

ORIGINAL ARTICLE

The Value of Lead aVR ST Segment Changes in Localizing Culprit Lesion in Acute Inferior Myocardial Infarction and Its Prognostic Impact

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Background: Identifying infarct-related artery (IRA) in patients with inferior ST elevation myocardial infarction (STEMI) has prognostic and therapeutic benefits.

Objectives: To differentiate IRA and the location of culprit lesion in inferior STEMI, using ST segment changes in lead aVR.

Methods: ST segment changes in lead aVR were recorded in 150 patients, admitted with first inferior STEMI. The association of IRA and the location of culprit lesion with ST segment changes in aVR were investigated.

Results: ST elevation ≥ 0.5 mm in lead aVR was present in 17 patients (11.3%), ST depression ≥ 0.5 mm in 74 patients (49.3%) and 59 patients (39.3%) did not have significant ST segment changes. Right coronary artery (RCA) was the IRA in 117 patients (78%) and left circumflex artery (LCX) in 33 patients (22%). Prevalence of RCA involvement as the IRA was different in three study groups (94.1% in ST elevation group, 83.1% in isoelectric group and 70.3% in ST depression group, $P = 0.049$). Presence of ST elevation had a sensitivity and specificity of 13.68 % and 96.97%, for detecting RCA lesions, respectively. ST depression had 66.67% sensitivity and 55.56% specificity for identifying LCX lesions. Clinical complications were low in our study with no significant difference among patients of three groups.

Conclusions: Presence of ST elevation is highly suggestive of RCA lesions versus LCX lesions, whereas absence of ST elevation cannot rule out RCA lesions. Presence of ST depression has a moderate sensitivity and specificity for LCX lesions.

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Identifying infarct-related artery (IRA), in patients with ST segment elevation myocardial infarction (STEMI), provides valuable prognostic information and guides therapeutic decision-making in an emergency setting.¹ Occlusion of either right coronary artery (RCA) or left circumflex artery (LCX) can lead to inferior STEMI and the preferred treatment, risk stratification and prognosis of patients may vary based on the site of the culprit lesion.² Although some electrocardiographic criteria are explained for rapid identification of IRA in patients

with inferior STEMI, differentiating the occluded vessel and proximity of lesion in the vessel can be challenging in clinical setting.^{3,4}

Electrocardiographic changes in lead aVR, which is not routinely evaluated in clinical practice, are recently explained as a source of additional information for determining culprit lesion and prognosis of patients with STEMI.^{5–8} Although, ST elevation in lead aVR is commonly associated with anterior STEMI,^{5,9} some recent studies proposed different patterns of ST segment changes of lead

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aVR in inferior STEMI, which may be useful in predicting IRA and possibly prognosis of the patients.¹⁰⁻¹³ However, data regarding both diagnostic and prognostic value of ST segment changes in this lead for inferior STEMI are not always consistent.¹¹⁻¹⁴ In addition, most studies have only investigated the role of ST changes of lead aVR in differentiating RCA from LCX infarctions with no extra information about the impact of the site of occlusion in either RCA or LCX.^{11, 15, 16}

Considering these facts, we conducted this study in patients with inferior STEMI to determine the association of ST segment changes in lead aVR with both IRA and the proximity of the culprit lesion in the occluded vessel. We further, investigated the impact of ST segment changes in lead aVR on clinical complications and mortality of patients in hospitalization and follow-up period.

MATERIALS AND METHODS

In this prospective cohort study, from March 2013 through December 2014, one hundred fifty consecutive patients, admitted to our cardiac care unit at Madani cardiovascular heart center with their first inferior STEMI, were enrolled. Patients who had electrocardiographic evidence of bundle branch blocks, ventricular hypertrophy or ventricular arrhythmias, history of myocardial infarction, coronary artery bypass grafting or pacemaker implantation and those who presented to hospital more than 12 hours after onset of cardiac chest pain were excluded from this study.

A cardiologist manually measured the magnitude of ST segment elevation or depression in lead aVR on first admission electrocardiogram (ECG). Comprehensive demographic, clinical and biochemical data as well as results of two-dimensional echocardiography performed on first day of admission were recorded for each patient. The Information about the occurrence of ventricular fibrillation (VF), ventricular tachycardia (VT), nodal or bundle branch blocks, other arrhythmias and acute heart failure was obtained for all study sample during hospitalization period. Based on diagnostic or therapeutic angiographic data, infarct related artery, site of culprit lesion, involvement of other coronary arteries and dominant coronary artery were recorded for each patient. To determine follow-up period mortality, patients were contacted by phone call six months after the index

hospital discharge. Mortality rate of patients during hospital stay and follow-up period was recorded.

Significant ST elevation (STE) was defined as ST elevation of 0.5 millimeters or more in lead aVR and ST depression (STD) was defined as ST depression of 0.5 millimeters (mm) or more in lead aVR. Based on ST segment changes in lead aVR, patients were allocated into three groups as follows: (1) STE (+) group; (2) STD (+) group; and (3) Isoelectric group. Three groups were compared regarding demographic, echocardiographic, electrocardiographic, and angiographic findings. In further analysis, patients were grouped based on presence or absence ST elevation and once again based on presence or absence of ST depression in lead aVR. Infarct related artery and site of culprit lesion were compared between groups in both settings.

Inferior STEMI was defined as the presence of typical chest pain lasting more than 20 minutes, with new ST elevation at the J point with the cutoff point of ≥ 0.1 mV in at least two contiguous inferior leads (II, III, and aVF) on the admission ECG as well as an increase in cardiac enzymes, which was defined as an increase of one point above the 99th percentile cutoff point for MB isoenzyme of creatine kinase (CK-MB) and cardiac-troponin I (cTNI).¹⁷ Standard 12-lead ECG was recorded on admission using a paper speed of 25 mm/s and standardization of 1mV/10mm. Based on standard therapeutic guidelines patients either underwent primary PCI or received thrombolytic therapy and a delayed angiography was scheduled for the following days during the same hospitalization. A reduction of at least 50% of the luminal diameter of coronary artery was considered as significant stenosis. The site of culprit lesion in RCA was described related to the first major RV branch as proximal versus nonproximal lesion.

Institutional review board committee at Tabriz university of medical sciences reviewed and approved the design of the study. The study was exempted from informed consent due to its descriptive design. Nevertheless, complete patient privacy was maintained during all study stages.

Statistical Analysis

Data were analyzed with statistical software SPSS (SPSS Inc. Released 2009. PASW Statistics for Windows, Version 18.0. Chicago, IL, USA). Continuous variables were presented as the mean \pm standard deviation and categorical variables were

Table 1. Comparison of Demographic and Biochemical Data in Patients of Three Groups

| | ST-Elevation N = 17 (11.3%) | Isoelectric N = 59 (39.3%) | ST-Depression N = 74 (49.3%) | P Value |
|-----------------------------------|--|---------------------------------------|---|----------------|
| Age | 54.24 ± 13.30 | 53.47 ± 11.04 | 57.64 ± 10.79 | 0.283 |
| Sex (male) | 12 (70.6%) | 49 (83.1%) | 61 (82.4%) | 0.480 |
| Hypertension | 11 (64.7%) | 27 (45.8%) | 34 (45.9%) | 0.342 |
| Diabetes mellitus | 5 (29.4%) | 12 (20.3%) | 14 (18.9%) | 0.627 |
| Hyperlipidemia | 4 (23.5%) | 12 (20.3%) | 14 (18.9%) | 0.909 |
| Family history | 0 (0.0%) | 4 (6.8%) | 5 (6.8%) | 0.542 |
| Active smoking | 8 (47.1%) | 35 (59.3%) | 35 (47.3%) | 0.352 |
| Creatinine (mg/dL) | 0.93 ± 0.13 | 1.03 ± 0.21 | 1.12 ± 0.59 | 0.203 |
| Hematocrit (%) | 41.63 ± 4.56 | 41.41 ± 4.96 | 42.02 ± 4.69 | 0.776 |
| Triglyceride (mg/dL) | 129.59 ± 84.02 | 171.65 ± 129.32 | 143.00 ± 103.49 | 0.272 |
| Total cholesterol (mg/dL) | 173.80 ± 49.07 | 191.35 ± 44.63 | 178.58 ± 39.99 | 0.202 |
| Blood glucose (mg/dL) | 158.73 ± 87.06 | 158.85 ± 79.72 | 160.04 ± 70.53 | 0.996 |
| Peak creatine phosphokinase (u/L) | 1549.23 ± 561.48 | 1257.41 ± 786.31 | 1681.05 ± 1215.42 | 0.101 |
| Peak CK-MB (ng/mL) | 181.50 ± 81.35 | 156.59 ± 108.92 | 165.93 ± 123.41 | 0.756 |
| Peak CTnI (ng/mL) | 12.97 ± 8.48 | 13.16 ± 12.06 | 19.59 ± 12.18 | 0.011 |

stated as frequencies and percentages. Chi-square analysis test was used to compare the frequencies of categorical variables. One-way between-groups analysis of variance was conducted to compare continuous variables among study groups and post hoc comparisons were done with use of Tukey HSD test. Sensitivity and specificity analysis performed using common statistical methods. Statistical significance was defined as $P < 0.05$.

RESULTS

A total of 150 patients with inferior STEMI were included in this study. The mean age of patients was 55.6 ± 11.29 years. Out of 150 patients, 122 (81.3%) were male and the remaining 28 patients (18.7%) were female. The mean time interval between the onset of chest pain and the recording of first ECG was 5.08 ± 3.32 hours. Thrombolytic therapy was administered in 76 patients (50.7%). Primary PCI was done in 56 (37.3%) patients and the remaining patients were conservatively treated. ST elevation ≥ 0.5 millimeters in lead aVR was present in 17 patients (11.3%) and 74 patients (49.3%) had ST depression ≥ 0.5 millimeters in lead aVR. The remaining 59 patients (39.3%) did not have significant ST segment changes. Right coronary artery was the IRA in 117 patients (78%) and in the remaining 33 patients (22%) left circumflex artery was IRA. The location of culprit lesion was proximal RCA in 41 patients (27.3%), distal RCA in

76 patients (50.7%), proximal LCX in 23 patients (15.3%), and distal LCX in 10 patients (6.7%).

The patients were allocated into three groups based on ST segment changes in lead aVR. There were no significant differences among three groups, regarding mean age, sex and prevalence of coronary artery disease risk factors. (Table 1)

Clinical Characteristics

Serum blood glucose, lipid levels and serum creatinine were similar in three groups. Mean peak CK-MB was 181.50 ± 81.35 ng/mL in the group with ST elevation; 165.93 ± 123.41 ng/mL in the group with ST depression and it was 156.59 ± 108.92 ng/mL in the isoelectric group ($P = 0.756$). Peak cTNI was 12.97 ± 8.48 ng/mL in the group with ST elevation, 19.59 ± 12.18 ng/mL in the group with ST depression and it was 13.16 ± 12.06 ng/mL in the isoelectric group, which was significantly different between groups ($P = 0.011$). Post hoc comparisons using the Tukey HSD test indicated that the group with ST depression had significantly higher peak-cTNI than the isoelectric group with a P value of 0.014. However, the group with ST elevation was not different from the isoelectric group or the group with ST depression regarding peak cTNI (Table 1).

The treatment method and occurrence of atrial and ventricular arrhythmia, bundle branch blocks and atrioventricular (AV) blocks were similar in three groups except for first degree atrioventricular block, which was significantly different in three

Table 2. Comparison of Clinical Complications and Outcomes in Patients of Three Groups

| | ST-Elevation N = 17 (11.3%) | Isoelectric N = 59 (39.3%) | ST-Depression N = 74 (49.3%) | P Value |
|---|--|---------------------------------------|---|----------------|
| Primary percutaneous coronary intervention | 6 (35.3%) | 20 (33.9%) | 30 (40.5%) | 0.721 |
| Percutaneous coronary intervention | 10 (58.8%) | 32 (54.2%) | 37 (50.0%) | 0.768 |
| Thrombolytic therapy | 8 (47.1%) | 32 (54.2%) | 36 (48.6%) | 0.775 |
| Left ventricular ejection fraction (%) | 47.06 ± 5.02 | 47.63 ± 4.08 | 45.81 ± 5.97 | 0.327 |
| Reduced right ventricular ejection fraction | 3 (17.6%) | 7 (11.9%) | 16 (21.6%) | 0.336 |
| Right ventricular enlargement | 2 (11.8%) | 3 (5.1%) | 10 (13.5%) | 0.265 |
| First day ventricular tachycardia/fibrillation | 0 (0.0%) | 2 (3.4%) | 3 (4.1%) | 0.703 |
| Ventricular tachycardia/fibrillation other days | 0 (0.0%) | 0 (0.0%) | 1 (1.4%) | – |
| Atrial fibrillation | 2 (11.8%) | 2 (3.4%) | 6 (8.1%) | 0.372 |
| Left bundle branch block | 0 (0.0%) | 0 (0.0%) | 0 (0.0%) | – |
| Right bundle branch block | 0 (0.0%) | 0 (0.0%) | 1 (1.4%) | – |
| First degree atrioventricular block | 3 (17.6%) | 3 (5.1%) | 1 (1.4%) | 0.016 |
| Second degree atrioventricular block | 1 (5.9%) | 1 (1.7%) | 3 (4.1%) | 0.621 |
| Complete heart block | 0 (0.0%) | 6 (10.2%) | 7 (9.5%) | 0.398 |
| Heart failure | 0 (0.0%) | 3 (5.1%) | 4 (5.4%) | 1 |
| In-hospital mortality | – | – | – | – |
| Mortality in follow-up period | 0 (0.0%) | 1 (1.7%) | 1 (1.4%) | 1 |

groups [17.6% in STE (+), 5.1% in isoelectric and 1.4% in STD (+), $P = 0.016$]. Based on the results of two-dimensional echocardiography, left ventricular ejection fraction was not significantly different in patients of three groups, [47.06 ± 5.02% in STE (+), 47.63 ± 4.08% in isoelectric and 45.81 ± 5.97% in STD (+), $P = 0.327$]. Reduced right ventricular ejection fraction and right ventricular enlargement were not significantly different among three groups (Table 2). Duration of hospitalization was 6.1 ± 2.6 days in the group with ST elevation, 5.4 ± 1.7 days in the group with ST depression and it was 5.6 ± 1.9 in the isoelectric groups ($P = 0.284$). Frequency of acute heart failure was not different between groups.

During hospitalization, all patients survived and discharged from the hospital. During follow-up period, which was 7.3 ± 1.2 months in average, 148 patients were alive and death occurred only in two patients (1.33%). (Table 2)

Infarct Related Artery and Culprit Lesion Location

Regarding angiographic data, the infarct related artery was significantly different among three groups with a marginal P value of 0.049. In the group with STE, RCA was the IRA in 16 patients (94.1%). It was IRA in 49 patients (83.1%) of the isoelectric group and in 52 patients (70.3%) of the group with ST depression in lead aVR. In the

group with STE, LCX was IRA in only one patient (5.9%) and it was IRA in 10 patients (16.9%) of the isoelectric group and 22 patients (29.7%) of the group with ST depression. Multivessel involvement and prevalence of dominant LCX artery were not different in three groups (Table 3).

The stenosis was due to proximal RCA lesion in nine out of 17 patients (52.9%) in the group with STE, in 15 out of 59 patients (25.4%) in the isoelectric group and in 17 out of 74 patients (23%) in the group with STD, which was significantly different in three groups ($P = 0.034$). However, considering only patients in whom RCA was determined as IRA by angiographic studies, prevalence of proximal versus nonproximal RCA culprit lesion was not significantly different in three groups (Table 3).

STE in lead aVR was present in 16 cases (13.7%) of the group with RCA involvement and it was present in 9 (22%) patients with proximal RCA involvement. one case (3%) with LCX involvement had STE in lead aVR.

STD in lead aVR was present in 52 cases (44.4%) of the group with RCA involvement and it was present in 17 (41.7%) patients with proximal RCA involvement. Among patients with LCX involvement, 22 cases (66.7%) had STD in lead aVR.

Nonsignificant ST segment changes, was present in 49 cases (41.9%) of the group with RCA involvement and it was present in 15 (36.6%)

Table 3. Comparison of Infarct Related Artery and Location of Culprit Lesion in Patients of Three Groups Based on ST Change in Lead aVR

| | Total N = 150 | ST Elevation N = 17 (11.3%) | Isoelectric N = 59 (39.3%) | ST Depression N = 74 (49.3%) | P Value |
|--------------------------|------------------|--------------------------------|-------------------------------|---------------------------------|--------------|
| RCA | 117 (78.0%) | 16 (94.1%) | 49 (83.1%) | 52 (70.3%) | 0.049 |
| LCX | 33 (22.0%) | 1 (5.9%) | 10 (16.9%) | 22 (29.7%) | |
| Proximal RCA | 41 (27.3%) | 9 (52.9%) | 15 (25.4%) | 17 (23.0%) | |
| Nonproximal RCA | 76 (50.7%) | 7 (41.2%) | 34 (57.6%) | 35 (47.3%) | 0.034 |
| LCX | 33 (22.0%) | 1 (5.9%) | 10 (16.9%) | 22 (29.7%) | |
| Proximal RCA | 41 (27.3%) | 9 (52.9%) | 15 (25.4%) | 17 (23.0%) | |
| Other than proximal RCA | 109 (72.7%) | 8 (47.1%) | 44 (74.6%) | 57 (77.0%) | 0.040 |
| Proximal RCA | 41 (35%) | 9 (56.3%) | 15 (30.6%) | 17 (32.7%) | 0.156 |
| Distal RCA | 76 (65%) | 7 (43.8%) | 34 (69.4%) | 35 (67.3%) | |
| Proximal LCX | 15 (45.5%) | 1 (100%) | 5 (50%) | 9 (40.9%) | |
| Distal LCX | 18 (54.5%) | 0 (0%) | 5 (100%) | 13 (59.1%) | 0.480 |
| Dominant LCX | 6 (4%) | 0 (0%) | 2 (3.4%) | 4 (5.4%) | 0.564 |
| Two/three vessel disease | 67 (44.7%) | 7 (41.2%) | 29 (49.2%) | 31 (41.9%) | 0.672 |
| One vessel disease | 83 (55.3%) | 10 (58.8%) | 30 (50.8%) | 43 (58.1%) | 0.760 |
| Two vessel disease | 57 (38%) | 5 (29.4%) | 25 (42.4%) | 27 (36.5%) | |
| Three vessel disease | 10 (6.7%) | 2 (11.8%) | 4 (6.8%) | 4 (5.4%) | |

LCX = left circumflex coronary artery; RCA = right coronary artery.

patients with proximal RCA involvement. Among patients with LCX involvement, 10 cases (30.3%) had nonsignificant ST segment changes.

In the second analysis, based on presence or absence of ST elevation in lead aVR, involvement of RCA as the infarct related artery was more common in the group with STE and involvement of LCX as the infarct related artery was more prevalent in the group without STE ($P = 0.121$). In the group with STE, RCA was the IRA in 16 patients (94.1%) and it was IRA in 101 patients (75.9%) of the group without STE in lead aVR. In the group with STE, LCX was IRA in only one patient (5.9%) and it was IRA in 32 patients (24.1%) of the group without STE. Although the result is not significant, due to low number of patients with STE, it should be interpreted with caution (Table 4).

In the third analysis, based on presence or absence of ST depression in lead aVR, involvement of LCX as the infarct related artery was more common in the group with STD and involvement of RCA as the infarct related artery was more prevalent in the group without STD ($P = 0.024$). In the group with STD, LCX was the IRA in 22 patients (29.7%) and it was IRA in 11 patients (14.5%) of the group without STD in lead aVR. In the group with STD, RCA was IRA in 52 patients (70.3%) and it was IRA in 65 patients (85.5%) of the group without STD (Table 4).

Presence of ST elevation in lead aVR had 13.68% sensitivity and 96.97% specificity for detecting

right coronary artery involvement. It has also 21.95% sensitivity and 92.66% specificity for identifying proximal RCA lesion as the culprit lesion from nonproximal RCA or LCX culprit lesions. Presence of ST depression had 66.67% sensitivity and 55.56% specificity for detecting LCX culprit lesions (Table 5).

DISCUSSION

According to the results of this study, ST segment deviation in lead aVR can help in differentiating RCA from LCX infarction in patients with inferior STEMI. Regarding the fact that patients with inferior STEMI may have different clinical complications and prognosis based on the involved artery, identifying IRA on admission with a noninvasive and accessible tool can provide valuable information. As a result, ECG is commonly being used to predict IRA before invasive managements.^{1,4} However, previously established electrocardiographic criteria have moderate ability for differentiating RCA from LCX lesions.^{3,4} Differences in coronary anatomy of the patients and multivessel involvement, may further complicate the interpretation of ECG patterns.¹⁸

In an attempt to discover any other ECG criteria for locating IRA in inferior STEMI, the role of ST segment changes in lead aVR is investigated in recent studies. However, diagnostic and prognostic ability of these changes are not

Table 4. Comparison of Infarct Related Artery and Location of Culprit Lesion in Patients with and Those without ST Elevation and in Patients with and Those without ST Depression in Lead aVR

| | ST Elevation N = 17 | No ST Elevation N = 133 | P Value | ST Depression N = 74 | No ST Depression N = 76 | P Value |
|--|------------------------|----------------------------|---------|-------------------------|----------------------------|--------------|
| Culprit artery RCA | 16 (94.1%) | 101 (75.9%) | 0.121 | 52 (70.3%) | 65 (85.5%) | 0.024 |
| Culprit artery LCX | 1 (5.9%) | 32 (24.1%) | | 22 (29.7%) | 11 (14.5%) | |
| Proximal RCA lesion | 9 (56.3%) | 32 (31.7%) | | 17 (32.7%) | 24 (36.9%) | |
| Distal RCA lesion | 7 (43.8%) | 69 (68.3%) | 0.088 | 35 (67.3%) | 41 (63.1%) | 0.699 |
| Proximal LCX lesion | 1 (100%) | 14 (43.8%) | – | 9 (40.9%) | 6 (54.5%) | 0.488 |
| Distal LCX lesion | 0 (0%) | 18 (56.3%) | | 13 (59.1%) | 5 (45.5%) | |
| Proximal RCA lesion in comparison to all other lesions | 9 (52.9%) | 32 (24.1%) | | 17 (23.0%) | 24 (31.6%) | |
| LCX dominance | 0 (0.0%) | 6 (4.5%) | – | 4 (5.4%) | 2 (2.6%) | 0.239 |
| Two/three vessel disease | 7 (41.2%) | 60 (45.1%) | 0.801 | 31 (41.9%) | 36 (47.4%) | 0.516 |

Table 5. Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Values of aVR Lead ST Changes for Detecting Infarct-Related Artery and Culprit Lesion Left Circumflex Coronary Artery (LCX), Right Coronary Artery (RCA)

| | Sensitivity | Specificity | Positive Predictive Value | Negative Predictive Value |
|--|-------------|-------------|---------------------------|---------------------------|
| For right coronary artery lesion | | | | |
| ST elevation | 13.68% | 96.97 % | 94.12% | 24.06 % |
| ST isoelectric | 41.88% | 69.70% | 83.05% | 25.27% |
| For proximal right coronary artery lesion ^a | | | | |
| ST elevation | 21.95% | 92.66 % | 52.94% | 75.94% |
| ST isoelectric | 36.59% | 59.63% | 25.42% | 71.43% |
| For left circumflex artery lesion | | | | |
| ST depression | 66.67% | 55.56% | 29.73% | 85.53% |

^aThe values are calculated for distinguishing proximal right coronary lesion from combined nonproximal right coronary lesion and left circumflex artery lesion.

consistent in different reports, which may partly originate from applying different cutoff points for defining significant ST elevation or ST depression in lead aVR.^{8,10–12,15,16} In this regard, we evaluated the importance of ST elevation in lead aVR for differentiating RCA from LCX lesions. It was found that ST elevation in lead aVR is suggestive of RCA involvement with a specificity of 96.97%; however, considering a sensitivity of 13.68%, absence of ST elevation in lead aVR cannot reliably rule out RCA lesions. In addition, ST depression in lead aVR has moderate sensitivity and specificity for differentiating LCX from RCA lesions in patients presenting with inferior STEMI.

Our findings regarding the direction of ST segment deviation for differentiating RCA from LCX culprit lesions are consistent with results of

other studies.^{8,11,15,16,19} In a paper published by Sun et al., the sensitivity and specificity of ST depression ≥ 1 mm for detecting LCX, as the IRA was 70 % and 94.3%, respectively.¹¹ But another study by Kanei et al. reported a sensitivity and specificity of 53% and 86% for ST depression of ≥ 1 mm in lead aVR to detect LCX, as the IRA.¹² Kohl et al. pooled data of five previous studies and examined the role of ST depression in identifying IRA in inferior STEMI. Their results revealed a sensitivity of 37% and specificity of 86% for finding LCX as IRA.¹⁴ In a recent study, by Mahmoud et al. 50 patients with inferior STEMI, were divided into two groups based on presence or absence of ST depression of ≥ 1 mm and compared regarding IRA. The sensitivity and specificity were reported to be 67% and 72%, respectively.¹⁶ In all these

studies, sensitivity is lower than specificity for detecting LCX lesions. As shown in our study, absence of ST elevation is highly sensitive for detecting LCX lesions; however, using ST depression criteria, decreases sensitivity for ruling out LCX lesions.

Detecting the location of culprit lesion in IRA may also yield important information about the risk of clinical complications. Lesions in proximal RCA can predispose patients to concomitant right ventricular myocardial infarction with subsequent hemodynamic compromise.² As found in this study, while ST segment changes cannot determine location of the lesion in whom RCA is the IRA, ST elevation has a high specificity for distinguishing proximal RCA lesions from combined nonproximal RCA and LCX lesions. In one study, Li et al. reported a sensitivity of 68.8% and specificity of 76.3% for ST depression in aVR to detect LCX lesions; however, they found no significant association between proximal RCA lesions and ECG deviation in lead aVR.²⁰

Occlusion of the RCA with a large posterolateral branch or occlusion of the LCX both causes a transmural ischemia in the inferolateral and apical regions of the left ventricle, which leads to depression of ST segment in lead aVR.²¹ Although occlusion of proximal RCA and subsequently right ventricular ischemia² causes ST elevation in lead aVR, presence of a large posterolateral branch and transmural ischemia in the inferolateral and apical regions of left ventricle,²¹ may neutralize the ST elevation in lead aVR and if large enough may even cause ST depression in lead aVR.

Due to exclusion of high-risk patients in this study for investigating the association of ECG changes with anatomic site of the lesions, the rate of clinical complications were low. As a result it was not possible to compare the prognostic value of these findings. The present evidence about the prognostic significance of ST segment deviation in lead aVR in inferior STEMI is conflicting.^{12, 13, 19, 22} In a report, published by Wong et al. the study population of the HERO-2 trial was investigated to determine prognostic importance of ST changes in lead aVR in acute myocardial infarction. In subset of patients with inferior STEMI, aVR ST elevation ≥ 1 mm was associated with higher 30-day mortality. In contrast, deeper ST depression in lead aVR was not related to 30-day mortality.¹³ Alherbish et al. described same findings in a substudy of APEX-AMI trial.²² However, neither

of them had angiographic data. According to our results, presence of ST elevation in lead aVR is highly suggestive of proximal RCA lesion, which may explain higher mortality in patients with ST elevation in lead aVR. But, this matter is not explored in this study and needs further investigations. Of note is that some studies indicated presence of deeper ST depression as a sign of larger infarction assessed by cardiac enzymes and echocardiographic data.^{7, 12, 23} In our study, comparing patients of three groups, revealed no significant differences regarding left and right ventricular ejection fraction; however, patients with ST depression had higher peak cTNI levels than those without significant ST segment changes.

LIMITATIONS

Due to our strict exclusion criteria, evaluation of clinical complications and outcomes may not applicable to all patients with inferior STEMI. We also grouped the location of culprit lesions into proximal and nonproximal including both mid-point and distal lesions. Larger studies are needed to evaluate the impact of multivessel involvement on ST changes of lead aVR and location of culprit lesions in more detail.

CONCLUSION

ST elevation in lead aVR is highly suggestive of RCA occlusion in patients with inferior STEMI. ST elevation is associated with proximal RCA involvement. However, it is not sensitive enough to exclude RCA lesions in absence of ST elevation. ST depression in lead aVR is suggestive of LCX occlusion but it has moderate sensitivity and specificity for detecting LCX lesions.

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