the entire loop is changed. In addition, altered myocardial activation appears to contribute to ST-segment elevation in hyperacute ischemia.[25] (2) When RCA was occluded, in PBL V2-V1, the strong systolic injury current moving from left to right coexisted with the current of guide links II, III, and aVF.[26] (3) In the case of LCx occlusion, there was a strong systolic injury current in different and sometimes opposite directions along the left and right axes, and the duration and amplitude of the QRS wave were ischemic altered.[27]

After IWMI, ST↑ in leads II, III, and aVF is the key to diagnosing myocardial injury affecting the inferior and posterior walls of the heart. To determine the extent of myocardial damage from the culprit vessel, it is necessary to assess ST-segment changes in N-IWL, including leads V1-V3 (ST↓ reflects inferior posterior wall), leads I, aVL, and V5-V6 (ST↓ mirrors lateral wall), and V3R-V5R (ST↓ reflects proper ventricular involvement).[28] Although ST-segment deviation in N-IWL can be explicitly described, it may disappear in the early stages of developing IWMI. At the same time, with cardiac conduction bypass involvement, V3R-V5R leads may not change.[29] Another disadvantage of diagnosis based on these clues is that documented evidence of these clues is often lacking in the emergency room. Therefore, we aimed to assess the combined outcome of ST-segment deviation in N-IWL in IWMI and to develop a new, simple, and diagnostic score for the localization of the RCA. This scoring is discussed in the remainder of this article.

The degree of ST↓ in leads aVL (usually more depression in lead aVL than in lead I) and I has been emphasized by many investigators as a marker for locating RCA occlusion. Standard lead III points to the lower right segment, lead II topics to the lower left part, and tends to point to the lateral and inferior wall of the left ventricle. Thus, lead III is more susceptible to the RCA, while lead II is more susceptible to the LCx.[30] Based on the vector mirror-image mechanism, ST↓ in lead aVR is more likely to be affected by the RCA, and ST↓ in lead I is more likely to be affected by the LCx. In a study of 83 patients with IWMI, Herz et al found that a greater degree of ST↑ in lead III than in lead II and a greater degree of ST↓ in lead aVR than in lead I were sensitive and specific markers for localizing the RCA.[31] If both criteria were negative, the accuracy of LCx location was 100%.[8] These observations are supported by Hasdai et al, who found a significant increase in the incidence of RCA occlusion with ST↓ ≥1mm in the lead I and aVL without ST↓ in the lead I, a sensitive marker of occlusion of the LCx.[32] Similar to our findings, ST↓ in lead I was independently associated with RCA location and conferred a score of 2 points on a scoring system. However, ST↓ in the lead aVL did not have independent predictive power after multivariate analysis and was therefore not included in the score.

The current injury caused by occlusion of the RCA would be more or less perpendicular to the aVR lead axis. In contrast, the recent damage caused by occlusion of the LCx would have an average vector at an obtuse angle to the aVR lead axis. Therefore, occlusion of the LCx is more likely to trigger ST↓ in lead aVR. In patients undergoing PCI with normal conduction, ST↑≥1mm in lead aVR is associated with 30-day mortality.[33] Sun et al showed that ST↓≥1mm in lead aVR is a sensitive indicator of occlusion of the LCx.[34] The frontal study by Kanei et al also found that ST↓ in lead aVR was more common in occlusion of the LCx but was associated with greater infarct size if it occurred in occlusion of the RCA.[35] Similarly, ST↓ in the lead aVR was independently related to non-RCA location and scored −1 on a scoring system.

When the RCA is occluded, the ST-segment in lead V1 may be elevated due to the influence of the right ventricular injury current. Similarly, Patients with (proximal) RCA occlusions have less ST↓ and even ST↑ in V1. In contrast, occlusion of the LCx leads to ST↑ in the posterior wall of the left ventricle, which may result in characteristic ST↓ in leads V1 and V2. Wong et al analyzed ECG in 7967 patients with acute inferior MI and found that baseline ST↑ in lead V1 was associated with increased mortality, suggesting that ST↑ in lead V1 can be used as a reliable tool for the identification of high-risk patients with inferior MI.[36] Analysis of electrocardiograms by Nair et al in 30 patients with ST↑ MI showed that ST↓ in leads V1 and V2 helped localize the LCx. However, they did not find a diagnostic role of ST↓ in leads V2 and V3 for the localization of the culprit vessel.[37] Similar to our findings, ST↓ in lead V1 was independently associated with non-RCA location and was assigned a score of −2 on the scoring system. Leads V2 and V3 did not have independent predictive power, so they were not included in the score.

In the precardiac plane, occlusion of the RCA may cause ST↓ in leads V4-V6, and occlusion of the LCx may cause ST↑ in leads V4-V6. An ECG analysis by Wong et al of 95 patients with IWMI showed that ST↑ in lead V5 or V6 predicted occlusion of the LCx with a sensitivity of 56% and a specificity of 92%.[38] In our study, ST↓ in lead V6 was independently associated with RCA localization and scored 1 point on the scoring system. Lead V5 did not have independent predictive power and was not included in the score. Interestingly, we found that ST↓≥1mm in lead V4 was independently associated with non-RCA localization and scored −2 points on the scoring system.

Mohammed et al proposed the ASSENT 4 PCI RCA scoring method in 2010. They analyzed the clinical data and ECG of more than 1700 patients with IWMI selected from medical centers in 29 different countries. To propose a scoring method for locating the RCA based on the ST-segment deviation in each lead of ECG.[7] They used ST↓ in leads I, V1, V3, and V6, ST↓≥1mm in lead aVL, and ST↑≥1mm in lead aVF as the six criteria for the score. We compared Mohammed et al' s score with ours; the sensitivity of the two scores was similar (77.1% vs 79.5%, P=0.326), and the specificity of the N-IWL score was higher in the RCA (72.7% vs.57.6%, P=0.028). The diagnostic efficacy of the two scores was similar (0.83 vs 0.82, P=0.842).

A recent meta-analysis summarized the diagnostic efficacy of the diagnostic criteria for the culprit vessel in patients with IWMI in recent years and found that the diagnostic efficacy of the ST↑ in lead III greater than lead II was the highest, which can be used as the cornerstone of diagnosis. Surprisingly, they found that known diagnostic algorithms performed no better than single diagnostic criteria. The risk of bias due to the case-control design may be the main reason. In addition, the algorithm involves many steps, determining that it is not popular in clinical practice.[22]

Therefore, we applied multivariate logistic regression analysis to develop an RCA score for N-IWL. We found that the diagnostic performance of our scoring system was excellent, with a C-index of 0.83. The probability of RCA involvement in patients with a score greater than 0 was 77.11%. We also compared the score with the known diagnostic criteria for locating the RCA. We found that the algorithm we developed had better sensitivity and specificity than other diagnostic criteria. Due to the simplicity of the scoring system, it can be easily applied in clinical practice. Similarly, patients with a score of 0 or less had a 72.73% probability of involvement with the LCx. However, whether the score applies to diagnosing LCx disease needs to be verified by more studies.

Our study also has certain limitations. First, our study is a retrospective, case-control study, which inevitably has the risk of patient selection bias. Second, we lacked information about the right precordial leads (particularly the V4R) and the morphology of the Q and QRS waves, which are increasingly being shown to be independent predictors of locating the culprit vessel. Third, our study did not include the correlation between ST segment bias in the inferior leads and the location of the RCA, which may have reduced the diagnostic power of the final score. Fourth, our score could not distinguish differences between proximal and distal lesions in the RCA. The broad inclusion and exclusion criteria did not make the score applicable to patients with inferior MI with poor vascular status. Finally, we enrolled patients with single-vessel disease, and the diagnostic efficacy of the score for patients with multi-vessel disease remains to be explored. The results of this study need to be verified by multi-center, prospective, and extensive sample studies.

Conclusion

ECG helps identify the RCA in patients with inferior wall myocardial infarction before percutaneous coronary intervention. The non-inferior wall leads RCA score may help identify the culprit vessel as the RCA in patients with inferior wall myocardial infarction.

Ethics Statement

All procedures performed in this study were in accordance with the ethical standards of the TEDA International Cardiovascular Hospital and the Second Hospital of Tianjin Medical University. The study complied with the Declaration of Helsinki and was permitted by the Second Affiliated Hospital of Tianjin Medical University (IRB number 2023-05-B023). Patients/participants all signed informed consent before enrollment.

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Disclosure

The authors report no conflicts of interest in this work.

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