Electrocardiographic Criteria for Predicting Site of Coronary Artery Occlusion in Acute Inferior Wall Myocardial Infarction

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Abstract

Background: The determination of infarct related artery in acute inferior myocardial infarction is extremely important for the prediction of potential complication and predicting the probable site of occlusion is worthwhile because proximal occlusions are likely to cause greater myocardial damage and early invasive strategy is indicated.

Objectives: To predict the culprit coronary artery whether right coronary artery or left circumflex artery by examining the surface electrocardiography in patients with acute inferior wall myocardial infarction and also to predict the correlation between the proximity of lesion in right coronary artery and severity of ST segment elevation in inferior electrocardiographic leads that is caused by right coronary artery occlusion.

Methods: A total of 56 patients with inferior wall myocardial infarction were included in this study underwent coronary angiography. The electrocardiography of these patients were then compared with angiographic finding to correlate with culprit artery (either right coronary artery or left circumflex) and also to correlate the proximity of culprit lesion in right coronary artery with the degree of ST segment elevation in inferior limb leads.

Results: After comparing the electrocardiographic findings in inferior and lateral leads, it was evident that the degree of ST segment elevation in leads III and AVF was significantly higher in right coronary artery group (46 patients) vs left circumflex group (10 patients) 3.16±1.14mm vs 1.53±0.24mm (p<0.001) and 2.78±0.92mm vs 1.2±0.25(p<0.001) respectively. While its comparable in lead II 2.18±0.95mm vs 1.7±0.34mm (p>0.05). In respect to lateral limb leads (AVL and I), we found that deeper ST segment depression was in right coronary artery group as compared to left circumflex group 1.11±0.25mm vs 0.2±0.34mm (p<0.001) and 0.69±0.25mm vs 0.25±0.42mm (p<0.001). In precordial leads V2 and V3 we found that the left circumflex group had deeper ST segment depression compared to RCA group although the difference was of no statistical significance 1.9±0.56mm vs 1.55±0.76mm (p>0.05) and 2.1±0.56mm vs 1.73±0.71mm (p>0.05) respectively. Also in right coronary artery group, 15(32.6%) patients had proximal culprit lesion, 19(41.6%) mid and 12(26%) distal culprit lesion. Patient with proximal right coronary artery disease showed a mean ST segment elevation of 11.7±1.8mm and with mid right coronary artery disease 7.2±0.97mm and with distal right coronary artery disease 5.8±0.82mm.

Conclusion

1- It is possible to predict the culprit artery whether right coronary artery or left circumflex by examining the surface electrocardiography in patients with acute inferior myocardial infarction.

2- The degree of ST segment elevation is correlated with proximity of the right coronary artery.

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خليفة الدراسة: ان تحديد الشريان السبب لانشطار عضلة القلب في غاية الأهمية لتنبؤ المضاعفات المحتملة ولتنبؤ موقع الاصابة في الشريان الامن للإنسداد يسبب ضررا أكبر في عضلة القلب وهذا النتائج الاغني يجب ان يكون في وقت مبكر.
Introduction

Cardiovascular disease is responsible for almost one half of all deaths in the United States and other developed countries and for one fourth of deaths in the developing world. By 2020, cardiovascular disease will cause one of every three deaths worldwide. Coronary heart disease is the leading cause of cardiovascular death [1].

Electrocardiography is the cornerstone for diagnosis of myocardial infarction, although many factors limit the ability of electrocardiography to diagnose and localize myocardial infarction like the presence of conduction defect, the presence of previous myocardial infarction, change in electrolyte concentration and administration of cardioactive drugs, but still serial standard 12 leads electrocardiography remain a potent and extremely clinically useful method for detection and localization of myocardial infarction [2-4].

In patient with acute inferior myocardial infarction, the infract related artery could be right coronary artery or left circumflex artery. Right coronary artery supplies blood mainly to the inferior myocardium while left circumflex artery supplies posterior, posterolateral, and posteroinferior myocardium [2].

Acute inferior myocardial infarction is sometimes complicated by hypotension and arrhythmia. In presence of complication, right coronary artery is generally the infarct related artery. So from the above mentioned the determination of infract related artery in acute myocardial infarction is extremely important with regard to prediction of potential complication. Furthermore the probable site of occlusion within right coronary artery is worthwhile because proximal
occlusions are likely to cause greater myocardial damage and early invasive strategy may be planned[5]. This study was conducted to localize the culprit coronary artery whether right coronary artery or left circumflex artery by examining the surface electrocar-diography in patients with acute inferior wall myocardial infarction and to predict the correlation between the proximity of lesion in right coronary artery and severity of ST segment elevation in inferior electrocardiographic leads.

Patients and Methods
A total of 56 patients admitted between January 2009 to June 2010 in Merjan teaching hospital and Ibn-Al Betar center for cardiac surgery whom they had diagnosis of inferior wall myocardial infarction when admitted and subsequently underwent coronary angiography within 3 months of their admission.

Patients with prior myocardial infarction, coronary artery bypass grafting, electrocardiographic evidence of left bundle branch block, right bundle branch block, left ventricular hypertrophy and pre-excitation syndrome were excluded.

Twelve leads electrocardiography were recorded at a paper speed 25 mm/s with amplitude of 10mm/mv, any ST-segment deviation from isoelectric line (T-P segment) was measured to nearest 0.5mm at 80 m sec after the J point.

Full history was taken from the patients regarding the age, sex, hypertension (patient was considered hypertensive if he had history of hypertension or blood pressure ≥140/≥90 mmHg) [6], diabetes (patient was considered diabetic if he had history of diabetes or had fasting plasma glucose at or above 126 mg/dL (7.0 mmol/L) or a random plasma glucose concentration ≥200 mg/dL (11.1 mmol/L) in the presence of symptoms)[7] and smoking.

The amplitude of ST-segment elevation in leads II, III and aVF as well as ST-segment depression in leads AVL and I were recorded and the results were compared between right coronary and left circumflex artery group. Repeated electrocardiography was done and highest level was taken. ST-segment depression in leads V2 and V3 was assessed as being representative of precordial leads. Also the total ST-segment elevations in inferior leads were compared between proximal, mid and distal right coronary artery group patients.

Coronary angiography
The coronary angiography was done by percutanes Judkin’s technique via femoral route with machine Philips Allura 9 model 2007.

Significant narrowing was considered if there was more than 70% of narrowing except left main stem where more than 50% narrowing was considered significant[8]. Patients with significant stenosis of both right coronary and left circumflex arteries were excluded together with those having concomitant significant left main stem or left anterior descending artery disease.

Right coronary artery was divided into proximal, mid and distal. The segment of right coronary artery from its ostium to the origin of first marginal branch was considered proximal, from the first acute marginal to that of last acute marginal was considered mid and from this point onward as distal.

Statistical Analysis
The statistical significance for observed differences was assessed by student T test, test of proportions & analysis of variance used to compare differences between variables[9,10]. P value < 0.05 was considered statistically significant, P value < 0.001 was considered statistically extremely
significant, P value > 0.05 was considered statistically not significant.

**Results**
The study population involved 56 patients (42 males & 14 females) their mean age was (65±7.2 years) with the diagnosis of acute inferior myocardial infarction.

According to the results of coronary angiography patients were divided into two groups.

- **Left circumflex artery occlusion group** 10 patients.
- **Right coronary artery occlusion group** 46 patients which also subdivided into proximal (15 patients), mid (19 patients) and distal (12 patients).

Baseline characteristics of both groups are shown in table 1, who shows that the left circumflex artery occlusion was significantly higher in older age group than right coronary artery occlusion group, while there was no significant effect of sex, hypertension, diabetes mellitus and smoking on occurrence of right coronary artery or left circumflex artery occlusion.

The electrocardiographic manifestations in relation to coronary angiographic findings are presented in table 2 in which there was significantly higher ST segment elevation in lead III, AVF in right coronary artery group as compared to left circumflex artery group (P value < 0.001). The ST segment elevation in lead II was found to be comparable between both groups (P value > 0.05).

When we evaluated lateral limb leads I and AVL, it was found that ST segment depression in these leads was significantly deeper in right coronary artery group as compared to left circumflex artery group (p value >0.001) table 2.

The degree of ST segment depression in leads V2, V3 was measured as shown in table 2. Deeper ST segment depression was found in these precordial leads within left circumflex artery group as compared to right coronary artery group however the difference did not reach a statistical significance (p value>0.05).

In the right coronary artery group the degree of ST segment elevation in lead III was higher than lead II while it was not in the left circumflex artery group. Accordingly we evaluated the ratio of ST segment elevation in lead III to lead II and it was found to be consistent with right coronary artery occlusion (not left circumflex artery occlusion) as shown in table 3.

The degree of ST segment depression in lead AVL was more than in lead I within right coronary artery group while it was comparable in left circumflex artery group for that we estimated the ratio of ST segment depression in lead AVL>1 consistent with right coronary artery group (p value > 0.001) table 3.

Table 4 shows that ST segment elevation in lead III more than lead II was sensitive 96% and specific 93% for right coronary artery occlusion and also ST segment depression in lead aVL more than lead I was also sensitive 97% and specific 90% for right coronary artery occlusion group.

Table 5 shows that sum of ST segment elevation in leads II, III and aVF was higher in proximal right coronary artery disease (11.7±1.8mm) with a range of 8.5-13.5mm than mid right coronary artery disease (7.2±0.97mm) with a range of 6-8.5mm and distal right coronary artery disease (5.08±0.82mm) with a range of 4-6mm.

By applying T test, total ST segment elevation in inferior leads was found to be significantly higher in proximal right coronary artery lesion as compared to mid and distal (P value <0.001) the maximum ST elevation in the inferior leads was found
significantly associated with proximity confirmed by coronary angiography.

**Table 1** Baseline demographics characteristic features of studied groups.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>right coronary artery (n=46)</th>
<th>left circumflex artery (n=10)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± standard deviation)</td>
<td>53.5(6.23)</td>
<td>58.5(8.18)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Male n(%)</td>
<td>34 (73.9%)</td>
<td>8 (80%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Hypertension n(%)</td>
<td>19 (41.3%)</td>
<td>3 (30%)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Diabetes mellitus n(%)</td>
<td>16 (43.7%)</td>
<td>2 (20%)</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Smoking n(%)</td>
<td>33(71.7%)</td>
<td>5 (50%)</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

**Table 2** Electrocardiograph findings in relation to culprit artery

<table>
<thead>
<tr>
<th>Electrocardiographic finding</th>
<th>right coronary artery (n=46) mean±(standard deviation)</th>
<th>left circumflex artery (n=10) mean±(standard deviation)</th>
<th>P.value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment elevation in mm</td>
<td>2.18±(0.95)</td>
<td>1.7±(0.34)</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>III</td>
<td>3.16±(1.14)</td>
<td>1.35±(0.24)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AVF</td>
<td>2.78±(0.9 2)</td>
<td>1.2±(0.25)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ST segment depression in mm</td>
<td>1.11±(0.25)</td>
<td>0.2±(0.34)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AVL</td>
<td>0.69±(0.28)</td>
<td>0.25±(0.42)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>I</td>
<td>1.55±(0.76 )</td>
<td>1.9± 0.56</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>V3</td>
<td>1.73±(0.71 )</td>
<td>2.1 ± (0.56)</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

**Table 3** Comparison of ST segment deviation in inferior and lateral leads.

<table>
<thead>
<tr>
<th>Character</th>
<th>Right coronary artery n= (46%)</th>
<th>Left circumflex artery n=(10%)</th>
<th>p.value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment elevation III&gt;II</td>
<td>44(95%)</td>
<td>2(20%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ST segment depression AVL&gt;I</td>
<td>45(97.8%)</td>
<td>1(10%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

**Table 4** Sensitivity, specificity, positive and negative predictive values of ratios in predicting the presence of right coronary artery occlusion.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Sensitivity</th>
<th>specificity</th>
<th>positive predictive value</th>
<th>Negative predictive value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment elevation in lead III more than lead II</td>
<td>96%</td>
<td>93%</td>
<td>95%</td>
<td>80%</td>
</tr>
<tr>
<td>ST segment depression in lead aVL more than lead I</td>
<td>97%</td>
<td>90%</td>
<td>97%</td>
<td>90%</td>
</tr>
</tbody>
</table>
Table 5 Comparison of total ST elevation in II, III, AVF in right coronary artery group patients.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Proximal right coronary artery</th>
<th>Mid right coronary artery</th>
<th>Distal right coronary artery</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total ST elevation in II, III, AVF in mm</td>
<td>11.7 ± (1.8)</td>
<td>7.2 ± (0.97)</td>
<td>5.08 ± (0.82)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(mean ± standard deviation)</td>
<td></td>
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Discussion

Localization for the territory supplied by culprit artery can explain the electrocardiographic changes during myocardial infarction[3].

In this study we tried to elaborate the capability of the surface electrocardiography in patient with inferior wall myocardial infarction in predicting culprit coronary artery occlusion to be right coronary artery or left circumflex artery. Also in this study we tried to elaborate the correlation of maximum ST segment elevation in the inferior leads with proximity of the culprit lesion along right coronary artery because proximity of culprit lesion along the course of right coronary artery is important with regard to the potential complications like sinoatrial nodal dysfunction, right ventricular infarction, etc.

The achieved data showed that patients with acute inferior myocardial infarction; the higher ST segment elevation in inferior leads III, AVF was correlated with right coronary artery rather than left circumflex artery occlusion [3.16±1.14mm vs 1.53±0.24mm (p<0.001) and 2.78±0.92mm vs 1.2±0.25 (p<0.001)] respectively. This is in agreement with finding in previous study by Masami et al[11] 1.6±0.38mm and 2.06±1.5mm vs 1.4±0.67mm(p<0.05)] respectively.

The ST segment elevation in lead II was found to be comparable in both groups 2.18±0.95mm vs 1.7±0.34mm (p>0.05). This is in agreement with finding in previous study by Masami et al[11] 2.4±1.4mm vs 1.7±1.1mm(p>0.05) and Moskovit et al[12] 1.5±1.05mm vs 1.8±0.6mm (p>0.5).

But the ratio of ST segment elevation in lead III to that in II was found to be sensitive and specific marker for right coronary artery occlusion with high positive and negative predictive value 96%, 93%, 95% and 80% respectively, this is in agreement with finding in previous study by Herz et al[13] 88%, 94%, 98% and 67% respectively and Chia et al[14] 90%, 94%, 94% and 70% respectively.

Standard lead III is oriented to the right inferior segment where as lead II is oriented principally to the left inferior segment and also tend to be oriented to inferior region of left lateral and superior wall of left ventricle.

Consequently lead III is influenced by right coronary artery related acute inferior myocardial infarction where as lead II is more influenced left circumflex artery related acute inferior myocardial infarction.

The other discriminatory finding in our study was that deeper ST segment depression in lead AVL than lead I (AVL/I ratio) was sensitive and
specific marker for right coronary artery occlusion with high positive and negative predictive value 96%, 93%, 95% and 80% respectively. This finding was in agreement with prior study by Herz et al [13] 80%, 94%, 98% and 55% respectively and Berry et al [15] 85%, 98%, 98% and 60% respectively.

In case of right coronary artery occlusion ST segment depression in lead AVL and I represent reciprocal changes that are more prominent in true reciprocal lead, AVL: while in acute myocardial infarction due to left circumflex artery occlusion the high posterolateral and apical segment are ischemic, therefore; the ST segment depression in these leads are canceled out and even ST elevation can appear [16].

In this study deeper ST segment depression in lateral limb leads (AVL and I) was found in right coronary artery group as compared to left circumflex artery group [1.11±0.25mm vs 0.2±0.34 mm (p<0.001)] and [0.69±0.25mm vs 0.25±0.42mm (p<0.001)] this is in agreement with Birubauam et al [17]show ST segment depression in AVL is sensitive early electrocardiographic sign of inferior wall myocardial infarction and Berry et al [15] show ST segment depression in lead AVL and I was significantly more common with right coronary artery related myocardial infarction than with left circumflex artery related myocardial infarction 0.9±0.25mm vs 0.4±0.34mm (p<0.01) and 0.76±0.9mm vs 0.34±0.3mm (p<0.01)

Huey et al [18]reported that ST segment depression in lead AVL and I is significantly less common with left circumflex artery related myocardial infarction than with right coronary artery related myocardial infarction. ST segment depression in leads V2 and V3 (as a representative of the precordial leads) are being truly apposed to posterior rather than inferior wall. This study showed that deeper ST segment in leads V2 and V3 was seen in the group with left circumflex artery occlusion though the difference did not reach statistical significance 1.9±0.56mm vs 1.55±0.76mm (p>0.05) and 2.1±0.56mm vs 1.73±0.71mm (p>0.05) respectively.

The study of Masami et al [11] showed that the degree of ST segment depression in lead V3 in patient with inferior wall acute myocardial infarction being deeper in left circumflex artery than right coronary artery occlusion 2.5±1.5mm vs 3.3±1.7mm (p>0.05) respectively.

Greater precordial ST segment depression in left circumflex artery occlusion might be explained by the reciprocal changes to inferior and posterior ST segment elevation. The relation of the vector direction between posterior and anterior wall is more strongly apposed to that between inferior and anterior wall and so reciprocal changes in posterior ST segment elevation due to left circumflex artery occlusion might cause more prominent ST segment than that caused by changes in inferior ST segment elevation in right coronary artery occlusion [11].

Also in our study a greater total ST segment elevation was observed in patients with proximal right coronary artery occlusion as compared to those with mid and distal right coronary artery, Patients with proximal right coronary artery showed a mean ST segment elevation of 11.7±1.8mm and with mid right coronary artery disease 7.2±0.97mm and with distal right coronary artery disease 5.8±0.82mm. Similar results were observed by Erdem A. et al [19]. Patients with proximal right coronary artery showed a mean ST segment elevation of 12.61±3.79mm, with mid right coronary artery disease 6.88±1.20 mm
and with distal right coronary artery disease 5.05±0.97mm and Naqvi MA et al[5] Patients with proximal right coronary artery showed a mean ST segment elevation of 12.55±1.38mm, with mid right coronary artery disease 8.39±0.89mm and with distal right coronary artery disease 6.0±0.54mm. This means when the lesion is proximal there is more myocardial damage and higher ST segment elevation.

**Conclusion and Recommendation**

1) It is possible to predict the culprit coronary artery in inferior wall acute myocardial infarction by using the readily obtainable measures on the admission electrocardiography.

2) A higher ST segment elevation in lead III than in lead II, and deeper ST segment depression in AVL than in lead I are sensitive and specific markers for right coronary artery related acute inferior wall myocardial infarction.

3) The presence of ST segment depression in lead V2 and V3 is suggestive of left circumflex artery rather than right coronary artery related acute inferior wall myocardial infarction.

4) The amplitude of ST segment elevation and the proximity of culprit lesion along the infarct related right coronary artery were found to be closely related.

5) We recommend analysis of electrocardiography in patients with myocardial infarction especially for interventional cardiologist to predict the culprit coronary artery and its proximity, for more proximal occlusions are likely to cause greater myocardial damage and early intervention is recommended.

**References**


13. Herz I, Assali AR, Adler Y, et al. New electrocardiographic criteria for predicting either the right or the left circumflex artery as the culprit coronary artery in inferior wall myocardial infarction. AM J Cardiol 1997;80:1343-1345.