CORRELATION BETWEEN P WAVE DISPERSION AND MYOCARDIAL FUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Abstract

Background: P wave dispersion because of its relation to the non-homogenous and interrupted conduction of sinus impulses both Intra and inter atrial is a non-invasive indicator that enables the calculation of Atrial fibrillation (AF) risk on the 12–lead surface ECG and correlates to LV end diastolic pressure.

Aim: to study the relationship between P wave dispersion and left ventricular function was investigated in patients presenting with acute anterior myocardial infarction.

Patients and Methods: The present study included 50 patients diagnosed as acute anterior myocardial infarction and admitted at coronary care unit of Benha university hospital from January 2011 to September 2011; there mean age was 57±9.4, 88% were male and only 12% were females. All patients were subjected for full history taken included age, sex, risk factors for coronary artery disease (CAD) as diabetes smoking, hypertension. ECG was done for every patients with analysis of P wave to determine maximum P wave dispersion (P max.), minimum P wave dispersion (P min.), and the difference in between P max. and P min. called PWD. Echocardiography was done for all patients to determine LA and LV dimensions, LVEF, and also tissue Doppler performed to determine Em/Am ratio and systolic S wave by pulsed wave tissue Doppler at lateral wall of mitral valve on apical four chamber view.

Results: We found that, positive correlation between P wave dispersion and age, LA size, LVEF, tissue Doppler Em/Am ratio, and P wave duration on ECG (p < 0.001); also there was increase in only P max. in patients with systolic dysfunction (P <0.05). There is no effect of thrombolytic therapy, Beta Blocker, and ACE inhibitor on P wave dispersion and duration (P > 0.05).
Conclusion: P wave dispersion is simple noninvasive ECG test can used as predictors of occurrence of arrhythmia in patients with AMI.

Introduction

Diastolic function usually declines before systolic function, and this precedes clinical signs, therefore, diagnosis of diastolic dysfunction is very important for early diagnosis, follow up and treatment (1).

Mitral annulus systolic and diastolic velocities determined by pulsed wave tissue Doppler are relatively preload-independent and reliable variables in evaluating systolic and diastolic left ventricular function (2).

P wave dispersion is related to the non-homogenous and interrupted conduction of sinus impulses intra- and inter atrial; PD is described as a noninvasive indicator of atrial fibrillation risk, which can be calculated easily on a 12-lead surface ECG (3)

Aim of the work

To study the relationship between P wave dispersion and left ventricular systolic and diastolic function in patients with acute anterior myocardial infarction.

Patients and methods

The study included 50 patients with acute anterior myocardial infarction. Patients were selected from patients attending to coronary care unit of Benha university hospital during the period from January 2011 to September 2011.
Acute anterior myocardial infarction was diagnosed when two or more of the following criteria were present: (according to European society and American college of cardiology committee of definition of myocardial infarction 2000) : Chest pain lasting > 30 minutes or ST segment elevation > 2 millimeters in two anterior chest leads or significant elevation in cardiac enzymes.

Patients with arrhythmias or with conduction abnormalities, or cardiogenic shock were excluded

**Methods:**
All patients included in the study were subjected to the following:
1- Careful history analysis which include data about age, sex, smoking, hypertension and diabetes.
2- Complete general and local examination of the heart, chest and abdomen.
3- Electrocardiogram:

The P-wave onset was defined as the first atrial deflection from the isoelectric line, and the offset was the return of the atrial signal to baseline (4). Patients whose measurements could be performed in at least 8 derivations were included in the study. In all patients, derivations were excluded if the beginning or the ending of the P wave could not be clearly identified. Maximum P wave duration (P max) is defined as the longest and minimum P wave duration (P min) is defined as the shortest P wave duration. Leads that showed P min and P max were observed. PWD defined as difference between P max and P min. All the measurements were repeated three times and average values were calculated for each of electrocardiographic parameter. All of the measurements were performed
using the same experienced investigators blind to the subject’s clinical status.

The 12-lead surface electrocardiogram will be calculated manually, with paper speed at 25 mm/s and 10mv/cm standardization, the measurements are performed manually in all 12 leads by using a 0.5 mm scale precision ruler, and magnifying lens. This method has been found to be more precise than the standard caliper method for QT interval (3).

4- Echocardiogram:

All patients will be evaluated by two-dimensional, M –mode, pulsed wave Doppler and pulsed wave tissue Doppler within hours of myocardial infarction. EF% will be calculated from apical two and apical four chamber views using modified Simpson’s method, LA size, FS% will be determined (5).

Trans mitral Doppler parameters will be analyzed; peak early (E) and late (A) trans-mitral filling velocities, the ratio of early to late peak velocities (E/A), deceleration time of (E) will be determined (6).

Pulsed wave tissue Doppler echocardiography will be performed with a 2 mm sample volume placed at the lateral corner of the mitral annulus from the apical four chamber view, Early (Em) and late diastolic (Am), and peak systolic (Sm) mitral annular velocities will be recorded, the ratio of (Em) to (Am) (Em/Am) and E/Em, which is an index for predicting elevated left ventricular filling pressure will be calculated (7).
Statistical Analysis

Data was analyzed using SPSS (Statistical Package for Social Sciences) version 15. Qualitative data was presented as number and percent. Quantitative data was tested for normality by Kolmogrov-Smirnov test. Normally distributed data was presented as mean ± SD. Student t-test was used to compare between two groups. Regression curve was done. P < 0.05 was considered to be statistically significant.(8)

Results

The present study included 50 patients with acute anterior myocardial infarction, their mean age was 57.5 ± 9.4 years, 88% are males and 12% are females, fifty percent were diabetic, 58% were hypertensive, 36% were smokers and 50% had abnormal lipid profile.

Systolic dysfunction (EF<50%) was present in 27 (54%) of patients, one patient had sever (2%), 16 (32%) with moderate systolic dysfunction and 10 (20%) had mild systolic dysfunction.

Diastolic dysfunction was present in 39 (78%) of patients, 15 patients (30%) had grade I, 18 patients (36%) had grade II and 6 patients (12%) had grade III diastolic dysfunction.

Thirty-nine patients (78%) received streptokinase, 32 patients (64%) received beta blocker and 46 patients (92%) received ACE inhibitor.

Fig. (1): shows that, age is the only parameter that correlates positively and significantly with P wave dispersion.
Table (1),(2),(3) shows no significant effect of thrombolytic therapy, patients received Beta blocker or ACE inhibitor on P wave duration and dispersion.
There is insignificant increase in P wave duration and dispersion with
increase in severity of systolic dysfunction (P > 0.05). Table (4)

Table (5): shows, there is insignificant increase in P wave duration and
dispersion in patients with diastolic dysfunction compared to those
without (P > 0.05).

In comparing p wave dispersion and different echocardiography
parameters, we found that significant increase in p wave dispersion
parameters with impaired LVEF, increased LA size (p value < 0.01, and
<0.001 respectively){Table (6),Fig.(2),(3)}, also there is significant
increase in p wave dispersion in correlation with increased left ventricular
filling pressure (Am/ Em ratio and E/Em indices) (p value < 0.001,
<0.05). Table (6) Fig.(5),(6).
Table (1): Comparison of P wave duration and dispersion between patients received and those not received thrombolytic therapy.

<table>
<thead>
<tr>
<th></th>
<th>Non thrombolysis group (n=11)</th>
<th>Thrombolysis group (n=39)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>P max.</td>
<td>114.27±11.22</td>
<td>116.95±15.2</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>P min.</td>
<td>57.55±14.64</td>
<td>57.62±11.03</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PWD</td>
<td>56.73±13.52</td>
<td>59.59±16.41</td>
<td>&gt;0.05</td>
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</tbody>
</table>

Table (2): Relation between beta blocker therapy and (P) wave parameters.

<table>
<thead>
<tr>
<th></th>
<th>Non beta blocker group (n=18)</th>
<th>Beta blocker group (n=32)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>P max.</td>
<td>123.06±13.92</td>
<td>122.59±13.39</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>P min.</td>
<td>59.17±14.06</td>
<td>56.72±10.38</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PWD</td>
<td>54.44±14.49</td>
<td>55.88±15.78</td>
<td>&gt;0.05</td>
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</tbody>
</table>

Table (3): Relation between ACE inhibitor therapy and (P) wave parameters.

<table>
<thead>
<tr>
<th></th>
<th>Non ACE. inhibitor group (n=4)</th>
<th>ACE. inhibitor group (n=46)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P max.</td>
<td>120.50±9.00</td>
<td>116.00±14.76</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>P min.</td>
<td>66.75±10.28</td>
<td>56.80±11.63</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PWD</td>
<td>53.75±13.15</td>
<td>59.41±15.99</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table (4): correlation between P wave dispersion and systolic dysfunction.

<table>
<thead>
<tr>
<th></th>
<th>Mild systolic dysfunction (n=10) (mean ±SD)</th>
<th>Moderate (n=16) (mean ±SD)</th>
<th>Sever (n=1) (mean ±SD)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P max. (msc)</td>
<td>114±8.2</td>
<td>122.5±13.1</td>
<td>130.0±0.0</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>P min. (msc)</td>
<td>54±5.2</td>
<td>61.1±12.2</td>
<td>65±0.0</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PWD (msc)</td>
<td>61±4.1</td>
<td>61.4±17.2</td>
<td>65±0.0</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>
Table (5): Comparison of P-wave duration and dispersion in patients with and without diastolic dysfunction.

<table>
<thead>
<tr>
<th></th>
<th>Patients without diastolic dysfunction (n=11) (mean ±SD)</th>
<th>Patients with diastolic dysfunction (n=39) (mean ±SD)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>P max. (msc)</strong></td>
<td>111.6±11.9</td>
<td>117.7±14.9</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td><strong>P min. (msc)</strong></td>
<td>57.3±13.3</td>
<td>57.7±11.5</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td><strong>PWD (msc)</strong></td>
<td>54.4±16.6</td>
<td>60.3±15.5</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table (6): Correlation between echocardiographic data and P wave dispersion:

<table>
<thead>
<tr>
<th></th>
<th>PWD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>r</strong></td>
</tr>
<tr>
<td><strong>EF</strong></td>
<td>-0.451</td>
</tr>
<tr>
<td><strong>Left atrium size</strong></td>
<td>0.688</td>
</tr>
<tr>
<td><strong>E/A ratio</strong></td>
<td>0.639</td>
</tr>
<tr>
<td><strong>Sm</strong></td>
<td>0.720</td>
</tr>
<tr>
<td><strong>EM</strong></td>
<td>-0.089</td>
</tr>
<tr>
<td><strong>Em/Am</strong></td>
<td>0.489</td>
</tr>
<tr>
<td><strong>E/Em</strong></td>
<td>0.317</td>
</tr>
</tbody>
</table>
Fig. (1) shows correlation between P wave dispersion and age.

Fig. (2) shows correlation between p wave dispersion and LVEF.
Fig.(3) correlation between p wave dispersion and LA size

Fig.(4) correlation between p wave dispersion and diastolic E/A ratio
Fig. (5) correlation between p wave dispersion and Em/Am ratio

Fig (6) correlation between p wave dispersion and E/Em
Discussion

Patients with acute myocardial infarction, frequently have both left ventricular (LV) systolic and diastolic dysfunction, although both isolated systolic and diastolic LV dysfunction can be identified (1).

There is a growing recognition that LV dysfunction is associated with marked alternation in the electro physiologic properties of the myocardium which is the precursor of the cardiac conduction and rhythm abnormalities (9).

Several non-invasive electrocardiographic (ECG) indicators have been investigated to predict the occurrence of arrhythmia in patients with left ventricular dysfunction (10).

In the present study, the relationship between P wave dispersion and left ventricular function was investigated in patients presenting with acute myocardial infarction.

In the present study, there is increase in the mean values of P wave maximum (119.63msc vs 112.52msc) and P wave dispersion (61.41 msc vs 56.09msc) in patients with systolic dysfunction compared to patients without LV systolic dysfunction.

In addition, the present study shows that the P wave duration and P wave dispersion increase along with the severity of LV systolic dysfunction.

Correlation studies in the current study have demonstrated significant positive correlation between LV ejection fraction and P wave dispersion (r =0.30, p =0.03).
These results demonstrate the close association of P wave dispersion and left ventricular systolic dysfunction and are in agreement with the results of other previous studies such as that of (11).

Yilmaz et al (2004). study is the first study in literature to demonstrate a direct correlation between P wave dispersion and tissue Doppler echocardiographic parameters of systolic LV function in patients with acute myocardial infarction.(10)

Additionally, Yilmaz et al. study demonstrated that worsening of LV systolic function parameters was related to the increase of P wave dispersion.(10)

Huseyin (2005) reported that there is a significant relationship between P wave dispersion and LV ejection fraction.(11)

In the current study, there is insignificant increase in P wave dispersion in patients with compared to patients without LV diastolic dysfunction (60.3 +15.5msc vs. 54.4 +16.6 msc, p >0.05).

In the present study, there is a strong positive correlation of P wave dispersion and different parameters of left ventricular diastolic dysfunction: E/A ratio (r=0.714, p <0.001), and E/Em (r=0.3, p<0.05).

As the ratio between E and Em has shown to be a reliable index of left ventricular filling pressure , it can concluded from these results that the increase in left ventricular filling pressure may be reflected on the surface ECG as an increase in P wave dispersion.(7)

Ilknur et al. (2010) studied the relationship between P wave dispersion and diastolic dysfunction in patients with significant and
insignificant coronary artery disease and reported similar results as the present study.\(^{(12)}\)

They concluded that P wave dispersion is a non-invasive marker for left ventricular end diastolic pressure; they stated that P wave dispersion is another alternative for assessment of left ventricular diastolic dysfunction in coronary artery disease. They failed to show any significant change in P wave dispersion with the three different stages of diastolic dysfunction and related this failure to the small studies groups.

**kato (2003)** compared hypertensive patients with LV diastolic dysfunction with hypertensive patients without LV diastolic dysfunction and found P wave dispersion to be higher in LV diastolic dysfunction patients.\(^{(13)}\)

**Yilmaz et al. (2005)** reported that there was a statistically significant positive correlation between the E/Em and P wave dispersion suggesting that the increase of LV filling pressure is associated with increase in P wave dispersion, they concluded that the E/Em ratio early after acute myocardial infarction can be used for predicting elevated LV end diastolic pressure and occurrence of atrial fibrillation.\(^{(10)}\)

In the present work, there is significant strong positive correlation between left atrial dimension and P wave dispersion \((r=0.677, p<0.001)\).

Similarly, **Ilknur et al. (2010)** showed that P wave dispersion is significantly correlated to left atrial volume and dimension in all studied population whether having significant or non-significant coronary artery disease.\(^{(12)}\)
However Dilaveris et al (2000), has been stated that left atrial
diameter is not an important predictor for atrial fibrillation and that P
wave duration is unrelated to left atrial diameter.(3)

The cause of difference between the present study and previous
studies may be due to small sample size (in the present study), different
population studied, different clinical sitting (hypertension vs, acute
myocardial infarction vs. coronary artery disease) and the different
methods used to calculate P wave dispersion.

In the current study, among the different clinical variables (age,
sex, coronary risk factors, heart rate and blood pressure), only the age
was found to be strongly correlated with P wave dispersion (r =0.689,
p<0.001).

In contrary, Huseyin et al. (2005) found that, there is insignificant
relationship between P wave dispersion and these variables including
age.(11)
References


8- **Raymond and Bayarri. 2003**: P Values are not Error Probabilities. A working paper that explains the difference between Fisher's evidential p-value and the Neyman–Pearson Type I error rate α.


