ORIGINAL ARTICLE

Predictive value of electrocardiographic ST-segment elevation myocardial infarction equivalents for detecting acute coronary artery occlusion in patients with non–ST-segment elevation myocardial infarction

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KEY WORDS
electrocardiography, non–ST-segment elevation myocardial infarction, NSTEMI, STEMI equivalent

ABSTRACT

BACKGROUND The sensitivity and accuracy of 12-lead electrocardiography (ECG) for the detection of acute total occlusion (TO) of the culprit coronary artery in non–ST-segment elevation myocardial infarction (NSTEMI) is still suboptimal, particularly for posterolateral circulation.

AIMS We evaluated the prevalence and predictive value of ECG ST-elevation myocardial infarction equivalents (ie, de-Winter ST/T-wave complex, N-wave, T-wave precordial instability, and posterior myocardial infarction) for detecting acute coronary artery occlusion in patients with NSTEMI referred for early invasive treatment.

METHODS A total of 165 patients with NSTEMI were enrolled. The patients were grouped according to the coronary angiography findings into those with TO (Thrombolysis in Myocardial Infarction [TIMI] grade 0) in the culprit artery (n = 43) and those with preserved flow in this vessel (TIMI grades 1–3) (n = 122).

RESULTS The main findings of this study were as follows: 1) 31.5% of patients had at least 1 STEMI equivalent, mostly the N-wave in lead II, III, or aVF; 2) the most common STEMI equivalent in patients with acute TO was T-wave precordial instability; 3) there was a relationship between the prevalence of STEMI equivalents and acute coronary artery occlusion; 4) among all evaluated ECG parameters, only ST-segment depression in leads I, aVL, and V_6 was an independent predictor of acute TO in a multivariate analysis; 5) ST-segment depression in leads I, aVL, and V_6 had higher specificity, positive and negative predictive values, as well as accuracy in predicting acute TO of the culprit vessel, as compared with STEMI equivalents.

CONCLUSIONS STEMI equivalents do not seem to have a relevant advantage over classic ischemic ECG changes in the prediction of acute coronary artery occlusion in patients with NSTEMI.

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INTRODUCTION The underlying pathophysiologic mechanisms for non–ST-elevation myocardial infarction (NSTEMI) are complex and involve incomplete and dynamic coronary thrombosis with or without increased cardiac work and / or transient coronary vasoconstriction.[1-5] However, approximately a quarter of patients with NSTEMI have acute total occlusion (TO) of the culprit coronary artery.[1,3,4] It is well known that these patients have larger infarcts and worse clinical outcomes compared with those with preserved coronary artery flow.[1,6] Early identification of individuals with suspected acute TO of the culprit vessel could probably improve the prognosis in this subpopulation of patients with NSTEMI.

WHAT'S NEW?

It is estimated that approximately a quarter of patients with non–ST-segment elevation myocardial infarction (NSTEMI) have acute total occlusion (TO) of the culprit coronary artery. The sensitivity and accuracy of 12-lead electrocardiography (ECG) for the detection of acute TO of the culprit coronary artery is still suboptimal, particularly for posterolateral circulation. Novel ECG abnormalities that may indicate myocardial ischemia have recently been described. These ECG changes, also known as ST-segment elevation myocardial infarction (STEMI) equivalents, include de Winter ST/T-wave complex, N wave, T-wave precordial instability, and posterior myocardial infarction. To our knowledge, this is the first study that evaluated the predictive value of STEMI equivalents in comparison with classic ischemic ECG changes for detecting acute coronary artery occlusion in patients with NSTEMI referred for early invasive treatment. STEMI equivalents do not seem to have a relevant advantage over classic ischemic ECG changes in the prediction of acute TO of the culprit coronary artery.

Despite the enormous developments in noninvasive diagnostic methods, particularly imaging techniques, electrocardiography (ECG) remains one of the most valuable diagnostic and prognostic tools that may be used for the evaluation of patients with acute coronary syndromes.[1-4] However, the sensitivity and accuracy of 12-lead ECG for the detection of acute TO of the culprit coronary artery is still suboptimal, particularly for posterolateral circulation.[3,4,7,8] Furthermore, it is well known that more than one-third of individuals with non–ST-segment elevation acute coronary syndromes and approximately 20% of patients with NSTEMI have no classic ischemic ECG changes.[1-4]

Recently, novel ECG abnormalities that may indicate myocardial ischemia due to acute TO of the culprit coronary artery have been described.[7-11] These ECG changes, also known as STEMI equivalents, include de Winter ST/T-wave complex,[9,10] N wave (delayed activation wave),[8] T-wave precordial instability,[11] and posterior myocardial infarction.[7] The clinical utility of STEMI equivalents has not been well established, and according to the current guidelines, they are not included in the indications for urgent coronary angiography in patients with NSTEMI.[1]

The aim of this study was to assess the prevalence and predictive value of ECG STEMI equivalents in the diagnosis of acute TO of the culprit coronary artery in patients with NSTEMI referred for early invasive coronary angiography (<24 hours).

METHODS

Patients Patients with NSTEMI included in this study were selected from a group of 510 consecutive individuals who had undergone a coronary angiography within 24 hours from hospital admission during a 3-year period (2015–2017). The following inclusion criteria were applied: presence of acute chest pain for 30 minutes or more; ST-segment depression of 0.5 mm or more in 2 or more contiguous leads, negative T waves of 2 mm or more in 2 or more leads, or absence of ST-segment or T-wave changes; and a rise and fall of serum troponin T levels. The exclusion criteria were as follows: 1) persistent ST-segment elevation at the J point of 2.5 mm or more in men younger than 40 years of age, of 2 mm or more in men aged 40 years or older, or of 1.5 mm or more in women in leads V_2 to V_3 and/or of 1 mm or more in the other 2 or more contiguous leads (except lead aVR); 2) atrial fibrillation; 3) left bundle branch block or ventricular paced rhythm; 4) previous coronary bypass graft surgery; 5) significant valvular heart disease; 6) hyperkalemia defined as potassium levels higher than 5.5 mmol/l. The universal definition of myocardial infarction was applied in this study.[12]

Finally, 165 patients, aged 36 to 91 years, with NSTEMI were enrolled, including 60 women (36.34%). Patients were grouped according to coronary angiography findings into those with acute TO (Thrombolysis in Myocardial Infarction [TIMI] grade 0) in the culprit artery (n = 43) and those with preserved flow in this vessel (TIMI grades 1–3) (n = 122).

Coronary angiography and echocardiography Coronary angiograms were evaluated by 2 experienced invasive cardiologists. The culprit artery was identified by the absence of anterograde coronary blood flow and/or by the presence of local intraluminal thrombus. In addition, ECG and bedside echocardiography supported coronary angiography in assessment of the culprit vessel. Flow in the culprit artery was graded according to the criteria of the TIMI trial.[13] Multivessel coronary disease (MVD) was defined as a stenosis of 70% or more in at least 2 major epicardial coronary arteries, or of 50% or more in the left main coronary artery. Global Registry of Acute Coronary Events scores were calculated using age, heart rate, systolic blood pressure, serum creatinine, Killip class at presentation, cardiac arrest on admission, ST-segment deviation on ECG, and elevated cardiac biomarkers. Left ventricular ejection fraction was assessed on the first echocardiography after hospital admission.

Electrocardiographic definitions The 12-lead ECGs were recorded during chest pain at a paper speed of 25 mm/s and amplification of 10 mm/mV. The heart rhythm, heart rate, deviation of the ST-segment at the J point, and T waves were evaluated on each ECG. An ST-segment depression of 0.5 mm or more in 2 or more contiguous leads was considered clinically significant. ST-segment elevation in lead aVR was defined as an elevation of 0.5 mm or higher.

De Winter ST/T-wave complex was defined as an ST-segment depression of 1 mm or more at the J point, followed by upsloping ST segments and

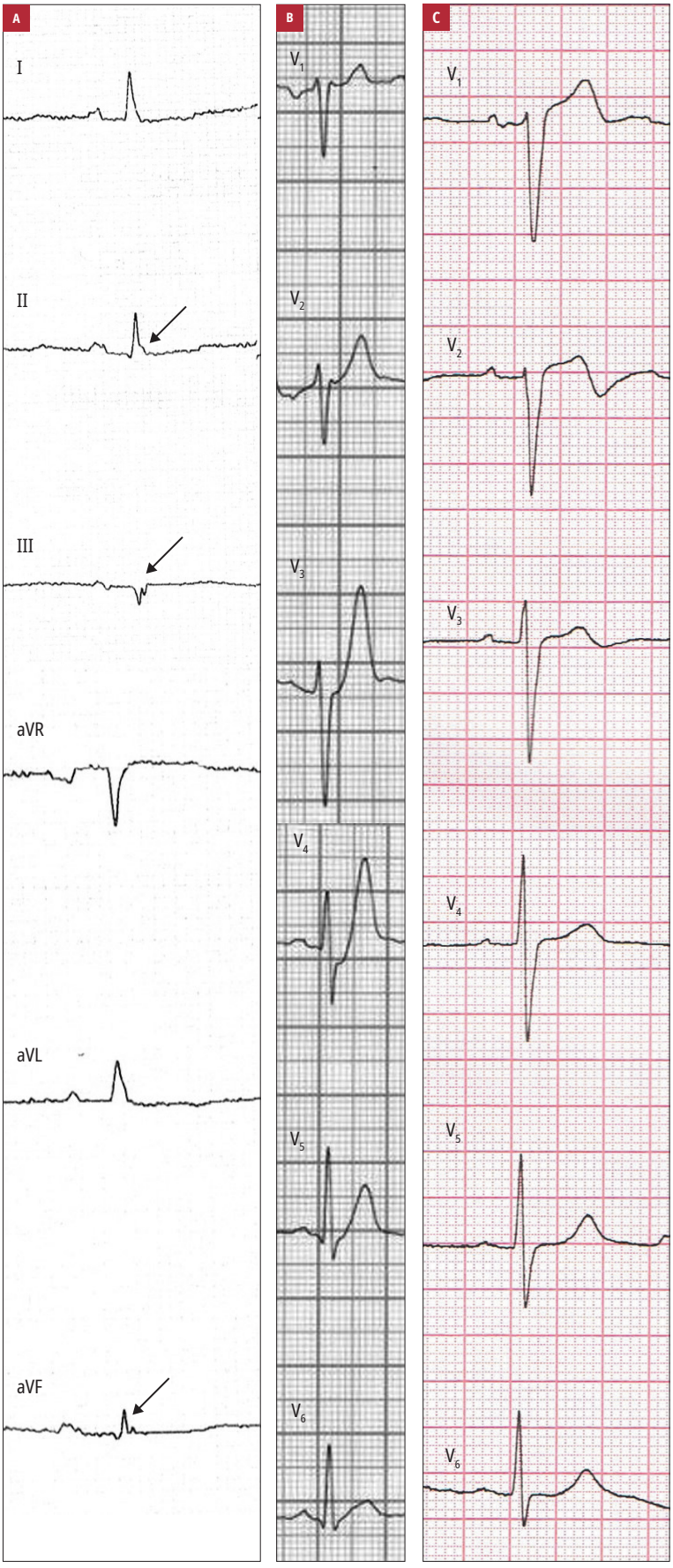

FIGURE 1 Exemplary representative electrocardiograms showing ST-segment elevation myocardial infarction equivalents: **A** – N wave (arrows), **B** – de Winter ST/T-wave complex, **C** – T-wave precordial instability

peaked symmetrical T waves (FIGURE 1).[7,10] N wave was defined as follows: a notch or deflection in the terminal QRS complex; the height of the notch or deflection of 2 mm or more, measured in reference to the PR segment.[7,8] T-wave precordial instability was defined as upright T wave in $V_1 > V_6$.[7,11] Electrocardiographic changes suggestive of posterior myocardial infarction were defined as ST-segment depression of more than 0.5 mm in leads V_1 to V_4 associated with the presence or lack of changes in T wave or the appearance of tall R waves in leads V_1 to V_2.[7]

Statistical analysis Data were expressed as numbers and percentages for categorical variables and as mean (SD) or median (interquartile range [IQR]) for continuous variables. The Shapiro–Wilk test was used to determine normal distribution among continuous variables. Differences between the groups were assessed using the *t* test for normally distributed continuous variables or by the Mann–Whitney test for nonnormally distributed variables. The χ^2 test or Fisher exact test (when any expected cell frequency was <5) were used to evaluate the differences in categorical variables between the respective study groups. A stepwise logistic regression analysis was performed to determine the independent ECG predictors of acute coronary occlusion in the study population. The final multivariate model included only variables that were significant univariate predictors and did not exhibit significant collinearity. The calibration and discrimination of the developed model were assessed using the Hosmer–Lemeshow statistic and the area under the receiver operating characteristic curve, respectively. Two-tailed *P* values of less than 0.05 were considered significant. All calculations were done using the STATISTICA 12.0 software package (StatSoft, Inc., Tulsa, Oklahoma, United States).

RESULTS A total of 165 patients with NSTEMI were included in the study, including 43 patients (26.1%) with an acute occlusion of the culprit artery. The baseline characteristics of the study population are shown in TABLE 1. The study groups did not differ in their baseline profile. There were also no sex differences in demographic and clinical characteristics.

The right coronary artery (RCA) was the most commonly occluded culprit vessel (44.2%), followed by the left circumflex coronary artery (LCx) and/or obtuse marginal branch (OM) (34.9%), and left anterior descending coronary artery (LAD) and/or diagonal branch (Dg) (20.9%). The most frequent culprit arteries in patients with acute TO were the LCx/OM (32.6%), followed by the LAD/Dg (30.2%) and RCA (25.6%) (TABLE 2).

TABLE 1 Baseline characteristics of the study groups

Variable	All patients (n = 165)	TIMI 0 (n = 43)	TIMI 1–3 (n = 122)	*P* value
Age, y, mean (SD)	69.8 (11.7)	67.8 (12.2)	70.5 (11.5)	0.18
Female sex	60 (36.4)	14 (32.6)	46 (37.7)	0.55
Hypertension	149 (90.3)	40 (93.0)	109 (89.3)	0.77
Hypercholesterolemia	150 (90.9)	39 (90.7)	111 (91.0)	1.00
Hypertriglyceridemia	34 (20.6)	7 (16.3)	27 (22.1)	0.42
Type 2 diabetes	65 (39.4)	18 (41.9)	47 (38.5)	0.7
Prediabetes	19 (11.5)	4 (9.3)	15 (12.3)	0.78
Obesity	57 (34.5)	16 (37.2)	41 (33.6)	0.67
Abdominal obesity	84 (50.9)	26 (60.5)	58 (47.5)	0.15
Current smokers	46 (27.9)	11 (25.6)	35 (28.7)	0.7
Former smokers	20 (12.1)	5 (11.6)	15 (12.3)	0.91
Medical history				
Previous STEMI	22 (13.3)	6 (14.0)	16 (13.1)	0.89
Previous NSTEMI	28 (17.0)	6 (14.0)	22 (18.0)	0.54
COPD	14 (8.5)	2 (4.7)	12 (9.8)	0.36
Chronic kidney disease	27 (16.4)	5 (11.6)	22 (18.0)	0.33

Data are presented as number (percentage) of patients unless otherwise indicated. A *P* value of less than 0.05 was considered significant.

Abbreviations: COPD, chronic obstructive pulmonary disease; NSTEMI, non–ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; TIMI, Thrombolysis in Myocardial Infarction

TABLE 2 Clinical and angiographic characteristics of the study group

Variable		All patients (n = 165)	TIMI grade 0 (n = 43)	TIMI grade 1–3 (n = 122)	*P* value
Symptom-to-PCI time, min, median (IQR)		860 (375–1990)	790 (360–1780)	900 (395–2280)	0.01
Heart rate, bpm, mean (SD)		74.9 (14.2)	80.5 (15.2)	72.9 (13.3)	0.01
Systolic BP, mm Hg, mean (SD)		149.6 (22.6)	141.4 (24.2)	152.5 (21.3)	0.01
Diastolic BP, mm Hg, mean (SD)		86.9 (11.0)	86.7 (14.6)	87.0 (11.1)	0.89
LVEF, %, mean (SD)		50.5 (10.6)	47.1 (11.1)	51.7 (10.2)	0.01
Killip class 3, n (%)		5 (3.0)	4 (9.3)	1 (0.8)	0.02
Killip class 4, n (%)		2 (1.2)	0	2 (1.6)	1.00
GRACE score, points, mean (SD)		139.4 (37.2)	143.8 (42.7)	137.8 (35.0)	0.37
cTnT peak, µg/l, median (IQR)		0.32 (0.10–1.03)	0.98 (0.20–2.96)	0.22 (0.08–0.75)	<0.001
CK-MB peak, U/l, median (IQR)		28 (17–56)	57 (23-153)	24 (16–38)	<0.001
CK peak, U/l, median (IQR)		235 (138–663)	655 (231–1577)	209.5 (124–425)	<0.001
Serum creatinine, µmol/l		85 (74–103)	86 (71–100)	84.5 (75–103)	0.99
Culprit lesion, n (%)	LAD / Dg	55 (33.3)	13 (30.2)	42 (34.4)	0.62
	LCx / OM	45 (27.3)	14 (32.6)	31 (25.4)	0.37
	RCA	53 (32.1)	11 (25.6)	42 (34.4)	0.29
	Other	12 (7.3)	5 (11.6)	7 (5.7)	0.3
MVD, n (%)		83 (50.3)	28 (65.1)	55 (45.1)	0.02

A *P* value of less than 0.05 was considered significant.

Abbreviations: BP, blood pressure; CK, creatine kinase; CK-MB, creatine kinase MB; cTnT, cardiac troponin T; Dg, diagonal branch; GRACE, Global Registry of Acute Coronary Events; IQR, interquartile range; LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery; LVEF, left ventricular ejection fraction; OM, obtuse marginal branch; MVD, multivessel coronary disease; PCI, percutaneous coronary intervention; RCA, right coronary artery; others, see TABLE 1

Patients with acute TO had higher heart rate on admission (mean [SD], 80.5 [15.2] bpm vs 72.9 [13.3] bpm; $P = 0.01$) and peak serum levels of cardiac troponin T (median [IQR], 0.98 [0.20–2.96] µg/l vs 0.22 [0.08–0.75] µg/l; $P < 0.001$), creatine kinase (median [IQR], 655 [231–1577] U/l vs 210 [124–425] U/l; $P < 0.001$), and the MB isoenzyme (median [IQR], 57 [23–153] U/l vs 24 [16–38] U/l; $P < 0.001$), but lower admission systolic blood pressure (mean [SD], 141.4 [24.2] mm Hgvs 152.5 [21.3] mm Hg; $P = 0.01$) and left ventricular ejection fraction (mean [SD], 47.1% [11.1%] vs 51.7% [10.2%]; $P = 0.01$), as compared with individuals with preserved coronary flow (TABLE 2).

The symptom-to-percutaneous-coronary-intervention time was shorter in patients with TO (median [IQR], 790 [360–1780] minutes vs 900 [395–2280] minutes; $P = 0.01$). There were no differences in culprit lesion location between the study groups (TABLE 2). However, the prevalence of MVD was higher in individuals with acute TO (65.1% vs 45.1%; $P = 0.02$).

The study groups did not differ in terms of the majority of evaluated ECG parameters (TABLE 3). We found that patients with acute TO had more frequent ST-segment depression in leads I, aVL, and V_6, as compared with those with preserved coronary flow (41.9% vs 17.2%; $P = 0.001$) (FIGURE 2).

TABLE 3 Electrocardiography characteristics and prevalence of ST-elevation myocardial infarction equivalents in the study patients

Variable		n = 165	TIMI grade 0 (n = 43)	TIMI grade 1–3 (n = 122)	*P* value
Sinus rhythm		165 (100)	43 (100)	122 (100)	1.00
Heart rate, bpm, mean (SD)		74.9 (14.2)	80.5 (15.2)	72.9 (13.3)	0.01
Normal QRS axis		134 (81.2)	36 (83.7)	98 (80.3)	0.62
Left QRS axis deviation		31 (18.8)	7 (16.3)	24 (19.7)	0.62
PR duration, ms		160 (160–180)	160 (160–180)	160 (160–180)	0.66
ST-segment elevation in lead aVR		15 (9.1)	2 (4.7)	13 (10.7)	0.36
ST-segment depression in leads:	I, aVL, V_6	39 (23.6)	18 (41.9)	21 (17.2)	0.001
	II, III, aVF	19 (11.5)	2 (4.7)	17 (13.9)	0.16
	V_1–V_6	62 (37.6)	21 (48.8)	41 (33.6)	0.08
Negative T wave in leads:	I, aVL, V_6	22 (13.3)	7 (16.3)	15 (12.3)	0.51
	II, III, aVF	20 (12.1)	5 (11.6)	15 (12.3)	0.91
	V_1–V_6	32 (19.4)	10 (23.3)	22 (18)	0.46
Biphasic T wave in leads:	I, aVL, V_6	3 (1.8)	0	3 (2.5)	0.57
	II, III, aVF	3 (1.8)	2 (4.7)	1 (0.8)	0.17
	V_1–V_6	14 (8.5)	5 (11.6)	9 (7.4)	0.52
Q wave or QS syndrome in leads:	I, aVL, V_6	3 (1.8)	1 (2.3)	2 (1.6)	1.00
	II, III, aVF	27 (16.4)	7 (16.3)	20 (16.4)	0.99
	V_1–V_6	10 (6.1)	2 (4.7)	8 (6.6)	1.00
STEMI equivalent		52 (31.5)	19 (44.2)	33 (27.1)	0.04
De Winter ST/T-wave complex		5 (3)	3 (7)	2 (1.6)	0.11
T wave precordial instability		31 (18.8)	11 (25.6)	20 (16.4)	0.19
N wave in leads:	I	6 (3.6)	2 (4.7)	4 (3.3)	0.65
	aVL	13 (7.9)	5 (11.6)	8 (6.6)	0.33
	I or aVL	13 (7.9)	5 (11.6)	8 (6.6)	0.33
	II	12 (7.3)	2 (4.7)	10 (8.2)	0.73
	III	20 (12.1)	7 (16.3)	13 (10.7)	0.33
	aVF	19 (11.5)	5 (11.6)	14 (11.5)	1.00
	II, III, or aVF	23 (13.9)	7 (16.3)	16 (13.1)	0.61

Data are presented as number (percentage) of patients unless otherwise indicated. A *P* value of less than 0.05 was considered significant.

Abbreviations: see TABLE 1

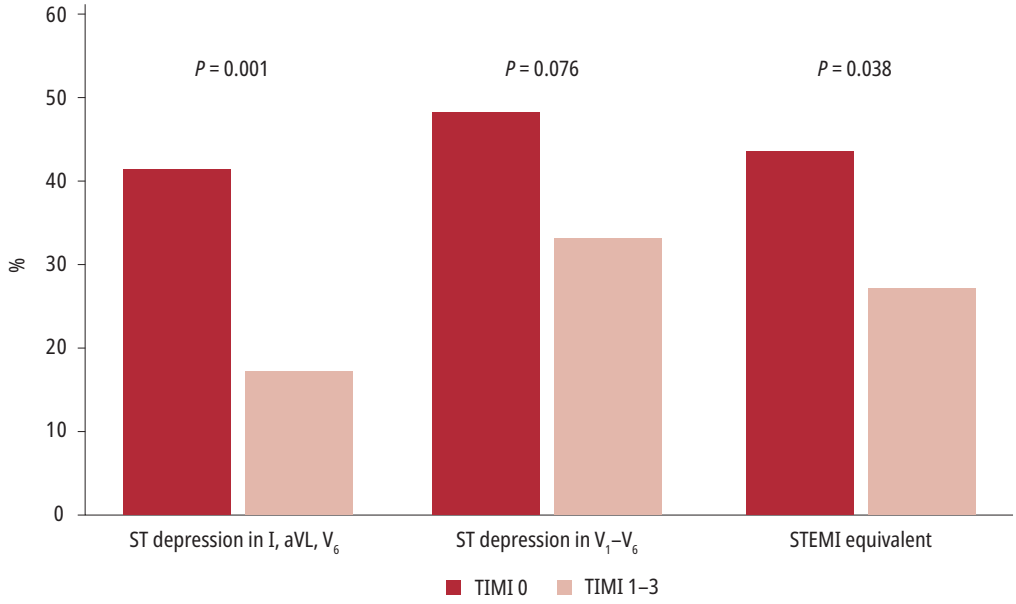

FIGURE 2 Proportion of patients from the Thrombolysis in Myocardial Infarction (TIMI) grade 0 group and TIMI grade 1 to 3 group (%) with ST-segment depression in leads I, aVL, V_6, ST-segment depression in leads V_1 to V_6, and ST-segment elevation myocardial infarction equivalent

TABLE 4 Electrocardiographic predictors of acute coronary artery occlusion in patients with non–ST-segment elevation myocardial infarction

Variable		Univariate analysis	
		OR (95% CI)	*P* value
Sinus rhythm		1 (1–1)	1.00
Heart rate, bpm		1.04 (1.01–1.07)	0.003
Normal QRS axis		1.12 (0.71–1.78)	0.63
Left QRS axis deviation		0.89 (0.56–1.42)	0.63
PR duration, ms		1 (0.99–1.02)	0.81
ST-segment elevation in aVR		0.64 (0.30–1.38)	0.25
ST-segment depression in leads:	I, aVL, V_6	1.86 (1.27–2.73)	0.002
	II, III, aVF	0.55 (0.26–1.17)	0.12
	V1–V_6	1.37 (0.97–1.96)	0.08
Negative T wave in leads:	I, aVL, V_6	1.18 (0.72–1.92)	0.51
	II, III, aVF	0.97 (0.57–1.66)	0.91
	V_1–V_6	1.17 (0.77–1.79)	0.46
Biphasic T wave in leads:	I, aVL, V_6	1 (1–1)	1.00
	II, III, aVF	2.43 (0.72–8.17)	0.15
	V_1–V_6	1.29 (0.72–2.29)	0.39
Q wave or QS syndrome in leads:	I, aVL, V_6	1.2 (0.36–4.02)	0.77
	II, III, aVF	1 (0.62–1.59)	0.99
	V_1–V_6	0.83 (0.38–1.85)	0.65
STEMI equivalent		1.46 (1.02–2.1)	0.04
De Winter ST/T-wave complex		2.12 (0.85–5.28)	0.11
T-wave precordial instability		1.32 (0.87–2.01)	0.19
N wave in leads:	I	1.2 (0.5–2.86)	0.68
	aVL	1.37 (0.76–2.47)	0.3
	I or aVL	1.37 (0.76–2.47)	0.3
	II	0.74 (0.34–1.61)	0.45
	III	1.28 (0.78–2.1)	0.34
	aVF	1.01 (0.59–1.73)	0.98
	II, III, or aVF	1.14 (0.70–1.84)	0.61

A *P* value of less than 0.05 was considered significant.

Abbreviations: OR, odds ratio; others, see TABLE 1

The prevalence of STEMI equivalents in patients with NSTEMI was 31.5%. T-wave precordial instability and de-Winter ST/T-wave complex were present in 18.8% and 3.0% of the participants, respectively. Twenty-three (13.9%) of all patients had N wave in lead II, III, or aVF, and 13 patients (7.9%) had N wave in lead I or aVL (TABLE 3). None of the participants had features of posterior myocardial infarction.

The most common STEMI equivalent in patients with acute TO was T-wave precordial instability (25.6%). However, there were no intergroup differences in the occurrence of T-wave precordial instability, de Winter ST/T-wave complex, as well as N wave (TABLE 3). Importantly, at least 1 STEMI equivalent was more frequent in patients who had a completely occluded culprit artery (44.2% vs 27.1%; $P = 0.04$). These findings were confirmed by a univariate logistic regression analysis (odds ratio, 1.46; 95% CI, 1.02–2.10; $P = 0.04$) (TABLE 4). In a multivariate analysis, only ST-segment depression in leads I, aVL, and V_6 remained associated with acute coronary artery occlusion (odds ratio, 1.82; 95% CI, 1.11–2.98; $P = 0.02$). The Hosmer–Lemeshow test showed good fit of the model ($\chi^2 = 4.75$; $P = 0.69$), and the area under the receiver operating characteristic curve was 0.724.

The sensitivity and specificity of STEMI equivalents in the prediction of acute coronary occlusion were 44.2% and 73.0%, respectively (TABLE 5). The accuracy of these ECG findings in the prediction of acute TO of the culprit vessel was 65.5% and was lower than the accuracy of ST-segment depression in leads I, aVL, and V_6, which was 72.1% (TABLE 5).

DISCUSSION To our knowledge, this is the first study that evaluated the predictive value of STEMI equivalents in comparison with classic ischemic ECG changes for detecting acute coronary

TABLE 5 The value of ST-segment elevation myocardial infarction equivalents and ST-segment depression in lead I, aVL, and V_6 in predicting acute coronary artery occlusion in patients with non–ST-segment elevation myocardial infarction

Variable	STEMI equivalent	ST depression in leads I, aVL, V_6
True positive, n	19	18
True negative, n	89	101
False-positive, n	33	21
False-negative, n	24	25
Sensitivity, %	44.2 (31.1–57.4)	41.9 (29.3–54.2)
Specificity, %	73.0 (68.3–77.6)	82.8 (78.4–87.1)
PPV, %	36.5 (25.7–47.5)	46.2 (32.3–59.7)
NPV, %	78.8 (73.8–83.8)	80.2 (75.9–84.4)
Accuracy, %	65.5 (58.6–72.4)	72.1 (65.6–78.5)
LR(+)	1.63 (0.98–2.57)	2.43 (1.36–4.21)
LR(–)	0.77 (0.55–1.01)	0.70 (0.53–0.90)
Diagnostic odds ratio	2.14 (0.98–4.68)	3.46 (1.50–8.01)
Youden index, J	0.17 (-0.01 to 0.35)	0.25 (0.08–0.41)

Values in parentheses are 95% confidence intervals.

Abbreviations: LR(+), positive likelihood ratio; LR(–), negative likelihood ratio; NPV, negative predictive value; PPV, positive predictive value; others, see TABLE 1

artery occlusion in patients with NSTEMI referred for early invasive treatment.

The main findings of our study were as follows: 1) almost one-third of patients with NSTEMI had at least 1 STEMI equivalent on 12-lead ECG, most frequently the N wave in lead II, III, or aVF; 2) the most common STEMI equivalent in patients with acute TO was T-wave precordial instability; 3) there was a relationship between the prevalence of STEMI equivalents and acute TO of the culprit artery; 4) among all evaluated ECG parameters, only ST-segment depression in leads I, aVL, and V_6 was an independent predictor of acute coronary artery occlusion in the multivariate analysis; 5) ST depression in leads I, aVL, and V_6 had higher specificity, positive and negative predictive values, as well as accuracy in predicting acute TO of the culprit vessel, as compared with STEMI equivalents.

In the present study, acute coronary artery occlusion was found in 26.1% of patients with NSTEMI. It is well known that acute TO of the culprit artery usually presents with ST-segment elevation myocardial infarction.[1-4] However, numerous clinical studies have demonstrated that approximately one-fourth of patients with NSTEMI had also acute TO, mostly located in the LCx or its branches.[1-4] As shown in a recent meta-analysis by Khan et al,[6] these patients with NSTEMI are at a higher risk for mortality and major adverse cardiac events as compared with those with preserved coronary flow.[1]

The standard 12-lead ECG is the first-line diagnostic tool in the assessment of patients with suspected acute coronary syndrome. However, the association of classic ischemic ECG changes (such as ST-segment depression, transient ST-segment elevation, and negative or biphasic T waves) and other ECG findings, including STEMI equivalents, with the affected coronary territory is poorly established.[1,14] This relationship is even more unclear in patients with NSTEMI and MVD.[1]

We found that T-wave precordial instability was the most prevalent STEMI equivalent in patients with NSTEMI, and this observation was consistent with the findings of Wall et al.[7] It has been suggested that T-wave precordial instability may indicate acute occlusion of the LAD.[11] Similarly, de Winter ST/T-wave complex was originally described as an ECG sign of proximal LAD occlusion.[9] In our study, this STEMI equivalent was present only in 3% of patients with NSTEMI. This figure is lower than the 14% quoted by Wall et al,[7] but similar to the result from the study by Verouden et al[10] (2%). Contrary to Wall et al,[7] we found that the LAD / Dg was the most frequent culprit artery, followed by RCA and LCx / OM. There was no association of these 2 STEMI equivalents with acute coronary occlusion in our study.

The N wave in lead II, III, or aVF was found in 13.9% of patients with NSTEMI and in 16.3% of those with acute coronary occlusion. The occurrence of the N wave in lead I or aVL was less common: 7.9% and 11.6%, respectively. Our results are similar to those reported recently by Wall et al.[7] However, these results are contrary to those obtained by Niu et al,[8] who found the N wave in 77% of patients with NSTEMI. Due to this discrepancy and the fact that our study is only the third to analyze the occurrence of the N wave in patients with NSTEMI, further research is needed before conclusions are drawn as to its clinical significance. It is hypothesized that N waves can be caused by delayed depolarization of the left ventricular basal region (which is supplied by the LCx and / or OM) due to ischemia.[8] In our study, however, there were no intergroup differences in the prevalence of N waves.

Our results suggest that none of the STEMI equivalents remained an independent predictor of acute TO in the multivariate analysis. The accuracy of these ECG findings in the prediction of acute coronary occlusion was lower than the accuracy of ST-segment depression in leads I, aVL, and V_6.

Study limitations Our study has several limitations. The first limitation is the retrospective design of the analysis. Second, the culprit vessel was determined by cardiologists in the catheterization laboratory (using ECG, echocardiographic, and angiographic findings), and identification

of this artery in individuals with MVD may have differed between operators. Third, data regarding detailed angiographic features, such as myocardial blush grade and thrombus burden are lacking. Finally, the sample size was relatively small and a larger sample would probably provide more robust findings.

Conclusions In conclusion, our study showed an association between ECG STEMI equivalents and acute coronary occlusion in patients with NSTEMI. However, none of these STEMI equivalents remained an independent predictor of TO in the multivariate analysis. We conclude that these novel ECG signs do not seem to have a relevant advantage over classic ischemic ECG changes, including ST-segment depression, in the prediction of acute TO of the culprit coronary artery. Future studies are needed to identify the independent predictors of acute coronary occlusion in patients with NSTEMI.

ARTICLE INFORMATION

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