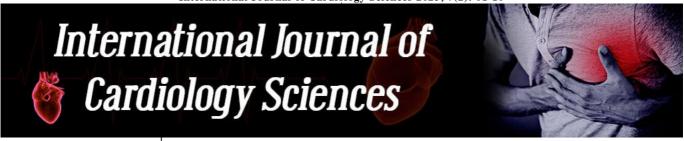
International Journal of Cardiology Sciences 2025; 7(2): 01-10



ISSN Print: 2664-9020 ISSN Online: 2664-9039 Impact Factor: RJIF 5.63 IJCS 2025; 7(2): 01-10 www.cardiologyjournals.net Received: 25-04-2025 Accepted: 27-05-2025

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Role of electrocardiographic findings in the prognosis of Non-ST-Segment elevation myocardial infarction at admission

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DOI: https://www.doi.org/10.33545/26649020.2025.v7.i2a.112

Abstract

Background: The four subgroups of the electrocardiogram (ECG) might be useful in risk stratification and in choosing the best treatment plans for individuals with non-ST elevation myocardial infarction (NSTEMI). Invasive reperfusion is advised for NSTEMI patients in current guidelines in order to enhance clinical results. The purpose of this study was to evaluate the prognostic significance of qualitative ECG results in individuals who presented with myocardial infarction that was not ST-segment elevation.

Methods: This prospective cohort study was carried out on 260 patients with non-STEMI according to European society of cardiology guidelines 2023 Symptoms of ischemia (e.g. chest discomfort, angina equivalent and silent ischemia) and ECG changes indicative of new ischemia new ST-T changes or new left bundle branch block. According to the Fourth Universal Definition of Myocardial Infarction, all patients were divided into four groups based on their ECG results upon presentation: Transient ST segment elevation (TSTE) in Group I: Group II ST segment depression (STD) is defined as new or presumed-new horizontal or downsloping ST depression >0.05 mV in two contiguous leads below the isoelectric line on the ECG. TSTE is defined as new or presumed-new ST-segment elevation lasting 0.1 mV in all leads except V2-V3, where the following cut points applied: >0.2 mV in men aged >40 years, >0.25 mV in men aged 40 years, or >0.15 mV in women. T wave inversion (TWI) in group III is characterised as new or presumed-new TWI of >0.1 mV in two adjacent leads with a noticeable R wave or an R/S ratio greater than 1. Group IV NIC: If the ECG did not indicate TSTE, TW, or STD at admission, it was deemed to demonstrate NIC.

Results: In multivariate regression, Cardiac arrest, Killip Class III-IV, GP IIb IIIa inhibitor, No. of diseased vessels and global registry of acute coronary events (GRACE) score were independent predictors of Mortality, major GUSTO bleeding, reinfarction and arrhythmia while Age, sex, smoking, HTN, DM, Previous MI, Previous Percutaneous coronary intervention (PCI), Previous coronary artery bypass grafting (CABG) I, signs of congestive heart failure (CHF) on presentation, Diagnostic catheterization, systolic blood pressure, diastolic blood pressure, Heart Rate, Previous congestive heart failure, Creatinine, Troponin, Aspirin, ticagrelor, Beta blocker, angiotensin-converting enzyme inhibitor/ angiotensin ii receptor blocker (ARB), statin, anticoagulant and dyslipidemia were not.

Conclusions: In NSTEMI patients, ST-segment depression signals higher risk, while T-wave inversion suggests a better short-term outlook. ECG aids initial risk assessment but doesn't predict outcomes after invasive treatment. A high Killip class, many diseased vasculature, the use of GP IIb/IIIa inhibitors, cardiac arrest, and GRACE score are important indicators of death.

Keywords: Electrocardiographic findings, prognosis of Non-St-Segment elevation, myocardial infarction, acute coronary syndrome

Introduction

The electrocardiogram (ECG) provides important information about presence, extent and severity of cardiovascular disease. When a patient is suspected of having acute coronary syndrome (ACS), electrocardiograms (ECGs) are the initial line of examination because they are the most accessible and non-invasive diagnostic technique [1].

Non-ST-elevation myocardial infarction (NSTEMI), a component of ACS, is typically brought on by partial or almost total blockage of a coronary artery, which reduces blood supply to the heart and causes myocardial damage or infarction, which raises troponin levels [2].

Transient ST-segment elevation (TSTE), ST-segment depression (STD), T-wave inversion (TWI), and no ischaemic changes (NIC) are among the differences in the ECG findings of NSTEMI at presentation. For patients with NSTEMI, the ECG variations may offer prognostic information [3].

Choosing the best management plans for NSTEMI patients and risk-stratifying them are made easier by the four ECG subgroups. Invasive reperfusion is advised by current recommendations for NSTEMI patients in order to enhance clinical outcomes [4].

The purpose of this study was to evaluate the prognostic significance of qualitative ECG results in individuals who presented with myocardial infarction that was not ST-segment elevation.

Patients and Methods

This prospective cohort study was carried out on 260 patients aged from 19 to 65 years old, both sexes, with non-STEMI according to European society of cardiology guidelines 2023 ^[5]: Symptoms of ischemia (e.g. chest discomfort, angina equivalent and silent ischemia) and ECG changes indicative of new ischemia new ST-T changes or new left bundle branch block.

The study was done from August 2022 to July 2023 after approval from the Ethical Committee Tanta University Hospitals, Tanta, Egypt (approval code: 33017/03/19) and registration of clinicaltrials.gov (ID: NCT03938220). An informed written consent was obtained from the patient.

Exclusion criteria were patients with acute persistent ST segment elevation MI, neoplastic diseases, cerebral hemorrhage or head trauma, recent major surgical procedure or trauma, thyroid disorder psychiatric disorder and pacemaker. All patients were categorized into four groups based on ECG findings at presentation according to the Fourth Universal Definition of Myocardial Infarction $^{[6]}$: Group I Transient ST segment Elevation (TSTE): TSTE is defined as new or presumed-new ST-segment elevation lasting 0.1 mV in all leads other than V2-V3, where the following cut points applied: >0.2 mV in men age > 40 years, >0.25 mV in men age < 40 years, or >0.15 mV in women.

ST segment depression (STD) in Group II: STD is defined as a new or suspected new horizontal or downsloping ST depression greater than 0.05 mV in two consecutive leads underneath the ECG's isoelectric line. Group III T wave inversion (TWI) is characterised by a distinct R wave or R/S ratio greater than one, and a new or presumed-new TWI of >0.1 mV in two adjacent leads. Group IV NIC: If the admission ECG did not indicate TSTE, TW, or STD, the ECG was deemed to exhibit NIC. The patient was placed in the TSTE group if the ECG showed TSTE, regardless of the

presence of other abnormalities (STD and TWI). The patient was placed in the STD group if the ECG showed TWI and STD but not TSTE. The TWI group consisted of patients whose ECGs only displayed TWI.

All patients were subjected to complete history taking, complete clinical examination, routine laboratory investigations [Hb, PLT, TLC, kidney function test, lipid test, cardiac biomarker (CK-MB, troponin], medication and procedure (aspirin, ticagrelor, β blocker, angiotensin-converting enzyme (ACE) inhibitor/ARB, statin, GP IIb-IIIa inhibitor, anticoagulant) in NSTEMI patients, Standard 12-lead ECG.

Systemic hypertension is defined as systolic blood pressure of 140 mm-19 Hg or more and/or diastolic blood pressure (DBP) of 90 mm Hg or more measured on 3 separate occasions with or without treatment before admission [7].

Sample Size Calculation

The statistical program Epi-Info, developed by the World Health Organisation and the Centres for Disease Control and Prevention in Atlanta, Georgia, USA, version 2002, was used to determine the sample size and power analysis. The following standards were applied while determining the sample size: The research design is cross-sectional with a 95% confidence level; the prognostic value of qualitative ECG findings on the prognosis is estimated at 80% with a 10% margin of error; the sample size based on the aforementioned criteria was found to be N>61 in each group; that number was raised to 65 in order to make up for missing data and enhance the study's data quality.

Statistical analysis

Microsoft SPSS version 27.0 was used for data administration and analysis. For both men and women, the quantitative variables' means and standard deviations are presented. The Shapiro-Wilk test allowed for the evaluation of the data's normalcy. Using Kruskal-Wallis for non-parametric variables, the four groups were compared. The mean and standard deviation (SD) of variables with a normal distribution are used to express them, while the median and 25th and 75th percentiles are used to convey variables without a normal distribution. For categorical data, the Chi square test was employed. If P was less than 0.05, the difference was deemed significant. Binary logistic regression was used to determine a 95% confidence interval (CI) and evaluate risk. When the two P values were less than 0.05, statistical significance was taken into account.

Results

There was insignificant difference among the four groups as regards age and sex. Table 1.

Table 1: Baseline characteristics, medical history and risk factors in NSTEMI patients classified by admission electrocardiographic findings

		ST-Segment Elevation	ST-Segment Depression	T-Wave Inversion	No Ischemic Changes	р
Ag	ge (years)	61.00 ± 5.65	62.43 ± 6.92	61.06 ± 5.90	59.54 ± 6.10	0.069
Sex	Male	46 (70.8%)	46 (70.8%)	44 (67.7%)	44 (67.7%)	0.962
sex	Female	19 (29.2%)	19 (29.2%)	21 (32.3%)	21 (32.3%)	0.962
			Medical history and risk f	actors		
	HTN	32 (49.2%)	38 (58.5%)	40 (61.5%)	37 (56.9%)	0.537
	DM	33 (50.8%)	34 (52.3%)	35 (53.8%)	37 (56.9%)	0.910
Dys	slipidemia	36 (55.4%)	40 (61.5%)	27 (41.5%)	37 (56.9%)	0.121
Pre	evious MI	14 (21.5%)	19 (29.2%)	12 (18.5%)	16 (24.6%)	0.514
Pre	vious CHF	9 (13.8%)	13 (20.0%)	11 (16.9%)	10 (15.4%)	0.807
Pre	vious PCI	16 (24.6%)	15 (23.1%)	14 (21.5%)	14 (21.5%)	0.971

Data are presented as mean ± SD or frequency (%). HTN: Hypertension, DM: Diabetes mellitus, MI: Myocardial infraction, CHF: Congestive Heart Failure, PCI: Percutaneous coronary intervention.

There was insignificant difference among the four groups as regards medical history and risk factors (Hypertension (HTN), diabetes mellitus (DM), Dyslipidemia, Previous MI, Previous Congestive Heart Failure (CHF) and Previous PCI), laboratory investigations, (aspirin, ticagrelor, β Blocker, ACE inhibitor/ angiotensin ii receptor blocker (ARB), Anticoagulant and statin) and ejection fraction (EF) in patients **NSTEMI** classified admission by electrocardiographic findings. There was insignificant difference among the four groups as regards systolic blood pressure (SBP), DBP and cardiac arrest. Heart rate and Killip Class IIIIV were statistically significantly different among the four groups. Heart rate was statistically significantly

higher in ST-Segment Depression group, T Wave Inversion group and NIC group than TSTE group (P value=0.003, 0.003 and 0.029 respectively) while was insignificantly different between TWI group and (ST Segment Depression group and NIC group). Killip Class III-IV was statistically significantly higher in (ST-Segment Depression group and TWI group) than TSTE group and NIC group and NIC group) while was insignificantly different between TSTE and NIC and between ST-Segment Depression and TWI. GP IIb-IIIa inhibitor was significantly higher in ST-Segment Depression and TWI than (TSTE and NIC group) (P value<0.05) while insignificantly different between TSTE group and NIC group and between ST-Segment Depression group and TWI group. Table 2.

Table 2: Clinical findings of NSTEMI patients, laboratory investigations, medication and procedure classified by admission electrocardiographic findings

	ST-Segment Elevation	ST-Segment Depression	T-Wave Inversion	No Ischemic Changes	р	
	Clinical findings of NSTEMI patients					
SBP (mmHg)	142.37±11.50	143.71±10.36	140.54±10.66	140.35±11.43	0.253	
DBP (mmHg)	81.34 ± 7.18	83.20 ± 7.25	81.55 ± 7.76	82.25 ± 7.46	0.478	
H (D (d)	79.75 ± 6.37	83.17 ± 6.81	83.00 ± 6.15	82.18 ± 6.22	0.001*1	
Heart Rate (bpm)	P1=0	.003*, P2=0.003*, P3=0.029	0*, P4=0.881, P5=0.3	88, P6=0.451	•	
K.II. CI HI IV	7 (10.8%)	28 (43.1%)	18 (27.7%)	5 (7.7%)	0.001*2	
Killip Class III-IV	P1< 0.	001*, P2=0.026*, P3=0.761	, P4=0.098, P5< 0.00	1*, P6=0.005*	•	
Cardiac Arrest	0 (0.0%)	2 (3.1%)	1 (1.5%)	1 (1.5%)	0.567	
		Laboratory investigations				
Hb (mg/dl)	10.57 ± 0.87	10.51 ± 0.77	10.62 ± 0.78	10.65 ± 0.79	0.764	
PLT (× 10 ⁹ /L)	265.97±58.55	275.05±63.47	274.09±54.05	272.37±67.49	0.832	
TLC (×10 ³ /μl)	7.63 ± 1.24	7.73 ± 1.08	7.75 ± 1.17	7.95 ± 1.18	0.465	
Urea (mg/dl)	34.85 ± 6.07	33.82 ± 5.31	35.40 ± 4.40	35.48 ± 4.94	0.242	
Baseline creatinine (mg/dl)	0.98 ± 0.18	0.99 ± 0.15	1.00 ± 0.16	1.0 ± 0.14	0.887	
Cholesterol (mg/dl)	240.45 ± 56.71	245.09 ± 57.78	243.63 ± 62.25	249.00 ± 54.86	0.866	
TG (mg/dl)	177.54 ± 73.73	189.51 ± 65.32	180.97 ± 74.52	195.72 ± 64.88	0.438	
]	Medication and procedure			•	
Aspirin	63 (96.9%)	63 (96.9%)	64 (98.5%)	65 (100%)	0.524	
Ticagrelor	63 (96.9%)	63 (96.9%)	64 (98.5%)	65 (100%)	0.524	
β Blocker	60 (92.3%)	61 (93.8%)	56 (86.2%)	60 (92.3%)	0.421	
ACE Inhibitor/ARB	42 (64.6%)	51 (78.5%)	48 (73.8%)	40 (61.5%)	0.126	
Statin	59 (90.8%)	62 (95.4%)	61 (93.8%)	57 (87.7%)	0.383	
CD III- III- I-l-il-it	14 (21.5%)	28 (43.1%)	26 (40.0%)	7 (10.8%)	0.001*1	
GP IIb-IIIa Inhibitor	P1=0.014*, P2=0.036*, P3=0.152, P4=0.858, P5< 0.001*, P6=0.003*					
Anticoagulant	65 (100%)	63 (96.9%)	64 (98.5%)	65 (100%)	0.295	
EF (%)	48.49 ± 7.81	46.85 ± 8.72	46.05 ± 7.61	47.92 ± 7.39	0.294	
D	1' (IOD) 1 CI	D	40.05 IOD I	. 1 CD C. 1		

Data represent as Mean \pm SD, Median (IQR) or numbChanges, Pntage), Statistically significant as p \leq 0.05, IQR: Inter quartile range, SD: Standard deviation, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, P1: P value between Transient ST-Segment Elevation and ST-Segment Depression, P2: P Value between Transient ST-Segment Elevation and No Ischemic Changes, P4: P value between ST-Segment Depression and T-Wave Inversion, P5: P value between ST-Segment Depression and No Ischemic Changes, P6: P value between T-Wave Inversion and No Ischemic Changes, Hb: Hemoglobin, PLT: Platelets, TLC: Total leukocyte count, TG: Triglyceride.

There was a significant difference among the four groups as regards global registry of acute coronary events (GRACE) risk score (P value=0.001), No. of diseased vessels, stenotic vessels, grading of stenosis and location of stenosis. GRACE

risk score was significantly higher in ST-Segment Depression group than TSTE group and NIC group and higher in T Wave Inversion group than NIC group (P value<0.05). Table 3.

Table 3: GRACE risk score in NSTEMI patients classified by admission electrocardiographic findings

	ST-Segment Elevation	ST-Segment Depression	T-Wave Inversion	No Ischemic Changes	р	
		Grace Ris	k Score	-		
Low	15 (23.1%)	5 (8.0%)	10 (15.4%)	19 (29.2%)		P1= 0.008*
Intermediate	30 (46.2%)	25 (38.5%)	27 (41.5%)	30 (46.2%)		P2 = 0.287
					0.001*1	P3 = 0.632
High	20 (30.8%)	35 (53.8%)	28 (43.1%)	16 (24.6%)		P4 = 0.283
Tilgii	20 (30.8%)	33 (33.6%)	28 (43.170)	10 (24.0%)		P5=0.004*
						P6= 0.044*
	No. of Diseased Vessels					
One	35 (53.8%)	14 (21.5%)	15 (23.1%)	39 (60.0%)	0.001*1	P1=0.001*
Two	21 (32.3%)	16 (24.6%)	25 (38.5%)	19 (29.2%)	0.381	P2=0.001*

Multiple	9 (13.8%)	35 (53.8%)	25 (38.5%)	7 (10.8%)	0.001*	P3= 0.753 P4= 0.159 P5<0.001* P6<0.001*
		Stenotic \	Vessels			
IM	0 (0.0%)	15 (23.1%)	10 (15.4%)	0 (0.0%)	0.001*2	P1=0.001*
AD	35(53.8%)	33 (50.8%)	32 (49.2%)	20 (30.8%)	0.036*	P2=0.002*
LCX	20(30.8%)	12 (18.5%)	12 (18.5%)	26 (40.0%)	0.012*	P3=0.022*
						P4=0.729
RCA	10(15.4%)	29 (44.6%)	20 (30.8%)	19 (29.2%)	0.004*	P5=0.001*
						P6=0.001*
		Grading of	Stenosis			
Minimal (<25%)	7(10.8%)	0 (0.0%)	0 (0.0%)	5 (7.7%)	0.004*3	P1<0.001*
Mild (25-49%)	19 (29.2%)	10 (15.4%)	3 (4.6%)	15 (23.1%)	0.002*	P2<0.001*
Moderate (50-69%)	11 (16.9%)	8 (12.3%)	10 (15.4%)	30 (46.2%)	<0.001*	P3=0.006*
Severe (Subtotal 70-99%)	26 (40.0%)	30 (46.2%)	32 (49.2%)	15 (23.1%)	0.011*	P4=0.231
Total Occlusion (100%)	2 (3.1%)	17 (26.2%)	20 (30.8%)	0 (0.0%)	0.001*	P5<0.001*
Minimal (<25%)	7(10.8%)	0 (0.0%)	0 (0.0%)	5 (7.7%)	0.004*3	P6<0.001*
		Location of	Stenosis			
Proximal	2 (3.1%)	14 (21.5%)	10 (15.4%)	0 (0.0%)	0.001*4	P1<0.001*
Mid	24 (36.9%)	6 (9.2%)	20 (30.8%)	18 (27.7%)	0.002*	P2=0.030*
Distal	22 (33.8%)	15 (23.1%)	11 (16.9%)	33 (50.8%)	<0.001*	P3=0.249
Proximal + Mid	9 (13.8%)	21 (32.3%)	14 (21.5%)	8 (12.3%)	0.017*	P4=0.036*
Mid + Distal	8 (12.3%)	9 (13.8%)	10 (15.4%)	6 (9.2%)	0.750	P< 0.001* P6<0.001*

^{*}Statistically significant as $p \le 0.05$, Data are presented as number (percentage), P1: P value between Transient ST-Segment Elevation and ST Segment Depression, P2: P Value between Transient ST-Segment Elevation and T-Wave Inversion, P3: P value between Transient ST-Segment Elevation and No Ischemic Changes, P4: P value between ST-Segment Depression and T-Wave Inversion, P5: P value between ST-Segment Depression and No Ischemic Changes, P6: P value between T-Wave Inversion and No Ischemic Changes.

There was insignificant difference among the four groups as regards stroke, major gusto bleeding and reinfarction. but all-cause mortality, arrhythmia and heart failure were statistically significant difference among the four groups inhospital. There was insignificant difference among the four

groups as regards stroke, major gusto bleeding, reinfarction Stroke. Heart failure and all-cause mortality and arrhythmia were statistically significantly different among the four groups after 30-days. Table 4.

Table 4: In-hospital and 30-days outcomes in NSTEMI patients classified by admission electrocardiographic findings.

Outcome	ST-Segment Elevation	ST-Segment Depression	T-Wave Inversion	No Ischemic Changes	р		
	In-hospital outcomes						
All-cause mortality	2 (3.1%)	11 (16.9%)	3 (4.6%)	2 (3.1%)	0.028*1		
Stroke	0 (0.0%)	2 (3.1%)	1 (1.5%)	1 (1.5%)	0.565		
Major GUSTO bleeding	2 (3.1%)	7 (10.8%)	1 (1.5%)	2 (3.1%)	0.072		
Reinfarction	0 (0.0%)	2 (3.1%)	1 (1.5%)	2 (3.1%)	0.052		
Arrhythmia	7 (10.8%)	20 (30.8%)	19 (29.2%)	2 (3.1%)	<0.001*		
Heart failure	3 (4.6%)	10 (15.4%)	4 (6.2%)	2 (3.1%)	0.032*		
		30-days outcomes					
All-cause mortality	1 (1.5%)	5 (7.7%)	1 (1.5%)	0 (0.0%)	0.034*2		
Stroke	3 (4.6%)	5 (7.7%)	1 (1.5%)	2 (3.1%)	0.344		
Major GUSTO bleeding	1 (1.5%)	1 (1.5%)	1 (1.5%)	0 (0.0%)	0.798		
Reinfarction	2 (3.1%)	5 (7.7%)	1 (1.5%)	0 (0.0%)	0.065		
Arrhythmia	3 (4.6%)	11 (16.9%)	4 (6.2%)	1 (1.5%)	0.004*		
Heart failure	5 (7.7%)	10 (15.4%)	6 (9.2%)	4 (6.2%)	0.299		

*Statistically significant as $p \le 0.05$, P1: P value between Transient ST-Segment Elevation and ST-Segment Depression, P2: P Value between Transient ST-Segment Elevation and Twave Inversion, P3: P value between Transient ST-Segment Elevation and No Ischemic Changes, P4: P value between ST-Segment Depression and T-Wave Inversion, P5: P value between ST-Segment Depression and No Ischemic Changes, P6: P value between T-Wave Inversion and No Ischemic Changes.

In multivariate regression, Cardiac arrest, Killip Class III-IV, GP IIb IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Mortality and major GUSTO bleeding while Age, sex, smoking, HTN, DM, Previous MI, Previous Percutaneous coronary intervention

(PCI), Previous CABG I, Signs of CHF on presentation, Diagnostic catheterization, SBP, DBP, Heart Rate, Previous CHF, Creatinine, Troponin, Aspirin, ticagrelor, Beta blocker, ACE inhibitor/ARB, statin, anticoagulant and dyslipidemia were not. Table 5.

Table 5: Multivariate regression of all parameters for prediction of mortality, major GUSTO bleeding

Mortality	OR (95% CI)	p. value
Age (years)	0.854 (0.259 - 2.632)	0.102
Sex	0.653 (0.415 - 1748)	0.165
Smoking	1.532 (0.748 - 5.632)	0.318
HTN	1.674 (0.748 - 2.483)	0.196

DM	1.749 (0.574 - 1.749)	0.236
Previous MI	2.531 (0.574 - 5.631)	0.236
Previous PCI	1.785 (0.742 - 2.308)	0.179
Previous CABG I	1.630 (0.485 - 2.146)	0.147
Signs of CHF on presentation	0.542 (0.116 - 2.103)	0.231
Diagnostic catheterization	1.250 (0.544 - 2.306)	0.132
		0.132
SBP (mmHg) DBP (mmHg)	0.845 (0.528 - 2.635) 0.359 (0.187 - 1.639)	0.303
		0.293
Heart rate (beats/min)	0.742 (0.429 - 2.068)	0.219
Previous CHF	0.754 (0.496 - 2.749)	
Cardiac arrest	0.486 (0.265 - 0.763)	0.001*
Killip Class III-IV	0.657 (0.473 - 0.873)	0.001*
Baseline creatinine (mg/dl)	0.748 (0.563 - 2.416)	0.198
Baseline troponin (× ULN)	0.493 (0.236 - 2.419)	0.237
Aspirin	0.745 (.369 - 3.415)	0.297
ticagrelor	0.586 (0.296 - 2.637)	0.351
Beta blocker	0.743 (0.552 - 2.309)	0.294
ACE inhibitor/ARB	0.486 (0.192 - 2.634)	0.270
Statin	0.621 (0.287 - 2.856)	0.309
GP IIb-IIIa inhibitor	0.528 (0.175 - 0.865)	0.001*
Dyslipidemia	0.963 (0.476 - 2.254)	0.197
No. of diseased vessels	0.742 (0.361 - 0.958)	0.005*
GRACE score	0.595 (0.246 - 0.852)	0.001*
	Major GUSTO bleeding	
Age (years)	0.357 (0.148 - 0.742)	0.274
Sex	0.850 (0.519 - 3.509)	0.326
Smoking	1.559 (0.242 - 2.289)	0.348
HTN	2.654 (0.627 - 6.859)	0.217
DM	0.682 (0.273 - 3.701)	0.198
Previous MI	1.909 (0.686 - 5.314)	0.206
Previous PCI	1.614 (0.992 - 2.626)	0.168
Previous CABG I	0.599 (0.023 - 3.429)	0.149
Signs of CHF on presentation	0.452 (0.195 - 2.851)	0.140
Diagnostic catheterization	1.658 (0.368 - 3.954)	0.216
SBP (mmHg)	0.325 (0.185 - 1.638)	0.306
DBP (mmHg)	0.254 (0.033 - 2.263)	0.298
Heart rate (beats/min)	8.980 (0.626 - 22.240)	0.119
Previous CHF	0.652 (0.418 - 0.853)	0.024*
Cardiac arrest	0.745 (0.452 - 0.839)	0.001*
Killip Class III-IV	0.541 (0.391 - 0.872)	0.001*
Baseline creatinine (mg/dl)	0.415 (0.287 - 2.536)	0.136
Baseline troponin (× ULN)	0.854 (0.407 - 2.563)	0.236
Aspirin	0.598 (0.208 - 1.663)	0.314
ticagrelor	0.784 (0.563 - 2.108)	0.241
Beta blocker	0.695 (0.476 - 2.635)	0.365
ACE inhibitor/ARB	0.751 (0.665 - 2.119)	0.179
Statin	0.695 (0.475 - 2.145)	0.327
GP IIb-IIIa inhibitor	0.345 (0.152 - 0.569)	0.001*
Anticoagulant	2.551 (0.375 - 3.685)	0.419
Dyslipidemia	0.668 (0.412 - 3.526)	0.321
No. of diseased vessels	0.659 (0.415 - 0.875)	0.001*
GRACE score	0.475 (0.108 - 0.657)	0.001*
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*Significant as P value <0.05, HTN: Hypertension, DM: Diabetes mellitus, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, CHF: Congestive Heart Failure.

In Multivariate regression, cardiac arrest, Killip Class III-IV, GP IIbIIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Reinfarction and arrhythmia while Age, Sex, Smoking, HTN, DM, Previous MI, Previous PCI, Previous CABG I, Signs of CHF on

presentation, Diagnostic catheterization, SBP, DBP, Heart Rate, Previous CHF, Creatinine, Troponin, Aspirin, ticagrelor, Beta blocker ACE inhibitor/ARB, statin, anticoagulant and dyslipidemia were not. Table 6.

Table 6: Multivariate regression of all parameters for prediction of reinfarction, arrhythmia

	OR (95% CI)	P
Age (years)	0.528 (0.362 - 2.632)	0.102
Sex	0.425 (0.284 - 1748)	0.165
Smoking	1.632 (0.481 - 5.632)	0.318
HTN	2.631 (0.529 - 2.483)	0.196

DM	2.174 (0.631 - 1.749)	0.236
Previous MI	1.306 (0.784 - 5.631)	0.179
Previous PCI	1.921 (0.653 - 2.308)	0.147
Previous CABG I	1.852 (0.691 - 2.146)	0.231
Signs of CHF on presentation	0.623 (0.465 - 2.103)	0.231
Diagnostic catheterization	1.874 (0.649 - 2.306)	0.132
SBP (mmHg)	0.435 (0.846 - 2.635)	0.305
DBP (mmHg)	0.365 (0.221 - 1.639)	0.295
Heart rate (beats/min)	0.562 (0.346 - 2.068)	0.219
Previous CHF	0.685 (0.328 - 2.749)	0.395
Cardiac arrest	0.534 (0.151 - 0.763)	0.001*
Killip Class III-IV	0.593 (0.368 - 0.873)	0.001*
Baseline creatinine (mg/dl)	0.651 (0.259 - 2.416)	0.198
Baseline troponin (× ULN)	0.423 (0.119 - 2.419)	0.237
Aspirin	0.851 (0.992 - 3.415)	0.297
ticagrelor	0.362 (0.285 - 2.637)	0.351
Beta blocker	0.459 (0.196 - 2.309)	0.294
ACE inhibitor/ARB	0.405 (0.234 - 2.634)	0.270
Statin	0.526 (0.156 - 2.856)	0.309
GP IIb-IIIa inhibitor	0.657 (0.264 - 0.865)	0.001*
Anticoagulant	2.563 (0.846 - 2.567)	0.195
Dyslipidemia	0.743 (0.396 - 2.254)	0.197
No. of diseased vessels	0.559 (0.258 - 0.785)	0.012*
GRACE score	0.674 (0.335 - 0.847)	0.001*
GRACE Score	Arrhythmia	0.001
Age (years)	0.358 (0.174 - 2.105)	0.214
Sex	0.856 (0.569- 2.635)	0.265
Smoking	2.985 (0.586 - 5.429)	0.174
HTN	2.419 (0.946 - 3.749)	0.130
DM	1.259 (0.619 - 1.804)	0.179
Previous MI	3.526 (0.843 - 5.631)	0.153
Previous PCI	2.103 (0.648 - 2.308)	0.106
Previous CABG I	2.596 (0.693 - 2.559)	0.179
Signs of CHF on presentation	0.684 (0.253 - 1.764)	0.320
Diagnostic catheterization	1.876 (0.943 - 2.749)	0.297
SBP (mmHg)	0.589 (0.649 - 3.529)	0.248
DBP (mmHg)	0.641 (0.239 - 2.319)	0.150
Heart rate (beats/min)	0.489 (0.176 - 1.749)	0.136
Previous CHF	0.593 (0.268 - 0.829)	0.027*
Cardiac arrest	0.695 (0.153 - 0.845)	0.001*
Killip Class III-IV	0.783 (0.239 - 0.909)	0.001*
Baseline creatinine (mg/dl)	0.536 (0.143 - 1.749)	0.265
Baseline troponin (× ULN)	0.649 (0.319 - 3.429)	0.316
Aspirin	0.593 (0.109 - 2.068)	0.369
ticagrelor	0.637 (0.796 - 4.159)	0.415
Beta blocker	0.712 (0.469 - 3.526)	0.405
ACE inhibitor/ARB	0.598 (0.367 - 3.649)	0.362
Statin	0.689 (0.229 - 0.849)	0.024*
GP IIb-IIIa inhibitor	0.693 (0.476- 0.951)	0.001*
Anticoagulant	1.762 (0.529 - 5.749)	0.274
Dyslipidemia	0.875 (0.641 - 3.527)	0.369
No. of diseased vessels	0.649 (0.246 - 0.864)	0.012*
GRACE score	0.517 (0.239 - 0.843)	0.001*

*Significant as P value <0.05, HTN: Hypertension, DM: Diabetes mellitus, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, CHF: Congestive Heart Failure.

In Multivariate regression, Cardiac arrest, Killip Class III-IV, GP IIb IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Heart failure and while Age, Sex, Smoking, HTN, DM, Previous MI, Previous PCI, Previous CABG I, Signs of CHF on presentation, Diagnostic catheterization, SBP, DBP, Heart Rate, Previous CHF, Creatinine, Troponin, Aspirin, ticagrelor, Beta blocker, ACE inhibitor/ARB, Statin, anticoagulant and dyslipidemia were not. In Multivariate regression, previous CABG I, Diagnostic

catheterization, Cardiac arrest, Killip Class III-IV, GP IIb-IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Stroke while Age, Sex Smoking, HTN, DM, Previous MI, Previous PCI, Signs of CHF on presentation, SBP, DBP, Heart Rate, Previous CHF, Creatinine, Troponin, Aspirin, ticagrelor, Beta blocker, ACE inhibitor/ARB, statin, anticoagulant and dyslipidemia were not. Table 7.

Table 7: Multivariate regression of all parameters for prediction of heart failure and stroke

	OR (95% CI)	P
Age (years)	0.419 (0.118 - 2.415)	0.250
Sex	0.326 (0.215 - 1.521)	0.197
Smoking	1.647 (0.953 - 4.251)	0.109
HTN	1.031 (0.415 - 3.627)	0.098
DM	2.164 (0.458 - 1.658)	0.134
Previous MI	1.475 (0.648 - 6.385)	0.226
Previous PCI	2.514 (0.989 - 4.523)	0.213
Previous CABG I	1.698 (0.587 - 2.631)	0.306
Signs of CHF on presentation	0.649 (0.254 - 2.583)	0.319
Diagnostic catheterization	1.649 (0.689 - 3.685)	0.246
SBP (mmHg)	0.648 (0.385 - 3.451)	0.267
DBP (mmHg)	0.553 (0.214 - 2.531)	0.179
Heart rate (beats/min)	0.684 (0.221 - 1.548)	0.193
Previous CHF	0.638 (0.158 - 3.985)	0.126
Cardiac arrest	0.564 (0.145 - 0.865)	0.001*
Killip Class III-IV	0.582 (0.351 - 0.743)	0.001*
Baseline creatinine (mg/dl)	0.658 (0.245 - 3.529)	0.136
Baseline troponin (× ULN)	0.549 (0.107 - 2.795)	0.234
Aspirin	0.605 (0.548 - 2.513)	0.271
ticagrelor	0.645 (0.148 - 1.589)	0.206
Beta blocker	0.648 (0.416 - 1.645)	0.332
ACE inhibitor/ARB	0.543 (0.169 - 1.603)	0.219
Statin	0.621 (0.287 - 2.856)	0.206
GP IIb-IIIa inhibitor	0.496 (0.226 - 0.7.526)	0.001*
Anticoagulant	2.621 (0.574 - 1.549)	0.369
Dyslipidemia	0.658 (0.362 - 1.527)	0.227
No. of diseased vessels	0.548 (0.267 - 0.756)	0.015*
GRACE score	0.389 (0.207 - 0.659)	0.001*
ORIGE Scote	Stroke	0.001
Age (years)	0.542 (0.150 - 2.261)	0.091
Sex	0.439 (0.245 - 2.306)	0.351
Smoking	1.854 (0.541 - 4.516)	0.216
HTN	1.621 (0.415 - 3.619)	0.258
DM	1.556 (0.527 - 3.496)	0.416
Previous MI	2.649 (0.859 - 4.753)	0.369
Previous PCI	2.451 (0.632 - 7.414)	0.276
Previous CABG I	2.369 (1.459 - 3.517)	0.026*
Signs of CHF on presentation	0.512 (0.245 - 2.653)	0.109
Diagnostic catheterization	2.556 (1.715 - 3.516)	0.037*
SBP (mmHg)	0.430 (0.234 - 2.416)	0.226
DBP (mmHg)	0.467 (0.220 - 1.803)	0.320
Heart rate (beats/min)	0.659 (0.361 - 1.647)	0.349
Previous CHF	0.548 (0.352 - 2.128)	0.276
Cardiac arrest	0.529 (0.361 - 0.843)	0.001*
Killip Class III-IV	0.559 (0.301 - 0.643)	0.001*
Baseline creatinine (mg/dl)	0.539 (0.519 - 0932)	0.261
Baseline troponin (× ULN)	0.549 (0.412 - 3.615) 0.615 (0.194 - 2.521)	0.261
	0.613 (0.194 - 2.321)	0.243
Aspirin ticagrelor	0.736 (0.319 - 3.416) 0.525 (0.129 - 2.012)	0.243
Beta blocker	0.323 (0.129 - 2.012)	0.206
ACE inhibitor/ARB	0.416 (0.631 - 1.534)	0.176
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Statin CD IIIs IIIs inhibitor	0.556 (0.137 - 2.412)	0.227
GP IIb-IIIa inhibitor	0.551 (0.264 - 0.785)	0.001*
Anticoagulant	1.652 (0.524 - 2.651)	0.249
Dyslipidemia	0.643 (0.315 - 2.102)	0.230
No. of diseased vessels	0.516 (0.294 - 0.753)	0.031*
GRACE score	0.754 (0.650 - 0.934)	0.001*

^{*}Significant as P value <0.05, HTN: Hypertension, DM: Diabetes mellitus, MI: Myocardial infarction, PCI: Percutaneous coronary intervention, CHF: Congestive Heart Failure.

Discussion

The most extensively used and accessible diagnostic method for people suspected of having ACS is the electrocardiogram (ECG) ^[8]. Admission Particularly for patients with non-ST-segment elevation myocardial infarction (NSTEMI), a diagnosis used to describe a diverse group of individuals with varying clinical features and outcomes, ECG results may potentially offer prognostic information and quick risk assessment in ACS patients ^[9].

According to our findings, there was a statistically significant difference between the four groups in terms of the number of

diseased arteries, stenotic vessels, stenosis grading, and stenosis location. While there was no significant difference between the TSTE and NIC groups or between the ST-Segment Depression and TWI groups, the number of diseased vessels was considerably higher in the ST-Segment Depression and TWI groups than in the TSTE and NIC groups. In terms of the number of diseased vessels, stenotic vessels, stenosis grading, and stenosis location, our findings show a statistically significant difference between the four groups. While the number of diseased vessels was not statistically different between the TSTE and NIC groups or between the ST-Segment Depression and TWI groups, it was

considerably greater in the ST-Segment Depression and TWI groups than in the TSTE and NIC groups.

These results are in consistent with Patel *et al.* [10] who came to the conclusion that individuals with ST-segment depression generally had coronary artery bypass grafting and had greater left main, proximal left anterior descending, and 3-vessel coronary artery disease, according to the angiography.

On the other hand, Tan *et al.* [11] were on different sides, as they demonstrated that, among the 2,708 patients with accessible angiographic data, the rates of left main disease or 3-vessel disease were comparable between TWI and non-TTI/STD individuals.

According to our research, the four groups did not differ significantly in terms of stroke, major gusto haemorrhage, or reinfarction. However, among the four groups in-hospital, there were statistically significant differences in heart failure, arrhythmia, and all-cause mortality. Hospital outcomes (all-cause mortality and heart failure) were insignificantly different between the TSTE and NIC groups, but significantly higher in the ST-Segment Depression and TWI groups than in the TSTE group, and higher in the ST-Segment Depression group than in the TWI and NIC groups (P value<0.05).

These results are in agreement with Chen *et al.* [12] who found that all-cause mortality was statistically different between groups, indicating that the incidence of hospital mortality was highest in the STD group, followed by the NIC and TSTE groups, and lowest in the TWI group. They contradicted our findings by concluding that stroke, major gusto bleeding, and reinfarction were indifferent between four groups in-hospital. NIC vs. TWI and STD vs. TWI were the only two of these differences that were statistically significant. Patients with STD had significantly higher hospital mortality rates and major adverse events (MAE) than those in the TWI group, whereas patients with NIC had significantly higher hospital mortality rates as well.

However, Patel *et al.* [10] demonstrated how the NIC group had the highest history of myocardial infarction, congestive heart failure, and percutaneous coronary intervention, followed by the ST-segment depression, TWI, and transient ST-segment elevation groups.

According to our findings, there was no discernible difference between the four groups in terms of stroke, major haemorrhage, and reinfarction. However, after 30 days, there were statistically significant differences between the four groups in heart failure, all-cause mortality, and arrhythmia. While there was no significant difference between the TSTE group and the TWI group and the NIC group, or between the TWI group and the NIC group, the 30-day outcomes (arrhythmia) were considerably greater in the ST-Segment Depression group than in the TSTE, TWI, and NIC groups (P < 0.05).

On the same line of our results, Chen *et al.* ^[13] supported what we found as they stated that stroke, major gusto bleeding and reinfarction were indifferent between four groups after 30-days. However, Chen *et al.* ^[13] disagreed with us since they discovered that the STD group had the highest incidence of death and MAE in the hospital or after 30 days of follow-up, followed by the NIC and TSTE groups, and the TWI group had the lowest incidence. Only the STD vs. TWI and NIC vs. TWI differences, however, were statistically significant. NIC patients had considerably higher in-hospital mortality rates and MAE, while STD patients had significantly higher 30-

day and in-hospital mortality rates and MAE when compared to the TWI group.

In multivariate regression, Cardiac arrest, Killip Class III-IV, GP IIb IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Mortality. Rigueira *et al.* [14] confirmed our findings, indicating that the length of cardiac arrest was an independent predictor of death (OR 1.015, 95%CI 1.0 - 1.05; p = 0.048). Further, Kumar *et al.* [15] determined what we discovered because they demonstrated that, according to multivariable analysis, GRACE score ≥150 and Killip class II-IV at presentation were independent predictors of death following six months of NSTE-ACS, with adjusted ORs of 8.43 [3.33-21.38] and 32.93 [2.65-408.8] respectively. Moreover, Siddiqui *et al.* [16] found that individuals with left main disease, multi-vessel disease, and one vessel disease had greater mortality rates.

In Multivariate regression, previous CHF, Cardiac arrest, Killip Class III-IV, GP IIb-IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Major GUSTO bleeding. These observations are in consistent with Loh *et al.* [17] who showed that previous CHF and GP IIb-IIIa inhibitor were associated Major GUSTO bleeding in NSTEMI patients. Also, Segev *et al.* [18] found matched results as they found that patients with in-hospital major bleeding were associated with Killip class > or = II. Added to that, Park *et al.* [19] agreed to ours as they concluded that multiple vessels diseases were MI type- or time-dependent predictors.

Heart arrest, Killip Class III-IV, GP IIb IIIa inhibitor, number of diseased vessels, and GRACE score were all independent predictors of reinfarction in multivariate regression. Additionally, the number of sick arteries, statin, GP IIb-IIIa inhibitor, Killip Class III-IV, prior CHF, cardiac arrest, and GRACE score were independent predictors of arrhythmia. These findings match with Chu *et al.* [20] who observed that

in NSTEMI patients, the predictors of occurrence of arrhythmia was related to severity symptomatic cardiac dysfunction, Arrhythmia significantly increased adverse events.

In Multivariate regression, Cardiac arrest, Killip Class III-IV, GP IIb IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Heart failure. Wu *et al.* [21] were in agreement to what we found as they stated that Killip Class III-IV were independent predictors of Heart failure. Alongside with our results, Chen *et al.* [13] assured our outcomes as they found that GRACE score was independent predictors of Heart failure in NSTEMI patients. Above that, Velagaleti and Vasan al. [22] supported our findings as they showed that number of diseased vessels associated with increased heart failure in NSTEMI.

In Multivariate regression, previous CABG I, Diagnostic catheterization, Cardiac arrest, Killip Class III-IV, GP IIb-IIIa inhibitor, No. of diseased vessels and GRACE score were independent predictors of Stroke. Magedanz *et al.* ^[23] were on our side of results as they revealed that previous CABG increase risk of stroke. Moreover, Setogawa *et al.* ^[24] found the same as they concluded that previous diagnostic catheterization was risk factor for development of stroke. Also, Hurskainen *et al.* ^[25] Killip Class III-IV, previous cardiac arrest was associated with increased risk of stroke in NSTEMI patients.

The study's single centre design and somewhat small sample size were among its limitations, which could have led to different results than those obtained elsewhere.

Conclusions

TWI was linked to benign short-term prognoses, whereas the presence of STD on admission ECG was linked to negative outcomes. One should not interpret the absence of ischaemic ECG abnormalities as a sign of low risk. For short-term outcomes, TSTE showed no predictive value. Therefore, NSTMI patients can be quickly risk-stratified at presentation using qualitative analysis of the admission ECG. Yet, it might not offer any indication of how NSTEMI patients will do in the short-term following invasive treatment. Cardiac arrest, Killip Class III-IV, GP IIb IIIa inhibitor, number of diseased vessels, and GRACE score were all independent predictors of mortality in multivariate regression.

Financial support and sponsorship: Nil Conflict of Interest: Nil

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How to Cite This Article

El Sawy NM, Shoeib OM, Draz EE, Elsetiha ME. Role of electrocardiographic findings in the prognosis of Non-ST-Segment elevation myocardial infarction at admission. International Journal of Cardiology Sciences. 2025;7(2):01-10

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