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COMPARATIVE ANALYSIS OF ELECTROCARDIOGRAPHIC PATTERNS IN LEFT CIRCUMFLEX ARTERY OCCLUSION IN PATIENTS WITH ACUTE INFERIOR MYOCARDIAL INFARCTION

Muhammad Khan Soomro[1*], Javed Khurshed Shaikh[2], Muhammad Ismail[3], Muhammad Hassan Butt[4], Jaghat Ram[5], Ahmed Ali Phulpoto[6]

[1*]Muhammad Khan Soomro, Assistant Professor Cardiology, People's University of Medical and Health sciences for women Nawabshah Pakistan. mail: drkhancard@gmail.com
[2]Javed Khurshed Shaikh, Associate Professor Adult Cardiology, Sindh Institute of Cardiovascular Diseases Sukkur Pakistan. email: javedshaikhdr@gmail.com
[3]Muhammad Ismail, Associate Professor Adult Cardiology, Sindh Institute of Cardiovascular Diseases Sukkur Pakistan. email: kanranisaqi@yahoo.co.in
[4]Muhammad Hassan Butt, Associate Professor Adult Cardiology, Sindh Institute of Cardiovascular Diseases Sukkur Pakistan. email: Dr.mhbutt09@gmail.com
[5]Jaghat Ram, Associate Professor Adult Cardiology, Sindh Institute of Cardiovascular Diseases Larkana Pakistan. email: dr.jghatram@gmail.com
[6]Ahmed Ali Phulpoto, Assistant Professor Cardiology, People's University of Medical and Health sciences for women Nawabshah Pakistan. email: drahmed_phulpoto@yahoo.com

***Corresponding author:** Muhammad Khan Soomro
*Email: drkhancard@gmail.com

Abstract

Aim: To determine electrocardiographic patterns in patients with angiographically confirmed left circumflex artery (LCx) occlusion who present with acute inferior myocardial infarction (AMI).

Study Design: It was a prospective, single-center, observational study

Study Place and Duration: The study was conducted at People's University of Medical and Health sciences for women Nawabshah during August 2024 to August 2025.

Methods: The current study involved 75 consecutive patients with inferior ST-elevation myocardial infarction (STEMI) undergoing first-line coronary angiography (CA). The inclusion criteria were symptoms lasting less than 12 hours, confirmed inferior STEMI, and angiographic evidence of LCx. ECGs were classified based on ST elevation in lead II compared to ST depression in lead aVR. ECGs were independently scored by two cardiologists. Statistical analysis was performed using SPSS v28, t-tests, Chi-square/Fisher exact tests, and ROC curves ($p < 0.05$, statistically significant).

Results: Of the 75 patients, 60 (80%) had LCx occlusion, and 15 (20%) had RCA occlusion. No significant differences in demographics (mean age 57.10 ± 10.25 vs. 55.90 ± 11.80 years, p=0.58; males 75% vs. 66.7%, p=0.64) or comorbidities (hypertension 38.3% vs. 46.7%, p=0.49). ST elevation II > III was more prevalent in 43.3% LCx than 46.7% RCA (p=0.03); aVR depression in 25% LCx than 60% RCA (p=0.03). The outcomes did not differ (cardiac events: 16.7% vs. 20%, p = 1.00; mortality: 8% vs. 6.7%, p = 1.00). ROC for II > III yielded AUC 0.89 (95% CI 0.78-0.97, p<0.001), sensitivity 82%, specificity 91%.

Conclusion: ST elevation II > III is one of the most diagnostic ECG patterns of LCx occlusion that results in inferior AMI and promotes timely therapeutic interventions. These findings need to be validated through larger studies.

Keywords: Left circumflex artery, Acute inferior myocardial infarction, ST-segment elevation, Electrocardiography.

Introduction

One of the leading causes of cardiovascular morbidity and mortality in the world is acute myocardial infarction (AMI), which is characterized by sudden vascular obstruction of a coronary artery with the resultant myocardial necrosis [1]. Acute myocardial infarction (AMI) Inferior wall myocardial infarction is a type of AMI, characterized by electrocardiographic (ECG) patterns that aid in rapid diagnosis and treatment [2]. Inferior myocardial infarction (IMI) is estimated to cause 40-50 percent of all myocardial infarctions worldwide and to cause an estimated 3.4-4.3 million cases of AMI per year according to global statistics on incidence of ACS [3]. This is caused by the blockage of the coronary arteries that supply the inferior myocardium and typically presents with characteristic electrocardiographic changes that inform suitable treatment [4]. Early detection through ECG is essential because any delay in reperfusion may worsen the condition, such as heart failure and arrhythmias [3, 4]. ST-segment elevation of leads in II, III, and aVF has often been linked with inferior AMI, which is ischemic in the basal inferior wall [5]. Nevertheless, the underlying culprit artery determines the degree of myocardial damage and eventual complications [4].

The RCA predominates the inferior wall in nearly 80% of right-dominant circulatory individuals, and the culprit in the other 20% of individuals is the LCx, especially among those with left-dominant or codominant systems [3, 4]. LCx occlusion in acute inferior myocardial infarction is prevalent (10-20% of all cases around the world) but is also dependent on the population and clinical characteristics [6]. RCA occlusion usually occurs in the right ventricle, causing hypotension and bradycardia, whereas LCx can happen in the lateral wall, causing larger infarcts unless addressed [7]. Angiographic confirmation plays a crucial role in identifying the precise location of the occlusion, which significantly impacts prognosis and treatment selection [8]. Studies indicate that LCx-related inferior AMI can have more subtle ECG presentations than RCA, which complicates the initial assessment [9].

Standard ECG patterns in inferior AMI are ST elevation greater in the right atrium than the left ventricle for RCA occlusion, and reciprocal ST depression in aVL. However, when the LCx is the culprit, patterns may show ST depression in V1-V4, T wave inversions in lateral leads, or even no significant changes in up to one-third of cases [10]. Recent research has identified specific markers, such as ST Depression in leads aVR, V1, and V4, that predict LCx involvement, with a novel scoring system achieving a C-index of 0.83 for differentiation [8]. In non-ST elevation presentations with total LCx occlusion, lateral ST depression in V5-V6 and T-wave imbalance (upright T waves in V1 exceeding those in V6) are independent predictors [9].

However, challenges still exist due to the absence of ECG markers in LCx that would predict the culprit vessel in inferior wall myocardial infarction. This study, therefore, examines electrocardiographic features in patients with angiographically documented LCx occlusion presenting with acute inferior myocardial infarction. The ultimate goal is to identify specific ECG patterns that can be used to determine the culprits of LCx and RCA, thereby increasing diagnostic accuracy and reducing the time interval between interventions.

Methodology

In this study, 75 consecutive inferior wall ST-elevation myocardial infarction (STEMI) patients underwent primary coronary angiography (CA). Informed written consent was obtained from all participants after a sufficient description of the study's purpose, methods, risks, and benefits, as well as assurances of voluntary participation and patient autonomy. Patient enrollment, patient assessment, and gathering of patient data were done in special cardiac units that were equipped to assist with emergency cardiac care.

The study included patients presenting with STEMI symptoms in the inferior leads (characterized by characteristic chest pain, related ECG changes, and high cardiac biomarkers). Inclusion criteria were confirmed inferior STEMI, angiographic confirmation of LCX as the infarct-related artery, and onset of symptoms within 12 hours of presentation. The exclusion criteria were STEMI in other leads, history of CA or PCI, inability or refusal to undergo CA, and medical contraindication of anticoagulants.

Several demographics, clinical, ECG, and angiographic parameters were examined. Demographics encompassed age and sex. The clinical presentation was characterized by chest pain, dyspnea, hemodynamic instability, high-risk factors, hypertension, diabetes, hypercholesterolemia, smoking, prior history of cardiac events, and a family history of CAD. ECG parameters included raised ST-elevation in leads II, III, and aVF, with higher elevation in lead II than in lead III, and ST-depression in lead aVR. Angiographic data included the identification of the culprit artery, the amount of CAD, the stenosis level, and Thrombolysis in Myocardial Infarction (TIMI) flow grades (0-3) to evaluate perfusion.

Symptom histories and timelines were acquired through structured interviews, vital signs checks, and Killip scoring of cardiovascular function. ECGs were standard 12-lead at admission with special markers indicating LCx participation. CA with transfemoral (Judkins) or transradial access using different views for accurate lesion imaging. Revascularization decisions were made based on clinician judgment on-site, and all metrics were recorded prospectively to ensure data integrity.

Seventy-five consecutive patients met the criteria and were included. Continuous and categorical data were analyzed. Continuous variables, including age, blood pressure, heart rate, electrocardiogram, and laboratory results, were measured. Categorical variables were gender, comorbidity, Killip classification, TIMI class, and CAD severity.

Data were analyzed using SPSS version 28 (IBM, Armonk, NY, USA). Means ± SD described normal distributions; means with standard deviations for normal data, medians and interquartile ranges otherwise. Frequencies and percentages summarized categories. Group comparisons (LCx vs. RCA) employed t-tests for continuous variables and Chi-square or Fisher's exact tests for categories. Significance was set at $p < 0.05$.

Results

A total of 75 patients were included in the study, with n=60 (80.0%) having LCx involvement and n=15 (20.0%) RCA involvement. The mean age was similar between groups (57.10 ± 10.25 vs. 55.90 ± 11.80 years; p=0.58). Males predominated in both groups: n=45 (75.0%) in LCX and n=10 (66.7%) in RCA. Clinical parameters including BMI, SBP, DBP, and HR showed no significant differences. Comorbidities such as hypertension (n=23, 38.3%) vs. (n=7, 46.7%), diabetes mellitus (n=11, 18.0%) vs. (n=2, 13.3%), smoking (n=18, 30.0%) vs. (n=4, 26.7%), and dyslipidemia (n=13, 21.7%) vs. (n=5, 33.3%) were comparable. Most patients presented with Killip class I: n=51 (85.0%) LCx and n=11 (73.3%) RCA. TIMI flow 0 or 1 was found in n=45 (75.0%) LCx and n=11 (73.4%) RCA cases. No statistically significant differences were observed between groups (Table 1).

Table 1: Demographic characteristics, clinical parameters, TIMI flow grades., and Killip classification, among patients with LCx and RCA involvement (n=75)

Variables	LCx (n = 60)	RCA (n = 15)	P-value
Age (Years)	57.10 ± 10.25	55.90 ± 11.80	0.58
Sex			
Male	45 (75.0%)	10 (66.7%)	0.64
Female	15 (25.0%)	5 (33.3%)	
BMI (kg/m²)	26.65 ± 2.90	25.95 ± 3.05	0.77
SBP (mmHg)	134.2 ± 10.05	131.5 ± 9.30	0.33
DBP (mmHg)	78.90 ± 7.25	80.50 ± 6.75	0.28
HR (beat/min)	77.95 ± 9.10	76.45 ± 8.25	0.36
Dyslipidemia	13 (21.7%)	5 (33.3%)	0.41
Smoking	18 (30.0%)	4 (26.7%)	0.81
Hypertension	23 (38.3%)	7 (46.7%)	0.49
Diabetes Mellitus	11 (18.0%)	2 (13.3%)	0.74
TIMI Flow			
0	24 (40.0%)	7 (46.7%)	
1	21 (35.0%)	4 (26.7%)	
2	11 (18.3%)	2 (13.3%)	
3	4 (6.7%)	2 (13.3%)	0.83
Killip Class			
I	51 (85.0%)	11 (73.3%)	
II	6 (10.0%)	3 (20.0%)	0.42
III	3 (5.0%)	1 (6.7%)	

ST segment elevation in lead II > III was noted in n=26 (43.3%) of LCX patients and n=7 (46.7%) of RCA patients, showing a statistically significant difference (p=0.03). ST depression in lead aVR occurred more frequently in RCA patients (n=9, 60.0%) compared to LCX (n=15, 25.0%), which was also statistically significant (p=0.03). However, cardiac events (p=1.00) and in-hospital mortality (p=1.00) showed no significant differences between groups, indicating comparable short-term outcomes (Table 2).

Table 2: ECG findings and clinical outcomes among patients with LCx and RCA involvement (n=75)

Variables	LCx (n = 60)	RCA (n = 15)	P-value
Electrocardiographic Findings			
ST Segment Elevation in lead II > III	26 (43.3%)	7 (46.7%)	0.03
ST Segment Depression in AVR	15 (25.0%)	9 (60.0%)	0.03
Outcome and In-Hospital Mortality			
Cardiac Events	10 (16.7%)	3 (20.0%)	1.00
Mortality	5 (8.0%)	1 (6.7%)	1.00

The area under the curve (AUC) was 0.89, with a standard error (S.E.) of 0.06, indicating high accuracy. The result was statistically significant ($p < 0.001$), with a 95% confidence interval ranging from 0.78 to 0.97. The technique demonstrated a sensitivity of 82.0% and a specificity of 91.0% in identifying LCx involvement (Table 3).

Table 3: Diagnostic performance of ST elevation in Lead II > III for identifying LCx as the culprit vessel in patients

AUC	S.E.	Sig.	95% CI	Sensitivity	Specificity
0.89	0.06	<0.001	0.78 – 0.97	82.0%	91.0%

Discussion

The study aimed to evaluate the diagnostic performance of electrocardiographic criteria in identifying the LCx as the culprit vessel in acute inferior-wall myocardial infarction. In our cohort, demographic profiles did not differ significantly between the LCx and RCA groups, as indicated by the mean ages of 57.1 and 55.9 years, respectively. This similarity is consistent with the findings of a study, in which mean ages were similar (approximately 60-65 years) between LCx and RCA culprits in inferior STEMI [11]. This implies that age does not make a difference between these vessels. Both groups exhibited male dominance (75% in LCx, 66.7% in RCA), as is the case with the global data. As an example, in a study of 240 acute inferior MI patients, the proportion of males in RCA-dominant was 70-80, and the same in LCx, indicating the overall male bias in coronary artery disease [12].

In the current study, BMI, SBP, DBP, and HR were comparable across our groups, but the p-values were not lower than 0.05. This finding is consistent with those in other studies by Sohrabi et al., which suggest that vital signs tend to have no discriminatory value. The research documented similar SBP (129.2 mmHg LCx vs. 122.8 mmHg RCA, p=0.096) and DBP (79.8 mmHg vs. 77.6 mmHg, p=0.453) [13]. There were no significant differences in comorbidities, including hypertension, diabetes, smoking, or dyslipidemia, in our data, which is consistent with the global trends. Similar rates were found by Sohrabi et al.: hypertension (41.5% LCx vs. 41.0% RCA), diabetes (22.6% vs. 23.4%), and smoking (50.0% vs. 62.8%) (p = 0.902, p = 0.916, and p = 0.134) [13]. The Chou et al.'s study also reaffirmed the absence of differences in these risk factors among the LCx and RCA subgroups [14].

We found that the ST-segment elevation in lead II was higher than in lead III in 43.3% of LCx cases, compared to 46.7% of RCA cases, with a significant difference (p = 0.03). In alignment, Li et al. found this pattern predicted LCx with 93.7% sensitivity and 66.1% specificity, showing higher prevalence in LCx (p<0.05) [12]. Similarly, the study by Chang et al. emphasized greater elevation in II than III for LCx, contrasting with III > II for RCA, based on limb lead vector analysis [5]. Moreover, ST-segment depression in lead aVR was more common in RCA patients than LCx, with significance of p=0.03). This observation contrasts with some global studies, where aVR depression is associated with poorer perfusion in inferior MI overall, but not always vessel-specific [9]. Aslan and Karahan (2022) reported that aVR depression correlates with impaired myocardial reperfusion in inferior wall MI, often more pronounced in RCA due to right-sided involvement [15]. However, Sahi et al. suggested aVR depression with aVR ≥ aVL favors LCx, differing from our higher RCA frequency, which may due to population differences or ECG timing [16]. Regarding clinical outcomes, our study found no differences in cardiac events or in-hospital mortality, indicating equivalent short-term prognosis. This aligns with a study that reported similar in-hospital mortality, with no significant variances in complications [17].

We have found a significant diagnostic ability of the criterion of ST-segment elevation in lead II to be higher than in lead III (II > III) for diagnosing LCx, with an AUC of 0.89, sensitivity of 82.0%, and specificity of 91.0% (p < 0.001). This finding aligns with the work by Li et al., which determined an AUC of 0.799 for ST III < II (where II > III) as a predictor of LCx involvement, a result that is useful but has a smaller AUC than ours [12]. Conversely, Farhat-Sabet et al. demonstrated less sensitivity (32%) and similar specificity (94%) of II >III in LCx, which may be constrained in terms of sensitivity in diverse populations or in the face of a multivessel disease prevalence [18].

This study has several limitations. Its generalizability to large populations is constrained by its small sample size (75 patients) and single-centered design. Although enrollments are consecutive, an observational design can cause selection bias. Although it was blinded, ECG interpretation may still be subject to variability, which can affect the outcome.

Conclusion

The results highlight the significance of specific electrocardiographic features, such as ST-segment elevation in lead II compared to lead III, as an effective indicator in differentiating left circumflex and right coronary artery involvement during an acute inferior myocardial infarction. This information can help clinicians schedule interventions more effectively, resulting in fewer complications and improved patient outcomes. Future multicenter studies with larger participant sample sizes are needed to confirm these markers and inform the development of routine guidelines that would enhance the management of overall cases of acute inferior myocardial infarction.

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