

A Novel Electrocardiographic Score Predicts the Severity of Coronary Artery Disease and Clinical Outcomes in Patients with Non-ST Segment Elevation Myocardial Infarction

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Background: There are many electrocardiographic (ECG) changes in non-ST segment elevation myocardial infarction (NSTEMI). However, the diagnostic power is limited in determining the severity of coronary artery disease (CAD) and clinical outcomes.

Objective: This study investigated the role of a risk-based ECG score in predicting the severity of CAD and clinical outcomes in NSTEMI patients.

Methods: One hundred and fifty-two patients were enrolled in the study. Severe CAD was defined as; intermediate (> 22) or high SYNTAX score (> 32), three-vessel disease, and left main coronary artery lesions. A risk-based ECG score was calculated, and the patients were categorized. All patients were followed up, and mortality and repeat revascularizations were evaluated.

Results: The severe CAD group had a significantly higher risk-based ECG score than the non-severe CAD group ($p = 0.013$). The patients with a high risk-based ECG score had more severe CAD ($p = 0.013$), higher SYNTAX score ($p < 0.001$), more three-vessel disease ($p = 0.003$), coronary artery calcification ($p = 0.02$), and one-year mortality ($p = 0.006$) than those with medium or low ECG scores. Multivariate logistic regression analysis showed that a 1-point increase in the risk-based ECG score was associated with a 1.573-fold [95% confidence interval (CI): 1.111-2.227, $p = 0.011$] increase probability of severe CAD. Kaplan-Meier analysis demonstrated that the high-risk group had a significantly higher one-year mortality rate than the low-risk and moderate-risk groups (hazard ratio: 2.383, 95% CI: 1.395-4.072, $p = 0.001$).

Conclusion: This study demonstrated that higher ECG scores were associated with a higher risk of severe CAD and worse clinical outcomes in NSTEMI patients.

Key Words: Coronary artery disease • Electrocardiogram • SYNTAX score

INTRODUCTION

In acute coronary syndrome (ACS), non-ST segment elevation myocardial infarction (NSTEMI) accounts for a significant proportion of patients. The SYnergy between percutaneous coronary intervention with TAXus and cardiac surgery (SYNTAX) score quantifies the severity and complexity of coronary artery disease (CAD) and helps clinicians decide the proper revascularization type. It has been observed that half of the patients who present with

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NSTEMI have multi-vessel disease, and that patients with high Synergy Between PCI With Taxus and Cardiac Surgery (SYNTAX) scores have a worse prognosis than those with low SYNTAX scores.¹ However, the SYNTAX score is not available until coronary angiography is performed, which is not helpful in the emergency department. Therefore, an easily accessible diagnostic tool is needed to help predict the severity of NSTEMI patients.

A 12-lead electrocardiogram (ECG) plays a pivotal role in managing patients with ACS; however, it has a limited diagnostic and prognostic value in NSTEMI patients. NSTEMI patients may present with different ECG changes, such as transient ST-segment elevation, persistent or transient ST-segment depression, T-wave inversion, flat T waves, and pseudonormalization T waves. However, more than 30% of patients do not have typical ischemic ECG findings.²⁻⁴

An ECG score that can help predict the severity of CAD in NSTEMI patients has not been defined in the current literature. The combination of various ECG findings can be more helpful in determining high-risk subjects before coronary angiography. A recently published risk-based ECG score composed of combinations of routinely measured ECG parameters was shown to be able to predict sudden or arrhythmic death in patients with CAD.⁵ Therefore, in the present study, we aimed to investigate the possible relationship between the risk-based ECG score and the severity of CAD in NSTEMI patients. In addition, we followed up the patients for approximately 12 months, and mortality and repeat revascularization rates were assessed.

METHODS

Study population

In this single-center retrospective study, 173 NSTEMI patients without a previous CAD history at Gebze Fatih State Hospital were screened between August 2020 and December 2020. Patients who had moderate to severe valvular heart disease ($n = 4$ cases), with left ventricular systolic dysfunction ($n = 2$ cases), cardiomyopathy ($n = 2$ cases), those with signs of ST-segment elevation myocardial infarction on ECG ($n = 3$ cases), second or third-degree atrioventricular block ($n = 1$ case), complete left bundle branch block ($n = 4$ cases) and uninterpretable

ECG data due to either the presence of significant artifacts and/or low image quality ($n = 5$ cases) were excluded from the study. Finally, 152 patients constituted the study population. Baseline demographic data and laboratory findings were obtained from the hospital's electronic database system. Complete blood counts and biochemical parameters, including blood glucose, creatinine, albumin, and cardiac troponin I level, and echocardiographic findings were obtained upon admission to the emergency department. All subjects were followed up for 12 months, and mortality and repeat revascularization were defined as an outcome; these data were obtained from national health insurance records. According to the Declaration of Helsinki, the present study was reviewed and approved by the Local Ethics Committee.

Angiographic analysis

Two experienced invasive cardiologists who were blinded to the patients' information other than angiographic images, evaluated all coronary angiograms. In cases of disagreement, a third cardiologist's opinion was obtained. A left main coronary artery (LMCA) lesion was defined as the presence of stenosis $\geq 50\%$, and three-vessel disease was defined as stenosis $\geq 70\%$ in all three main coronary arteries. The SYNTAX score of each patient was calculated for each coronary artery lesion (SYNTAX calculator, version 2.28). The presence of any of the following angiographic features was defined as severe CAD; intermediate (> 22) or high (> 32) SYNTAX score, three-vessel disease, and LMCA lesions.

Electrocardiographic analysis and risk-based electrocardiographic score

In the present study, the ECG of each patient (Nihon Kohden Corporation, Cardiofax M Model ECG-1250, Tokyo, Japan) was obtained on admission with a 25 mm/sec paper speed and 10 mm/mV voltage calibration. All ECG recordings were analyzed with digital image processing software (imagej.nih.gov/ij/). Two experienced cardiologists performed all measurements. ST-segment depression (≥ 1 mm in at least two contiguous leads), ST-segment elevation in aVR (≥ 0.5 mm), T wave inversion (≥ 1 mm negative deflection from the isoelectric line in at least two contiguous leads other than aVR) were noted. Q waves were defined as those > 40 ms wide, > 2

mm deep, and > 25% of the depth of QRS. Left ventricular hypertrophy (LVH) was defined according to Sokolow-Lyon or sex-specific Cornell voltage criteria. The QRS duration was defined as the interval from the beginning of the QRS complex to the J point, and the most prolonged duration was recorded. Corrected JT interval (JTc) was derived by subtracting the QRS duration from the QTc. The risk-based ECG score was calculated by summing the points of the following parameters: the presence of contiguous Q wave (1 point); QRS duration ≤ 80 ms (0 points), 81-110 ms (1 point), > 110 ms (2 points); LVH (Sokolow-Lyon or Cornell voltage criteria) (1 point); and prolonged JTc (≥ 360 ms) (1 point). The total score ranged from 0-5. The patients were divided into low (0-1), medium (2), and high-risk (3-5) groups according to their total ECG score.

Statistical analysis

All data were analyzed using SPSS version 17.0 (SPSS Inc., Chicago, Illinois). Data normality was analyzed using the Kolmogorov-Smirnov test. Numerical variables with a normal distribution are presented as mean $\pm$ standard deviation, whereas those without a normal distribution are presented as median [interquartile range (IQR)]. Frequency distribution was calculated for categorical variables [numbers and percentages (%)]. Continuous variables of three groups were compared using either the Kruskal-Wallis test or ANOVA. Continuous variables of two groups were compared using the Mann-Whitney U test or Student's t-test. Categorical data were compared using either the chi-square test or Fisher's exact test. Statistical significance was defined as a p-value < 0.05 . Multivariable logistic regression analysis was performed to identify the independent predictors of severe CAD using variables that showed an association in univariable analysis. Receiver operating characteristic (ROC) curve analysis was used to calculate the ideal value to predict severe CAD with the best specificity and sensitivity. Survival analysis was performed using the Kaplan-Meier method, and differences in survival parameters were evaluated using the log-rank test.

RESULTS

A total of 152 patients [n = 58 females (38.2%), mean

age = 65 ± 12 years] were stratified into two groups according to CAD severity [non-severe CAD (n = 99 cases) and severe CAD (n = 53 cases)]. Baseline characteristics, laboratory, ECG and angiographic data, and one-year mortality and repeat revascularization rates of all patients are demonstrated in Table 1. There was a higher percentage of female patients and the left ventricular ejection fraction (LVEF) was lower in the severe CAD group than in the non-severe CAD group. There were no statistically significant differences in the other baseline characteristics, laboratory findings, and outcomes (one-year mortality and repeat revascularization) between the two groups.

With regards to ECG findings, patients in the severe CAD group had more frequent ST-segment depression, ST elevation in aVR, and LVH than those without severe CAD. There was a numerical increase in the patients with severe CAD regarding the presence of Q waves, QRS duration, and JTc duration, none of which reached a statistical significance. According to the risk-based ECG score, 31.6% (n = 46 cases) of the study population had high-risk ECG scores, and the percentage was significantly higher in the severe CAD group than in the non-severe CAD group (41.5% vs. 26.3%; $p = 0.013$, respectively). The study population was then divided into three groups according to risk-based ECG scores [low (0-1 point) (n = 47 cases), moderate (2 points) (n = 59 cases), and high (≥ 3 points) (n = 46 cases) risk]. The baseline characteristics, laboratory, ECG and angiographic data of the patients are listed in Table 2. We observed that the patients with high risk-based ECG scores were older, and their cardiac troponin I levels were significantly higher. On the other hand, their LVEF was significantly lower. According to the coronary angiography data, higher rates of severe CAD (19.1% vs. 37.3% vs. 47.8%, $p = 0.013$, respectively), SYNTAX score [13 (IQR = 9-17) vs. 15 (IQR = 9-20) vs. 21 (IQR = 15-26), $p < 0.001$, respectively], three-vessel disease (2.1% vs. 11.9% vs. 26.1%, $p = 0.003$, respectively), and coronary artery calcification (10.6% vs. 22.0% vs. 34.8%, $p = 0.02$, respectively) were seen in the high risk-based ECG score group. Mortality rates were significantly higher in the high-risk group at one year of follow-up (2.1% vs. 1.7% vs. 15.2%, $p = 0.006$, respectively).

All variables associated with severe CAD, including female gender, LVEF, ST-segment depression, ST eleva-

Table 1. Baseline properties, electrocardiogram, angiographic findings, one-year mortality, and repeat revascularization rates of all cases

	All patients (n = 152)	Non-severe CAD (n = 99)	Severe CAD (n = 53)	p value
Age	65 ± 12	64 ± 11	66 ± 13	0.685
Gender (female), n (%)	58 (38.2)	32 (32.3)	26 (49.1)	0.043
Diabetes mellitus, n (%)	40 (26.3)	23 (23.2)	17 (32.1)	0.238
Hypertension, n (%)	97 (63.8)	63 (63.6)	34 (64.2)	0.950
Smoking, n (%)	65 (42.8)	47 (47.5)	18 (34.0)	0.109
Fasting blood glucose, mg/dl	106 (95-127)	106 (95-127)	118 (93-127)	0.754
Creatinine, mg/dl	0.9 (0.7-1)	0.9 (0.7-1)	1.0 (0.7-1.1)	0.988
Total cholesterol, mg/dl	159 (139-194)	161 (140-194)	164 (141-194)	0.515
LDL-C, mg/dl	102 (79-121)	103 (78-120)	104 (88-127)	0.542
HDL-C, mg/dl	35 (29-40)	35 (29-40)	33 (28-40)	0.481
Triglyceride, mg/dl	108 (79-158)	104 (77-151)	112 (95-165)	0.179
Hemoglobin, g/dl	13.7 ± 1.9	13.8 ± 2.0	13.4 ± 1.5	0.257
Troponin I, ng/ml	1.1 (0.1-6.1)	1.0 (0.1-6.5)	1.5 (0.3-4.7)	0.609
Left ventricular ejection fraction, %	55 (49-64)	56 (50-64)	54 (49-59)	0.01
Heart rate, bpm	72 ± 13	71 ± 13	74 ± 13	0.212
ST segment depression, n (%)	9 (5.9)	3 (3)	6 (11.3)	0.039
ST elevation in aVR, n (%)	18 (11.8)	8 (8.1)	10 (18.9)	0.052
T wave inversion, n (%)	49 (32.2)	36 (36.4)	13 (24.5)	0.137
Q wave, n (%)	49 (32.2)	28 (28.3)	21 (39.6)	0.154
Left ventricular hypertrophy, n (%)	26 (17.1)	12 (12.1)	14 (26.4)	0.026
Prolonged JTc, n (%)	43 (28.3)	25 (25.3)	18 (34)	0.256
QRS duration, n (%)				0.418
≤ 80 ms	11 (7.2)	8 (8.1)	3 (5.7)	
81-110 ms	80 (52.6)	55 (55.6)	25 (47.2)	
> 110 ms	61 (40.1)	36 (36.4)	25 (47.2)	
Risk-based ECG score categories, n (%)				0.013
Low risk (0-1)	49 (32.2)	38 (38.4)	11 (20.8)	
Moderate risk (2)	55 (36.2)	35 (35.4)	20 (37.7)	
High risk (3-5)	48 (31.6)	26 (26.3)	22 (41.5)	
High SYNTAX score, n (%)	20 (43.5)	9 (19.1)	18 (30.5)	0.04
SYNTAX score	15 (9-21)	12 (8-16)	25 (22-28)	< 0.001
Three-vessel disease, n (%)	20 (13.2)	0 (0)	20 (37.7)	< 0.001
LMCA disease, n (%)	13 (8.6)	0 (0)	13 (24.5)	< 0.001
Chronic total occlusion, n (%)	53 (34.9)	20 (20.2)	33 (62.3)	< 0.001
Coronary artery calcification, n (%)	34 (22.4)	11 (11.1)	23 (43.4)	< 0.001
Coronary artery bifurcation lesions	27 (17.8)	11 (11.1)	16 (30.2)	0.003
Coronary artery lesion length > 20 mm	104 (68.4)	64 (64.6)	40 (75.5)	0.171
One-year mortality, n (%)	9 (5.9)	4 (4)	5 (9.4)	0.179
Repeat revascularization, n (%)	7 (4.6)	4 (4)	3 (5.7)	0.650

CAD, coronary artery disease; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LMCA, left main coronary artery; SYNTAX, the synergy between percutaneous coronary intervention with Taxus and Cardiac Surgery.

tion in aVR, and risk-based ECG score, in univariable analysis were included in multivariable logistic regression analysis. After adjusting for these confounding factors, a 1-point increase in the risk-based ECG score was associated with a 1.573-fold [95% confidence interval

(CI): 1.111-2.227, $p = 0.011$] increased probability of severe CAD in the coronary angiogram (Table 3). In addition to the prediction of severe CAD, a high-risk score was also associated with clinical outcomes. In the Kaplan-Meier analysis, the high-risk group had a significantly

Table 2. Baseline properties, electrocardiogram, angiographic findings, one-year mortality, and repeat revascularization rates of all cases based on the risk-based ECG score categories

	Low risk (0-1 points) (n = 47)	Moderate risk (2 points) (n = 59)	High risk (≥ 3 points) (n = 46)	p value
Age	63 $\pm$ 11	63 $\pm$ 12	69 $\pm$ 12	0.023
Gender (female), n (%)	30 (63.8)	40 (67.8)	24 (52.2)	0.248
Diabetes mellitus, n (%)	11 (23.4)	15 (25.4)	14 (30.4)	0.729
Hypertension, n (%)	29 (61.7)	33 (55.9)	35 (76.1)	0.096
Smoking, n (%)	21 (44.7)	29 (49.2)	15 (32.6)	0.224
Fasting blood glucose, mg/dl	110 (95-127)	102 (93-120)	113 (96-139)	0.242
Creatinine, mg/dl	0.8 (0.7-0.9)	0.8 (0.7-0.9)	0.9 (0.8-1)	0.588
Total cholesterol, mg/dl	159 (142-211)	164 (140-192)	152 (130-177)	0.214
LDL-C, mg/dl	103 (79-142)	104 (83-124)	91 (76-110)	0.229
HDL-C, mg/dl	35 (29-38)	34 (28-39)	38 (31-43)	0.182
Triglyceride, mg/dl	116 (86-163)	109 (77-155)	101 (77-162)	0.575
Hemoglobin, g/dl	13.9 $\pm$ 1.9	13.9 $\pm$ 2.1	13.2 $\pm$ 1.7	0.123
Troponin I, ng/ml	0.4 (0.05-3.1)	1 (0.3-5.8)	3.4 (0.2-12.4)	0.006
Left ventricular ejection fraction, %	60 (54-65)	59 (49-65)	50 (47-59)	0.002
Heart rate, bpm	71 $\pm$ 14	71 $\pm$ 11	74 $\pm$ 14	0.487
ST segment depression, n (%)	1 (2.1)	4 (6.8)	4 (8.7)	0.381
ST elevation in aVR, n (%)	3 (6.4)	9 (15.3)	6 (13.0)	0.356
T wave inversion, n (%)	14 (29.8)	15 (25.4)	20 (43.5)	0.132
Q wave, n (%)	3 (6.4)	16 (27.1)	30 (65.2)	< 0.001
Left ventricular hypertrophy, n (%)	2 (4.3)	4 (6.8)	20 (43.5)	< 0.001
Prolonged JTc, n (%)	3 (6.4)	17 (28.8)	23 (50)	< 0.001
QRS duration, n (%)				< 0.001
≤ 80	9 (19.1)	2 (3.4)	0 (0)	
81-110	38 (80.9)	33 (55.9)	9 (19.6)	
> 110	0 (0)	24 (40.7)	37 (80.4)	
Severe CAD, n (%)	9 (19.1)	22 (37.3)	22 (47.8)	0.013
High SYNTAX score, n (%)	9 (19.1)	18 (30.5)	20 (43.5)	0.04
SYNTAX score	13 (9-17)	15 (9-20)	21 (15-26)	< 0.001
Three-vessel disease, n (%)	1 (2.1)	7 (11.9)	12 (26.1)	0.003
LMCA disease, n (%)	3 (6.4)	5 (8.5)	5 (10.9)	0.741
Chronic total occlusion, n (%)	13 (27.7)	19 (32.2)	21 (45.7)	0.164
Coronary artery calcification, n (%)	5 (10.6)	13 (22.0)	16 (34.8)	0.02
Coronary artery bifurcation lesions	6 (12.8)	10 (16.9)	11 (23.9)	0.364
Coronary artery lesion length > 20 mm	32 (68.1)	40 (67.8)	32 (69.6)	0.980
One-year mortality, n (%)	1 (2.1)	1 (1.7)	7 (15.2)	0.006
Repeat revascularization, n (%)	1 (2.1)	2 (3.4)	4 (8.7)	0.272

CAD, coronary artery disease; ECG, electrocardiographic; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LMCA, left main coronary artery; SYNTAX, the synergy between percutaneous coronary intervention with Taxus and Cardiac Surgery.

Table 3. Univariable and multivariable predictors of coronary artery disease severity assessed by the SYNTAX score

	Univariable analysis				Multivariable analysis			
	p value	OR	95% CI		p value	OR	95% CI	
			Lower	Upper			Lower	Upper
Female gender	0.044	0.496	0.250	0.983	0.178	0.607	0.294	1.255
LVEF	0.103	0.970	0.935	1.006	0.259	0.977	0.938	1.017
ST-segment depression	0.054	4.085	0.978	17.056	0.366	2.106	0.418	10.599
ST elevation in aVR	0.056	2.645	0.975	7.176	0.251	1.994	0.613	6.485
Risk-based ECG score	0.007	1.602	1.138	2.256	0.011	1.573	1.111	2.227

CI, confidence interval; ECG, electrocardiographic; LVEF, left ventricle ejection fraction; OR, odds ratio; SYNTAX, synergy between percutaneous coronary intervention with Taxus and Cardiac Surgery.

higher one-year mortality rate than the low-risk and moderate-risk groups (hazard ratio: 2.383, 95% CI: 1.395-4.072, $p = 0.001$) (Figure 1). In ROC curve analysis, a risk-based ECG score of ≥ 3 had the best sensitivity of 41.5% and specificity of 73.7% (AUC: 0.638, 95% CI: 0.556-0.714, $p = 0.001$), positive predictive value of 45.8%, and negative predictive value of 70.2%, and it was superior to LVH (AUC: 0.571, 95% CI: 0.489-0.651, $p = 0.039$) in predicting severe CAD (Figure 2).

DISCUSSION

In the present study, we focused on the potential relationship between the severity of CAD and clinical outcomes, including one-year mortality and repeat revascularizations, and a new ECG-based scoring system called 'the risk-based ECG score' in NSTEMI. The risk-based ECG score was significantly associated with the complexity of CAD and clinical outcomes. This is the first study to investigate the role of the risk-based ECG score in predicting severe CAD.

ACS is one of the leading causes of mortality and morbidity globally despite significant advances in the diagnosis and treatment modalities in the last decade. A risk-based approach in which high-risk subjects are referred for early invasive management has been shown to improve prognosis. Various clinical, laboratory, ECG, and imaging parameters that usually reflect the severity of CAD and culprit artery patency have been used for this purpose. ECGs have a unique role in the early diagnosis of high-risk ACS subjects who may benefit

from early revascularization. In the setting of NSTEMI, the diagnostic and prognostic value of ECG parameters remain challenging. Repolarization abnormalities, including ST-segment depression, ST-segment elevation in aVR, and T wave abnormalities, are the most studied parameters for this purpose. Several studies have demonstrated that ST-segment elevation in aVR was associated with the severity of CAD.⁶⁻⁸ The mechanism of ST-segment elevation in aVR in ACS patients reflects transmural ischemia of the basal segment of the inter-ventricular septum, where the current is directed towards the right shoulder.⁹ Gorgels et al. reported an association between ST-segment elevation in aVR in ACS patients and the severity of CAD, LMCA, and three-vessel disease.¹⁰ Two studies conducted by Mirvis et al. and Nikus et al. showed that the presence of ST-T-wave abnormalities was related to severe coronary obstruction, a higher number of diseased vessels, and LMCA disease.^{11,12}

In the present investigation, ST-segment elevation in aVR and ST-segment depressions were associated with severe CAD. However, we did not find a statistically significant association of T wave inversion with CAD severity. We also investigated another repolarization parameter, JTc, calculated by QRS duration extracted from QTc interval, in this study. There was an increase in JTc time in the patients with severe CAD, however it did not

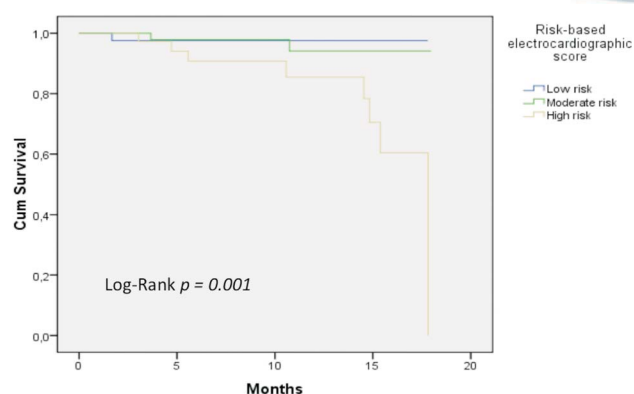


Figure 1. Kaplan-Meier analysis showed high-risk group patients significantly had higher one-year mortality than low-risk and moderate-risk patients.

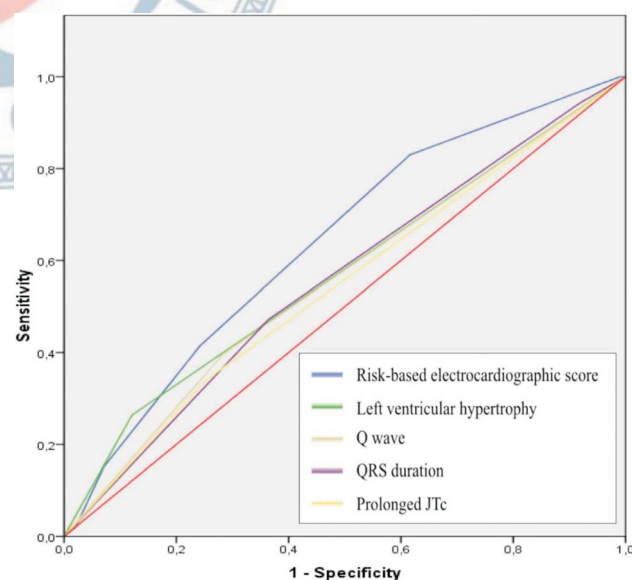


Figure 2. ROC curve analysis demonstrated the comparison of the risk-based ECG score of ≥ 3 with other ECG parameters. ECG, electrocardiographic; ROC, receiver operating characteristic.

reach statistical significance. JTc has been associated with future events in CAD patients, however the relationship between JTc and CAD severity is unclear. Parameters that reflect myocardial mass, including Q wave, LVH, depolarization abnormalities and QRS duration have been studied in patients with CAD. The presence of Q waves classically represents the loss of myocardial mass, and large Q waves have been associated with a worse prognosis than small Q waves. The size of Q waves was shown to determine the extent of myocardial damage and loss of cardiac function resulting in a poor prognosis in a prior study by Godsk et al. However, there are no data regarding CAD severity and Q waves in the literature.¹³ We observed that the presence of Q waves was more prevalent in the severe CAD group, although it did not reach statistical significance. Another ECG parameter of myocardial mass, LVH, was associated with a greater degree of coronary plaque burden in a study conducted by Truong et al.¹⁴ Consistent with this observation, we found that LVH was associated with a 2.603-fold increase in the probability of having severe CAD in univariable analysis. Although myocardial ischemia related to prolonged conduction has been demonstrated in various experimental and clinical studies, we did not observe this relationship.

Integrating various parameters into a single score is clinically essential to make a stronger diagnostic and prognostic tool without increasing costs. ECG risk scores are widely used in daily practice. A newly introduced risk-based ECG score was derived and validated in two large studies to predict sudden and arrhythmic death in patients with CAD.⁵ Because the severity of CAD was associated with all-cause mortality, we hypothesized it might be related to CAD severity. After adjusting for possible confounding factors, a 1-point increase in the risk-based ECG score was associated with a 1.563-fold increase in the probability of severe CAD. There are several possible explanations for these results. First, LVH is observed in clinical conditions, such as hypertension, and develops due to substances secreted from atherosclerotic plaque.^{15,16} Second, prolonged QTc is associated with CAD severity. The essential factor for QT prolongation is remodeling of Na ion channels and myocardial repolarizing ion channels.¹⁷ Data from the Atherosclerosis Risk in Communities Study demonstrated that JTc was a significant independent predictor of CAD events

in a wide QRS complex.¹⁸ However, data regarding the severity of CAD are limited. A high risk-based ECG score was associated with older age, the presence of hypertension, and low LVEF, all of which have been associated with the severity of CAD. We also investigated the relationship between anatomical, depolarization, and repolarization abnormalities and CAD severity in the present study. When these parameters were examined individually, they were all insignificant in independently predicting the severity of CAD. However, when all of these parameters were included in the risk-based ECG score, their power in predicting the severity of CAD was shown. Based on the study results, the risk-based ECG score can help to determine the severity of CAD in NSTEMI patients in daily practice and may give a clue for clinical outcomes.

Limitations of the study

The present study had several limitations. First, the study population was relatively small, and it was a single-center, retrospective study. In addition, several ECG parameters studied in recent years, including frontal QRS-T angle, fragmented QRS complexes, P wave peak time, corrected QT dispersion, terminal QRS distortion, and R wave peak time were not evaluated in our study.

CONCLUSIONS

In this investigation, we showed that the risk-based ECG score, which is simple and easily accessible, may help determine the severity of CAD and clinical outcomes in NSTEMI patients. However, the risk-based ECG score had low sensitivity and low positive predictive value. Therefore, this score should be validated in a large number of patients.

SOURCE OF FUNDING

None.

DECLARATION OF CONFLICTS OF INTEREST

All the authors declare that there are no conflicts of interest.

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