ORIGINAL ARTICLE

Significance of ST-segment deviation in lead aVR for prediction of culprit artery and infarct size in acute inferior wall ST-elevation myocardial infarction

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Abstract Background: In patients with acute ST-segment elevation myocardial infarction identification of the culprit artery either due to right coronary artery or left circumflex artery was studied. The electrocardiogram can help in earlier risk stratification and better guidance of therapy for reperfusion.

Patients and methods: 50 patients with acute inferior myocardial infarction were divided into two groups. Group A: patients with ST segment depression in lead aVR ≥ 1 mv. Group B: patients with isoelectric ST segment or with ST segment depression in lead aVR < 1 mv. All patients were subjected to coronary angiography, and echocardiography.

Results: Fifty patients with acute inferior myocardial infarction were included in the present study. There were 35 males (70%) and 15 females (30%), with a mean age 55.6 ± 8.8. In Group A, left circumflex artery was the culprit artery in 8 (47%) and right coronary artery was the culprit artery in 9 (53%). Group B, left circumflex artery was the culprit artery in 4 (12%) and right coronary artery was the culprit artery in 29 (88%) patients with aVR depression had significantly larger infarctions (estimated by peak creatine phosphokinase (CPK-MB) levels and transthoracic echocardiography) than patients without aVR depression.

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Standard 12-lead electrocardiography (ECG) is a widely available technology that is routinely applied in the setting of chest pain to identify patients with ST-elevation myocardial infarction (STEMI) who would benefit from emergent revascularization. The use of ECG to predict the location of the culprit coronary lesion within the infarct-related artery (IRA) could provide additional valuable information to augment clinical decision making and expedite reperfusion therapy. Previous studies in patients with acute coronary syndromes have correlated ECG findings to the results of coronary angiography, leading to the formulation of ECG criteria capable of identifying the coronary artery housing the culprit lesion and the site of the culprit lesion within that artery. The culprit artery of anterior STEMI is nearly always the left anterior descending artery (LAD), but inferior STEMI can be caused by an occlusion of either the right coronary artery (RCA) or left circumflex (LCX) artery. Various ECG criteria have been suggested to predict the culprit artery based on the analysis of QT interval, PR interval, R wave, ST deviation, T wave, and QT ratio. Other findings, such as the presence of Q waves or left axis deviation, have also been used. A comprehensive approach to ECG interpretation is necessary in order to determine the most likely culprit artery and to provide additional valuable information to augment clinical decision making.

Conclusion: In patients with inferior wall STEMI, ST-segment depression in aVR was more common in LCX infarcts than RCA infarcts. Patients with aVR depression had significantly larger infarctions, (estimated by peak creatine phosphokinase (CPK-MB) levels and transthoracic echocardiography) than patients without aVR depression.

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2.1. Exclusion criteria

- A history of previous myocardial infarction.
- Right or left bundle branch block.
- Patients not candidate for thrombolytic therapy.
- Pericarditis.

During the hospitalization, CPK-MB was measured every 8 h from admission until the peak. 12-Lead electrocardiograms were recorded at a speed of 25 mm/s and voltage of 10 mm/mV. The ECGs were analyzed by two independent expert readers blinded to the angiographic results. ST-segment deviation was measured at the J-point compared to the T-P segment.

The following findings were identified:

1. ST-segment elevation in lead III > lead II.
2. ST-segment depression in lead 1 ≥ 0.05 mV
3. The ratio of ST-segment depression in V3 to ST-segment elevation in III ≥ 1.2.
4. ST-segment depression in lead aVR ≥ 0.1 mV.

Left ventricular ejection fraction was estimated with transthoracic echocardiography. All patients were subjected to coronary angiography. Angiographic findings were evaluated by an experienced angiographer blinded to the results of the ECG findings. The culprit artery was determined from angiographic characteristics of occlusion (occlusion due to thrombus formation or ulceration with decreased contrast density). Coronary artery stenosis of more than 70% was defined as obstructive and multivessel coronary artery disease was defined as having two or more coronary arteries with obstructive lesions. The coronary flow was determined using the TIMI flow grading system. Based on the origins of the posterior descending artery (PDA) and the posterolateral branch (PL), the RCA was classified as dominant RCA (both PDA and PL were provided by RCA), non-dominant RCA (both PDA and PL were provided by LCX), or co-dominant RCA (PDA was provided by RCA and PL was provided by LCX). A “large” PL branch was defined as a PL branch larger than 2 mm in diameter.

2.2. Statistical analysis

Data were analyzed using the Statistical Package for the Social Science (SPSS version 17 for windows). Statistical significance was defined as a probability level of \( P \leq 0.05 \).
– Chi-square test, for categorical data to test the significance of differences between the study groups. Sensitivity and specificity are the basic measures of accuracy of a diagnostic test. However, they depend on the cut point used to define “positive” and “negative” test results. As the cut point shifts, sensitivity and specificity shift; the sensitivity increases while the specificity decreases, or vice versa.

1) Sensitivity: The test ability to correctly identify those individuals who truly have the disease. High sensitivity implies few false negatives, which is important for very rare or lethal diseases.

2) Specificity: The test ability to correctly identify those individuals who do not have the disease. High specificity implies few false positives.

3) Positive predictive value: Probability of the person having the disease when the test is positive.

4) Negative predictive value: Probability of the person not having the disease when the test is negative.

3. Clinical results

Fifty patients with acute inferior myocardial infarction were included in the present study, the clinical characteristics and associated risk factors are reported in Tables 1 and 2.

Patients were divided into two groups according to the presence or absence of ST-segment depression in lead aVR with acute inferior infarction.

Group A: 17 patients (with ST-depression in lead aVR \( \geq 0.1 \) mV).

Group B: 33 patients (without ST-segment depression in lead aVR, or with ST-depression in lead aVR \( < 0.1 \) mV).

3.1. Electrocardiographic results

Standard 12-lead electrocardiograms were analyzed in all the studied patients and ST-segment depression in lead aVR was measured. Group A (patients with ST-depression in lead aVR \( \geq 0.1 \) mV) had 17 patients (33%), and Group B (without ST-segment depression in lead aVR) had 33 patients (66%).

3.2. Results of cardiac enzymes

There was a significant increase in Group A as regards the level of CPK-MB measured after 8 h than Group B \( (P = 0.001) \). But this difference became non-significant after 24 h (Table 3).

Another comparison was done between patients with RCA infarction (38 patients) with and without ST segment depression in aVR (9 patients and 29 patients respectively), there was a significant difference in the level of CPK-MB on admission and 8 h later but this difference became non-significant after 8 h. (Table 4).

3.3. Results of echocardiograph

(Table 5). Results showed that there was a significant difference \( (P = 0.03) \) between the two groups regarding ejection fraction.

3.4. Results of coronary angiography

In Group A, LCX was the culprit artery in 47% and RCA was the culprit artery in 53%.

In Group B, LCX was the culprit artery in 4 (12%) and RCA was the culprit artery in 29 (88%) (Table 6).

The results of sensitivity and specificity of ST-segment depression in aVR to predict the culprit artery are presented in (Table 7).

The results of sensitivity, specificity, PPV and NPV of various ECG criteria to predict the culprit artery are presented in (Table 8).

4. Discussion

The standard 12-lead ECG, which is an inexpensive, noninvasive, and readily available clinical tool, has a central role in the

Table 1 Risk factors of IHD among the studied patients.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Range 40–73</td>
</tr>
<tr>
<td></td>
<td>Mean ± SD 55.6 ± 8.8</td>
</tr>
<tr>
<td>Gender</td>
<td>Male 35 (70%)</td>
</tr>
<tr>
<td></td>
<td>Female 15 (30%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Positive 23 (46%)</td>
</tr>
<tr>
<td></td>
<td>Negative 27 (54%)</td>
</tr>
<tr>
<td>Diabetes M</td>
<td>Positive 17 (34%)</td>
</tr>
<tr>
<td></td>
<td>Negative 33 (66%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>Positive 24 (48%)</td>
</tr>
<tr>
<td></td>
<td>Negative 26 (52%)</td>
</tr>
<tr>
<td>Family history of IHD</td>
<td>Positive 9 (18%)</td>
</tr>
<tr>
<td></td>
<td>Negative 41 (82%)</td>
</tr>
</tbody>
</table>

Table 2 Comparison of the clinical data of patients in Groups A and B.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Group A no. = 17</th>
<th>Group B no. = 33</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>43–72</td>
<td>40–73</td>
<td>0.56</td>
</tr>
<tr>
<td></td>
<td>Mean ± SD 54.6 ± 9.3</td>
<td>56.2 ± 8.6</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>Male 13 (76%)</td>
<td>22 (67%)</td>
<td>0.47</td>
</tr>
<tr>
<td></td>
<td>Female 4 (24%)</td>
<td>11 (33%)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>Positive 7 (41%)</td>
<td>16 (48%)</td>
<td>0.62</td>
</tr>
<tr>
<td></td>
<td>Negative 10 (59%)</td>
<td>17 (52%)</td>
<td></td>
</tr>
<tr>
<td>Diabetes M</td>
<td>Positive 6 (35%)</td>
<td>11 (33%)</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td>Negative 11 (65%)</td>
<td>22 (67%)</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>Positive 9 (53%)</td>
<td>15 (45%)</td>
<td>0.61</td>
</tr>
<tr>
<td></td>
<td>Negative 8 (47%)</td>
<td>18 (55%)</td>
<td></td>
</tr>
<tr>
<td>Family history of IHD</td>
<td>Positive 2 (12%)</td>
<td>7 (21%)</td>
<td>0.41</td>
</tr>
<tr>
<td></td>
<td>Negative 15 (88%)</td>
<td>26 (79%)</td>
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</tr>
</tbody>
</table>
Patients with aVR depression had significantly larger infarctions, as estimated by peak creatine phosphokinase (CPK) levels, than patients without aVR depression. Among patients with RCA infarction, on the other hand, Kanei et al. in their study reported that ST-segment depression in lead aVR was associated with a large infarct size. Another study had similar findings, and they proposed several anatomical explanations, in those patients in whom ST depression in lead aVR incorrectly predicted the culprit artery, a significant proportion had anatomically left or co-dominant coronary systems. Furthermore, compared to those without ST-segment depression in lead aVR, patients with culprit RCA occlusions with ST depression in aVR had larger size of infarction and impaired myocardial e perfusion compared with patients without ST-segment depression in lead aVR. This was estimated by levels of peak CPK-MB and ejection fraction detected by transthoracic echocardiography.

These results were consistent with the results reported by Menown and Adgey, who found that ST-segment depression in lead aVR was associated with a large infarct size. Another study had similar findings, and they proposed several anatomical explanations, in those patients in whom ST depression in lead aVR incorrectly predicted the culprit artery, a significant proportion had anatomically left or co-dominant coronary systems. Furthermore, compared to those without ST-segment depression in lead aVR, patients with culprit RCA occlusions with ST depression in aVR were significantly more likely to have a large posterolateral branch thus supplying the same inferolateral territory conventionally supplied by the LCX artery. This illustrated the principle that ST-segment depression in lead aVR was actually a reflection that the vector of injury caused by the myocardial infarction was directed toward the apical and inferolateral walls, usually between +120° and –60° in the frontal plane, whether this territory was supplied by the LCX or the RCA with a large posterolateral branch, as proposed by Kanei et al.

5. Conclusion

In patients with inferior wall STEMI, ST-segment depression in aVR was more common in LCX infarcts than RCA infarcts. Patients with aVR depression had significantly larger infarctions, (estimated by peak creatine phosphokinase (CPK-MB) levels and transthoracic echocardiography) than patients without aVR depression.
Significance of aVR depression during inferior wall STEMI 149

Table 8  Sensitivity, specificity, PPV and NPV of various ECG criteria to predict the culprit artery.

<table>
<thead>
<tr>
<th>ECG findings</th>
<th>Number of patients with ECG changes</th>
<th>Culprit artery by CA</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) ST elevation in lead III/lead II &gt; 1</td>
<td>30</td>
<td>RCA: 27</td>
<td>85</td>
<td>83</td>
<td>90</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCX: 3</td>
<td>17</td>
<td>83</td>
<td>10</td>
<td>25</td>
</tr>
<tr>
<td>(2) ST depression in lead I ≥ 0.5 mm</td>
<td>20</td>
<td>RCA: 19</td>
<td>60</td>
<td>94</td>
<td>95</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCX: 1</td>
<td>6</td>
<td>40</td>
<td>5</td>
<td>43</td>
</tr>
<tr>
<td>(3) ST depression in V3/ST elevation in lead III &gt; 1.2</td>
<td>8</td>
<td>RCA: 6</td>
<td>19</td>
<td>89</td>
<td>75</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LCX: 2</td>
<td>11</td>
<td>81</td>
<td>25</td>
<td>62</td>
</tr>
</tbody>
</table>

PPV, positive predictive value; NPV, negative predictive value; CA, coronary angiography.

Conflict of interest

No conflict of interest regarding this paper.

References