QRS Complex Duration is a Marker of Reperfusion in Patients Presenting with Acute ST Segment Elevation Myocardial Infarction

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Abstract

Background: Acute myocardial ischemia decreases electrical conduction velocity through the ischemic myocardium, the aim of the study is to evaluate the value of the change in QRS complex duration as a marker of reperfusion in patients presenting with ST segment elevation myocardial infarction.

Methods: The study included 100 patients presented with ST segment elevation myocardial infarction, 50% treated with fibrinolysis and 50% with primary Percutaneous intervention, all patients were subjected to 12 lead electrocardiography with measurement of QRS complex duration (in millisecond) on admission, sixty and ninety minutes post reperfusion, coronary angiography was done to all patients and they were divided into two groups according to myocardial blush grade, group A (55%) with normal reperfusion (grade 2 and 3) and group B (45%) with impaired reperfusion (grade 0 and 1).

Results: We found that the admission QRS duration didn’t differ in the two groups (p=0.859), and QRS complex duration was found to be significantly shorter in group A at both 60 and 90 min post reperfusion (p<0.001 for both), and found a strong positive correlation between myocardial blush grade and QRS complex narrowing at both 60 min and 90 min post reperfusion respectively (r=0.731, p<0.001 and r=0.739, p<0.001). A cut off 10 msec was determined to be the best QRS complex narrowing duration for prediction of reperfusion at both 60 and 90 min post reperfusion with 100% sensitivity, and a specificity of (43.64% and 58.18%) at 60 min and 90 min post reperfusion respectively.

Conclusion: QRS duration changes post reperfusion is strongly correlated to myocardial reperfusion in patients presenting with ST segment elevation myocardial infarction.

Keywords: QRS complex; Acute myocardial infarction; Myocardial blush grade

Introduction

Acute ST segment elevation myocardial infarction (STEMI) represents the most lethal form of acute coronary syndrome [1], 12 lead electrocardiogram (ECG) is pivotal in providing the diagnosis and subsequent therapeudic decision to initiate thrombolytic therapy, or primary percutaneous coronary intervention (PCI) [2,3]. The primary goal of reperfusion therapy in acute STEMI is to achieve complete and sustained myocardial reperfusion in a timely fashion. Although the post reperfusion electrocardiogram has shown promise as a noninvasive reperfusion marker, electrocardiographic assessment of reperfusion is traditionally based solely on changes of the ST segment, the prolongation of the QRS duration in STEMI has been identified as an independent predictor of adverse outcome, reperfusion therapy was reported to cause decreases in QRS duration and mortality in STEMI [4,5]. This study aimed to evaluate the value of the change in QRS complex duration as a marker of reperfusion in patients with STEMI.

Patients and Methods

An informed consent was obtained from all patients. And the study was approved by the ethical committee. The study is a prospective one carried on 100 patients presented with acute STEMI admitted to cardiology department, from first of August 2014 to end of February 2015, STEMI was defined as typical chest pain of more than 30 min duration with ST-segment elevation >1 mm in at least two consecutive precordial or inferior leads [2]. The study was approved by local ethical committee, all patients included should be within 12 h from the onset of symptoms, 50% of them had received fibrinolytic therapy using streptokinase in a dose of 1.5 million units over one hour then the patients referred for doing control coronary angiography within 24 h after successful fibrinolysis, and 50% underwent primary PCI, Patients that were excluded from the study previous coronary artery bypass grafting, previous PCI, or old myocardial infarction, ECG with bundle branch block, electrolytes abnormalities, left ventricular hypertrophy, or severe ST segment elevation obscuring J point identification, patients with history of recent haemorrhagic stroke, active bleeding or bleeding diathesis, recent history of trauma or major surgery within one month. All patients were subjected to the following investigations including full history taking, clinical examination and cardiac biomarkers CK-MB and troponin I.

Echocardiography

Transthoracic echocardiography was done for all patients, left ventricular ejection fraction (LVEF) was measured by the modified Simpsons method, and the 17-segment model was used for scoring the severity of segmental wall motion abnormalities by wall motion score index (WMSI) according to the American Society of Echocardiography [6].

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Electrocardiogram

Twelve leads electrocardiogram with a paper speed of 50 mm/s and amplification of 10 mm/v was done on admission and was repeated at sixty and ninety minutes following fibrinolysis, or PCI, ST segment resolution using ST-segment deviation score which was calculated by adding the number of millimeters that the ST segment deviates (elevation or depression) from the isoelectric line in all 12 ECG leads on admission and after reperfusion at 60th min and 90th min [7-11], and QRS duration was measured manually from the onset to the J-point in three consecutive beats in the infarct related leads on admission and after reperfusion at 60th and 90th min [12-14].

angiographic evaluation

angiographic evaluation was done to all patients whether treated by primary PCI or received fibrinolytic therapy that followed by coronary angiography at a mean of 3 days after the myocardial infarct. Using the Thrombolysis in Myocardial Infarction (TIMI) flow classification. Primary PCI is considered successful when TIMI-3 flow without significant residual stenosis (>20%) in the infarct related artery (IRA) is achieved. Myocardial blush grade (MBG) is based on the visual assessment of contrast opacification of the myocardial territory subtended by the IRA. MBG grades 0 and 1 are taken as indicator of injured microvasculature (impaired reperfusion) and MBG grades 2 and 3 as indicators of preserved microvasculature (successful reperfusion) [15,16].

Statistical analysis

Continuous variables were presented as mean ± SD and were compared by Student's t-test or Mann-Whitney U test for variables with or without normal distribution, respectively. Categorical variables were expressed as percentages and evaluated with a Chi square test or Fisher's exact test. The Spearman correlation coefficient was calculated to evaluate the association between 2 continuous variables. In order to determine the predictive impact of post-angioplasty QRS duration narrowing on assessment of reperfusion status, receiver operating characteristics (ROC) analyses was performed and best cut off value was determined and at that point sensitivity and specificity were calculated. Statistical analysis was performed using SPSS 20 (Chicago, IL, USA). A probability value of p<0.05 was considered significant.

Results

The 100 patients included in the study were divided into two main groups according to the reperfusion status detected by MBG, group A (n=55) with MBG 2-3 and group B (n=45) with MBG 0-1.

Baseline clinical, echocardiographic and angiographic characteristics of the patients

As shown in Table 1, patients in group B were significantly older (p=0.001) and had a significantly lower EF and higher wall motion score index in comparison to group A. The angiographic characteristics of both groups are shown in Table 2.

**Electrocardiographic findings**

When the two groups compared the in terms of electrocardiographic parameters (Table 3), they had a similar QRS duration on admission (78.0 ± 11.30 vs. 78.64 ± 11.72 msec, p=0.859 respectively), QRS duration was found to be longer in group B compared to group A at both the 60th min ECG (78.44 ± 11.37 vs. 66.55 ± 9.71 msec, p<0.001) and the 90th min post reperfusion ECG (79.56 ± 11.22 vs. 64.37 ± 8.83 msec).

**Table 1:** Comparison between the two studied groups according to baseline data.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A (n=55)</th>
<th>Group B (n=45)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>48.18 ± 8.74</td>
<td>52.62 ± 8.94</td>
<td>0.014</td>
</tr>
<tr>
<td>Gender(male) %</td>
<td>42 (76.4)</td>
<td>33 (73.3)</td>
<td>0.728</td>
</tr>
<tr>
<td>Diabetes mellitus No. ( %)</td>
<td>28 (50.9)</td>
<td>25 (55.6)</td>
<td>0.643</td>
</tr>
<tr>
<td>Hypertension %</td>
<td>14 (25.5)</td>
<td>18 (40)</td>
<td>0.121</td>
</tr>
<tr>
<td>Smoker No. ( %)</td>
<td>27 (49.1)</td>
<td>28 (62.2)</td>
<td>0.189</td>
</tr>
<tr>
<td>Systolic BP (mmHg) (Mean ± SD)</td>
<td>131.09 ± 16.85</td>
<td>135.33 ± 20.40</td>
<td>0.258</td>
</tr>
<tr>
<td>Diastolic BP (mmHg) (Mean ± SD)</td>
<td>84.55 ± 10.33</td>
<td>85.33 ± 9.91</td>
<td>0.99</td>
</tr>
<tr>
<td>Heart rate (bpm) (Mean ± SD)</td>
<td>81.64 ± 11.67</td>
<td>81.67 ± 12.84</td>
<td>0.99</td>
</tr>
<tr>
<td>Killip class I</td>
<td>52 (94.5)</td>
<td>40 (88.9)</td>
<td>0.016</td>
</tr>
<tr>
<td>Killip class II-III</td>
<td>3 (5.4)</td>
<td>5 (11.1)</td>
<td></td>
</tr>
<tr>
<td>CK-MB (µ/dl) (Mean ± SD)</td>
<td>57.32 ± 64.54</td>
<td>75.48 ± 69.90</td>
<td>0.041</td>
</tr>
<tr>
<td>Troponin I (µ/dl) (Mean ± SD)</td>
<td>69.22 ± 77.08</td>
<td>84.11 ± 60.44</td>
<td>0.019</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl) (Mean ± SD)</td>
<td>1.06 ± 0.28</td>
<td>1.14 ± 0.31</td>
<td>0.197</td>
</tr>
<tr>
<td>LDL (mg/dl) (Mean ± SD)</td>
<td>137.27 ± 51.19</td>
<td>140.27 ± 93.73</td>
<td>0.37</td>
</tr>
<tr>
<td>HDL (mg/dl) (Mean ± SD)</td>
<td>47.73 ± 10.10</td>
<td>44.13 ± 10.34</td>
<td>0.083</td>
</tr>
<tr>
<td>Triglycerides (mg/dl) (Mean ± SD)</td>
<td>204.95 ± 98.15</td>
<td>194.76 ± 82.15</td>
<td>0.58</td>
</tr>
<tr>
<td>Myocardial infarction subtypes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior (No.) (%)</td>
<td>28 (50.9)</td>
<td>26 (57.8)</td>
<td>0.493</td>
</tr>
<tr>
<td>Non anterior (No.) (%)</td>
<td>27 (49.1)</td>
<td>19 (42.2)</td>
<td></td>
</tr>
<tr>
<td>Method of reperfusion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrinolytic therapy No. (%)</td>
<td>16 (29.1)</td>
<td>34 (75.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Primary PCI No. (%)</td>
<td>39 (70.9)</td>
<td>11 (24.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pain to needle (hours) (Mean ± SD)</td>
<td>2.87 ± 1.24</td>
<td>4.33 ± 1.86</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>WMSI (Mean ± SD)</td>
<td>1.40 ± 0.37</td>
<td>1.72 ± 0.59</td>
<td>0.007</td>
</tr>
<tr>
<td>Ejection fraction (%) (Mean ± SD)</td>
<td>56.60 ± 7.07</td>
<td>50.67 ± 10.63</td>
<td>0.002</td>
</tr>
<tr>
<td>BP: Blood Pressure; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein; PCI: Percutaneous Coronary Intervention; WMSI: Wall Motion Score Index</td>
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**Table 2:** The angiographic characteristics of the studied groups.
When the amount of change in QRS duration post reperfusion was taken into account, a significant difference was detected between the two groups post-reperfusion at both the 60th min (0.44 ± 1.79 vs. 11.73 ± 8.12 msec, p<0.001) and the 90th min ECG (0.44 ± 1.97 vs. 13.91 ± 9.94 msec, p<0.001).

On correlation analysis there was a strong positive correlation between 60 min and 90 min QRS complex narrowing and MBG (r=0.731 and 0.739 respectively, p<0.001 for both) Figure 1, and there was a strong negative correlation between pain to needle time and 60 min and 90 min QRS complex narrowing (r=−0.367 and −0.360 respectively, p<0.001 for both) Figure 2. Roc analysis was performed to determine the best cut off value of the QRS duration post reperfusion at 60th and 90th min for predicting reperfusion, we found that QRS complex narrowing of 10 msec at 60 min and 90 min post reperfusion was determined to be the best discriminating value for reperfusion assessment, at 60 min (AUC=0.89, sensitivity 100%, specificity 43.64%, p<0.001), and at 90 min (AUC=0.88, sensitivity 100%, specificity 58.18%, p<0.001) as shown in Figure 3, also QRS complex duration cut off of 65 msec at both 60 min and 90 min post reperfusion was determined to be the best discriminating value for reperfusion assessment, at 60 min (AUC=0.79, sensitivity 80%, specificity 67.27%, p<0.001), and at 90 min (AUC=0.822, sensitivity 82.22%, specificity 78.18%, p<0.001) as shown in Figure 4.

Discussion

Although ST segment deflections have been widely utilized as a means of assessing the degree of underlying ischemic injury, the relationship of QRS complex alterations to the ischemic process is poorly understood. Prolongation of QRS that occurs during acute coronary
obstruction is mostly due to slowing of conduction within the ischemic areas and this mechanism was proved in several experimental models and human studies [17-20]. In our study, we analyzed QRS complex duration changes on admission, at 60 and 90 min post reperfusion and we found that there is no significant difference between the two groups regarding the admission QRS but there is a significant prolongation of the QRS duration in 60 min and 90 min post reperfusion ECG in the group with impaired perfusion compared to group with a successful perfusion and our findings are in agreement with several published studies. Cantor et al. [21], in his study had measured the QRS duration in 51 patients undergoing elective PCI and concluded that QRS prolongation was an ischemia marker in most patients during PCI and was more sensitive than chest pain or ST-T changes. Erdogan et al. [22], enrolled 148 patients presented with STEMI and treated by primary PCI, the patients in the impaired reperfusion group had a significantly longer QRS duration both at immediate post-angioplasty (78 ± 18 ms, 68 ± 17 msec, p=0.001) and at the 60th min ECG (77 ± 17 vs. 60 ± 17 msec, p<0.001) and after adjusting all variables, QRS narrowing in the 60th min ECG was determined as an independent electrocardiographic predictor of reperfusion, his findings are in much agreements with our study findings, also Tsukahara et al. [23] reported QRS duration normalization within 24 h following successful angioplasty in 79% of patients having intermediately prolonged QRS duration on admission that suggest that the QRS prolongation on presenting ECG might have been caused primarily by extensive ischemia and poor metabolic state, rather than by myocardial fibrosis and increased myocardial mass, which are associated with persistent QRS prolongation.

In our study, there was a strong negative correlation between pain to needle time and 60 and 90 min QRS narrowing duration and this indicate that prolonged ischemia and delayed reperfusion leads to QRS prolongation and this was investigated by Henriques JP et al. [24], as they found that delayed reperfusion results in thrombus organization with distal embolization that will decrease the QRS narrowing post reperfusion.

Conclusion

QRS duration changes post reperfusion is strongly correlated to myocardial reperfusion in patients presenting with STEMI.

Study Limitations

One of the limitations in this study is the small sample size, and measuring the QRS by manual methods instead of the automatic methods but we did that because the manual method was more accurate in presence of the ST segment elevation, also measuring QRS duration for every STEMI patient on admission and after revascularization, is not standard in clinical practice.

Conflict of Interest

None declared.

References