Prognostic value of ST segment depression in lead aVR as a predictor of in hospital outcomes in patients with acute inferior myocardial infarction

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ST-segment depression in lead aVR is a predictor for culprit artery in patients with inferior myocardial infarction. We assessed the utility of ST-segment depression in lead aVR in patients with inferior myocardial infarction. 100 patients with first time inferior STEMI were included in this study. According to ST-segment depression in lead aVR on admission ECG, patients were divided into two groups: Group I: No ST-segment depression. Group II: ST-segment depression \(\geq 1\)mm. All patients underwent primary PCI. The culprit artery was RCA in 80% of all patients and LCX in 20%. In group I, 90% had RCA, and 10% had LCX as the culprit artery. In group II, 43% had RCA, and 57% had LCX as the target vessel. There was strong correlation between the presence of ST-segment depression in lead aVR and the culprit artery (\(P = 0.0001\)). The mean peak CK-MB was significantly higher in group II (250.62 ± 119.79 u/l) than in group I (191.73 ± 76.19 u/l), \(P = 0.007\). In-Hospital mortality was higher in ST-segment depression group (14.3% vs 2.5%), \(P= 0.03\). Patients with ST-segment depression have higher rate of Killip class IV (14.3% vs 1.3%), \(P= 0.007\). ST-segment depression in lead aVR predicted culprit artery occlusion with high sensitivity and specificity for LCX and was associated with larger infarction size and worse in hospital outcome.

Key words: Inferior myocardial infarction, aVR, culprit artery, ST segment

INTRODUCTION

Inferior wall myocardial infarction (MI) is generally considered as being low risk, compared with anterior wall MI. The reported in hospital mortality rate ranges from 11% in the pre-thrombolytic era to 3.5%–9% in the thrombolytic era which is about half that of anterior wall MI (Reeder, and Gersh, 2000). The 12-lead electrocardiogram (ECG) serves as an essential diagnostic tool in the diagnosis of an acute myocardial infarction (AMI) (Matetzky et al, 1999). The classical 12-lead ECG has been extended using right pericardial (leads V3-6R) and posterior leads (V7-9) helping in the diagnosis of right ventricular and true posterior myocardial infarction (Kinch, and Ryan, 1994). With reference to the 12-lead ECG, all leads except aVR are evaluated in diagnosing the presence, the extent and the location of an AMI. The lead aVR has generally been ignored with the notion that it provides no useful information. In patients with acute ST-segment elevation myocardial infarction (STEMI), identifying the culprit artery on presenting ECG can lead to earlier risk stratification and better guide therapy for reperfusion (Kosuge et al, 1998). 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 analysis of ST-segment elevation and ST-segment depression in different leads (Tierala et al. 2009). More recently, ST-segment depression in lead aVR has been suggested as a predictor of LCX artery involvement (Sun et al. 2007). In the current study, we evaluated the value of ST-segment depression in the lead aVR as a predictor of in-hospital outcome and the culprit artery in patients with acute inferior STEMI.

PATIENTS AND METHODS

Study Design

Between March 2009 and August 2012, 100 consecutive patients with first time inferior STEMI were admitted at the coronary care unit (CCU) at the National Heart Institute, Cairo, Egypt. All patients were included in this retrospective study which was designed to test the clinical significance of ST-segment depression in lead aVR in prediction of in hospital outcome in this category of patients, and to make a correlation between ST-segment depression in lead aVR and the culprit artery detected by coronary angiography. All patients were treated with primary percutaneous coronary intervention (PPCI). Key inclusion criteria were: patients with first inferior STEMI presented within 12 hours of symptoms onset. While key exclusion criteria were: prior history of other cardiovascular diseases (valvular disease or congestive heart failure). In hospital outcome including mortality, re-infarction, heart failure, arrhythmias. In addition, the relation between ST-segment depression in lead aVR and the culprit artery.

Baseline Evaluation

All patients had review of their medical history on admission to emergency department including: analysis of demographic data (age, sex), presence of risk factors of coronary atherosclerosis, associated comorbidities, general and cardiac examination, 12 leads ECG, routine laboratory investigations including: cardiac biomarkers (CK & CK-MB), kidney function tests (S. Creatinine), lipid profile including (total cholesterol, low density lipoprotein (LDL) high density lipoprotein (HDL), triglycerides), random blood sugar.

Electrocardiogram

Patients were classified into two groups according to ST-segment depression in lead aVR on the ECG obtained at hospital admission.

- **Group I:** 79 patients without ST-segment depression.
- **Group II:** 21 with ST-segment depression ≥ 1 mm.

**Coronary angiography and PPCI**

Patients were given 600 mg clopidogrel plus 300mg aspirin. The procedure was performed according to the standard technique for cardiac catheterization. Transfemoral approach was done in all patients using 6 fr. sheaths. 10.000 units of unfractionated heparin (UFH) were given pre intervention. Coronary angiography was performed before PPCI to detect: Infarct related artery (IRA), TIMI flow in the culprit artery, and anatomical status of non-infarct arteries. Extra back up (XB) or judkin left (JL) guiding catheters were used in case of LCX as the culprit artery, while judkin right (JR) catheters were used in case of RCA as the culprit artery. Floppy wire 0.014 was advanced to the distal segment. Aspiration device and use of glycoprotein inhibitors were operator dependant according to the thrombus burden and TIMI flow. Pre dilatation and post dilatation were performed according to presence of residual stenosis. The operator determined the size, type, and length of stents to be implanted in the occlusion segment. Sheaths were removed after 6 hours from the end of PPCI and patients continued on full antithrombotic treatment.

**Study End Points**

In hospital outcome including mortality, re-infarction, heart failure, arrhythmias. In addition, the relation between ST-segment depression in lead aVR and the culprit artery.

**Statistical Analysis**

Data are presented as mean ±SD for normally distributed variables, and as median (range) for skewed data. Between groups comparison was performed using student t-test or (Mann Whitney) for continuous data or by Chi square test for qualitative data. Pearson’s correlation was performed to assess relation between 2 variables. Level of significance p < 0.05 was used, highly significance result is considered if p < 0.01. Data was analyzed using SPSS version 14(SPSS Inc., Chicago, Illinois, USA).

**RESULTS**

**Study population**

The mean age was 55.75 ± 5.22 years (56.7 ± 6.2 vs 54.8 ± 4.3 years in group I and group II respectively, p=0.19), 82% were males, 43% had diabetes mellitus, 53% were smoker, 56% were hypertensive, 49% had dyslipidemia, 23% had positive family history of coronary artery disease (CAD). We did not report significant
Table 1. Baseline characteristics of study population

<table>
<thead>
<tr>
<th>Variables</th>
<th>All Patients N=100</th>
<th>Group I N=79</th>
<th>Group II N=21</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age/year:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>55.75 ± 5.22</td>
<td>56.68 ± 6.15</td>
<td>54.81 ± 4.29</td>
<td>0.19</td>
</tr>
<tr>
<td>Male sex, n(%)</td>
<td>82(82%)</td>
<td>65 (82%)</td>
<td>17 (81%)</td>
<td>0.89</td>
</tr>
<tr>
<td>Positive family history</td>
<td>23(23%)</td>
<td>20 (23%)</td>
<td>3 (14%)</td>
<td>0.29</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>43(43%)</td>
<td>35 (44 %)</td>
<td>8 (38%)</td>
<td>0.61</td>
</tr>
<tr>
<td>Hypertension</td>
<td>56(56%)</td>
<td>45 (57%)</td>
<td>11 (52.4%)</td>
<td>0.71</td>
</tr>
<tr>
<td>Smoking</td>
<td>53(53%)</td>
<td>41 (52%)</td>
<td>12 (57%)</td>
<td>0.67</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>49(49%)</td>
<td>39 (49 %)</td>
<td>10 (47.6%)</td>
<td>0.89</td>
</tr>
</tbody>
</table>

Table 2. Angiographic findings.

<table>
<thead>
<tr>
<th>Angiographic findings</th>
<th>All patients n=100</th>
<th>Group I n=79</th>
<th>Group II n=21</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Culprit artery:</td>
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<td></td>
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<tr>
<td>LCX</td>
<td>20(20%)</td>
<td>8(10%)</td>
<td>12 (57%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>RCA</td>
<td>80(80%)</td>
<td>71 (90%)</td>
<td>9 (43%)</td>
<td></td>
</tr>
<tr>
<td>Multi-vessel disease</td>
<td></td>
<td></td>
<td></td>
<td>0.89</td>
</tr>
<tr>
<td>TIMI flow:</td>
<td></td>
<td></td>
<td></td>
<td>0.89</td>
</tr>
<tr>
<td>0</td>
<td>82(82%)</td>
<td>65(82.3%)</td>
<td>17 (81%)</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>18(18%)</td>
<td>14(18%)</td>
<td>4 (19%)</td>
<td></td>
</tr>
<tr>
<td>Co dominant</td>
<td>20(20%)</td>
<td>16(20%)</td>
<td>4 (19%)</td>
<td>0.9</td>
</tr>
</tbody>
</table>

Clinical presentation on admission

Acute chest pain was the dominant presenting symptom (74%), 68% had diaphoresis, 4% had syncope/dizziness, 31% had nausea/vomiting, 7% had hypotension/shock, 9% had congestive heart failure, and 4% of patients had arrhythmias. Between groups analysis did not reveal significant difference.

ECG on admission

ST- segment depression in lead aVR was reported in 21(21%) patients. 9(43%) patients had ST- segment depression equal to 1mm, while 12 (57%) patients had >1mm depression. Right ventricular infarction was reported in 6 (7.5%), and 3(14.5%) patients in group I and II respectively, p=0.1.

Time from symptoms onset to admission

Twenty percent of patients were presented less than one hour (60 min) from onset of chest pain, 38% presented from 1-2 hours, 42% of patients within 2- 6 hour. We did not report significant difference between both groups for time from symptoms onset to admission, p=0.1.

Door to balloon time

In 79% of patients the door to balloon time was within 90 minutes from admission, while in 21% of patients was more than 90 minutes. There was no significant difference between both groups for door to balloon time, p=0.34.

Angiographic Findings

The culprit artery was RCA in 80% of all patients and LCX in 20%. Within groups analysis showed that in group I, 90% had RCA, and 10% had LCX as the culprit artery. In group II, 43% had RCA, and 57 % had LCX as the target vessel. There was strong correlation between the presence of ST- segment depression in lead aVR and the culprit artery, with 60% sensitivity and 88.7% specificity (P =0.0001). All patients had occlusion in the proximal segment of the culprit artery. Multi vessel disease ( 2 or more vessels with ≥70% diameter stenosis) was reported in 49% of patients in group I versus 48% in group II (P =0.89). Both groups were comparable regarding the distribution of the non culprit arteries. 82% of all patients had TIMI flow 0 pre PCI (82% versus 81%, in group I and group II respectively). 18 % of study patients had pre PCI TIMI flow I (18% versus 19%, in group I and group II respectively). 20% of patients in group I were co dominant versus 19% in group II , P =0.9, Table 2.
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**Procedure related data**

Transfemoral approach was done in all patients. XB guiding catheters were used in patients who had LCX as the infarct related artery. While Judkin right (JR) catheters were used in case of RCA intervention. Floppy wire was used in all patients. Intracoronary glycoprotein inhibitors were used in 31% of patients (29% versus 38%, in group I and II respectively, p=0.42). Aspiration devices were used in 12% of cases (11% versus 14%, in group I and II respectively, p=0.72). Bare metal stents (BMS) were implanted in 79% of patients (79.7%, 76.2% in group I, II respectively, p=0.7), while drug eluting stents (DES) in 21% of patients (20.3%, 23.8% in group I, II respectively, p=0.7). Timi flow at the end of PPCI was III in 78% (81%, 75%, in group I, II respectively, p=0.32), II in 17% (15.2%, 23.8% in group I, II respectively, p=0.32), I in 5% (3.8%, 9.5%, in group I, II respectively, p=0.32). Distal embolization occurred in 7% of cases, no reflow in 4% of patients. No reported cases of coronary dissection or perforation.

**Cardiac biomarkers**

The mean peak CK value was higher in patients with ST-segment depression in lead aVR (2653 ± 1365 u/l) compared to those without ST-segment depression in lead aVR (2065 ± 834 u/l), P = 0.01. The mean peak CK-MB was significantly higher in group II (250.6 ± 119.7 u/l) than in group I (191.7 ± 76.1 u/l), P = 0.007. When RCA was the culprit artery of STEMI the peak value of cardiac marker were still higher in group II (2839.4 ± 1278.6u/l, 288.8 ± 123.2 u/l for CK and CK-MB respectively) than in group I (191.7 ± 76.1 u/l), P = 0.001. However these were no significant differences between groups when the LCX was the culprit artery, group II (2405.6±1502.84u/l, 199.67 ± 99.4u/l for CK and CK-MB respectively) group I (2666.5±1719.4u/l, 219.6 ± 117.9u/l for CK and CK-MB respectively), P = 0.7. Figure 1

**In- Hospital Echocardiographic data**

There was no statistical significant difference between two groups in the EF% level. The mean LVEF% was 50.8 ± 4.8 vs. 49.5 ± 6.5 for group I and group II respectively. There was no evidence of significant mitral regurgitation in either group (P = 0.3)

**In- Hospital outcome**

In- Hospital mortality was reported in 5 patients (5%). Between groups analysis showed higher rates of mortality in-group II (14.3%) compared to group I (2.5%), P = 0.03. The cause of death in group I was VF, and cardiogenic shock (one patient for each). In group II, all patients died from cardiogenic shock ended by VF. Reinfarction was reported in 3 patients (3%), 4.8% versus 2.5% in group II and group I respectively, P = 0.5. Heart failure was classified according to Killip class. There was higher rate in Killip class IV in group II (14.3%) compared to group I (1.3%), P = 0.006. There were no significant differences between groups regarding other Killip classes. Arrhythmias were reported in 24% of patients. Between groups analysis showed that VF was higher in-group II (14.3%) vs group I (1.3%), P = 0.006.

**DISCUSSION**

Various ECG criteria have been suggested to predict the culprit artery in acute inferior AMI based on analysis of ST-segment elevation and depression in different leads (Verouden et al, 2009). The aVR lead is frequently ignored during the analysis of inferior myocardial infarction, but some investigators have suggested that this lead can provide information useful for the
characterization of inferior myocardial infarction (Gorgels et al, 2001). In our work we pointed out the importance of ST-segment depression in lead aVR as a sign of LCX occlusion. And the relation between the ST segment depression in lead aVR and in hospital outcome in patients with inferior wall myocardial infarction. In this retrospective analysis, 80% of patients had RCA as the culprit artery while 20% had LCX. It was found that LCX was the culprit vessel in 57% of patients with ST-segment depression in lead aVR. RCA was found to be the culprit artery in 90% of patients without ST-segment depression in lead aVR. In hospital mortality, reinfarction, heart failure and arrhythmias were higher in patients who had ST-segment depression in lead aVR.

In the present study we did not report significant difference between the two groups in age, sex, or risk factors. This was consistent with Menown and Adgy, 2000, in a study which included 173 patients with inferior wall AMI. Patients with and without ST-segment depression in lead aVR were comparable, with no significant difference between them with respect to baseline demographics and risk factors. In our analysis the correlation between ST-segment depression in lead aVR and LCX as culprit vessel is consistent with Sun et al, 2007 who investigated whether the ST changes in the aVR lead can be used to identify infarct related artery (IRA) in patients with acute inferior myocardial infarction. They found that ST-segment depression in aVR \( \geq 1 \text{ mm} \) was reported in 70% of patients who had LCX as the IRA, and in 4 (5.7%) patients with RCA as IRA. Our results also consistent with Nair and Glancy, 2002 who found that 80% of patients with ST-segment depression \( \geq 1 \text{ mm} \) in lead aVR, the culprit artery was the LCX. with a sensitivity and specificity in differentiating LCX as IRA was 69% and 91%, respectively. However, Kosugue et al, 2005 reported that the infarct related artery in RCA or the LCX did not correlate with ST changes in lead aVR. In this study, patients with ST-segment depression in lead aVR had higher peak CK, CK-MB compared to those without ST segment depression. This may reflect larger infarction size in this category of patients. Menown and Adgey, 2000 reported that the ST-segment depression in lead aVR might be a reciprocal change resulting from ST-segment elevation in the apical and inferolateral walls, which none of the standard 12 leads directly faces. The large posterolateral branch of the LCX or the atroventricular branch of the RCA usually supplies such regions of the left ventricle. Therefore, concurrent ST-segment depression in lead aVR during inferior AMI might reflect transmural ischemia extending to the apical and inferolateral walls in addition to the inferior wall. They reported that ST-segment depression in lead aVR was associated with a large infarct size. However, their study lacked angiographic data and did not confirm the presence or absence of revascularization of the infarct related artery. Senaratn et al, 2003 reported that patients with ST-segment depression in lead aVR appeared to have a higher CPK level compared to patients with non-ST-segment depression in lead aVR, (2.196 \( \pm \) 368 U/L vs 1.566 \( \pm \) 151 U/L). In our work, patients with ST-segment depression in lead aVR have higher in hospital complications than those without ST-segment depression. Piotr et al, 2012 found that patients with ST-segment depression in lead aVR had higher rates of death (16.5% vs 1.0%), composite end-point (27.0% vs 3.2%) and VF (12.1% vs 4.8%) than patients without ST-segment changes in lead aVR (p < 0.001 for all).

CONCLUSION

Our study confirmed the utility of ST-segment depression in lead aVR for predicting culprit artery occlusion with high sensitivity and specificity for LCX and was associated with larger infarction size and worse hospital outcome.

Study limitations

1. Relatively small number of patients may affect the predictive power for ECG criteria.
2. The size of infarction was estimated using peak cardiac markers measured every 6 hours. This is not necessarily the most accurate method to evaluate size of infarction, although it is a standard clinical practice to estimate extent of myocardial damage and infarct.
3. The retrospective design of the study.

REFERENCES

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