

Relationship between P Wave Dispersion and Left Ventricular Diastolic Dysfunction in Hypertensive and Ischemic Heart Disease Patients

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Abstract:

Key Words :

P wave, LV function, Diastolic function, Ischaemic heart disease, Hypertension.

Background: There is growing recognition that congestive heart failure caused by a predominant abnormality in left ventricular diastolic function is common and causes significant morbidity and mortality. Diastolic function usually declines before systolic function, and this precedes clinical signs. 12-lead electrocardiogram is a commonly used tool to assess left atrial enlargement, which is a marker of left ventricular diastolic dysfunction. We investigated the relationship between P wave dispersion, which is easily measured on the surface electrocardiogram and left ventricular diastolic function.

Methods: There were 100 patients: 50 with diastolic dysfunction and 50 without. P wave dispersions were calculated by measuring minimum and maximum P wave duration values on the surface electrocardiogram. The relationships between P wave dispersion and echocardiographic measurements of diastolic dysfunction were assessed.

Results: Maximum P wave duration was observed significantly ($p=0.001$) in patients with left ventricular diastolic dysfunction (119.60 ± 8.2 ms vs 114.0 ± 6.4 ms). Minimum P wave duration was observed significantly ($p=0.001$) higher in patients without diastolic dysfunction (72.6 ± 7.5 ms vs 62.70 ± 7.4 ms). P wave dispersion was observed significantly ($p=0.001$) higher in patients with left ventricular diastolic dysfunction (56.6 ± 6.3 ms vs 41.5 ± 5.2 ms).

When patients were grouped according to grades of diastolic dysfunction, P wave dispersion was observed sequentially increased among 3 grades of left ventricular diastolic dysfunction (55.8 ± 5.2 ms vs 55.9 ± 7.0 ms vs 61.4 ± 4.7) but the differences were not statistically significant ($p=0.09$).

Conclusion: We conclude that P wave dispersion increases in diastolic dysfunction of LV. When clinical and echocardiographic variables are taken into account, there is a weak but significant correlation between P wave dispersion and left ventricular ejection fraction.

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Introduction:

Thirty to forty percent of patients who show clinical signs of heart failure have normal systolic function but left ventricular diastolic dysfunction (LVDD). There is growing recognition that congestive heart failure caused by a predominant abnormality of LVDD is common and causes significant morbidity and mortality. Diastolic function usually declines before systolic function, and this precedes clinical signs.¹ Ischemic heart disease (IHD) is one of the most important causes of diastolic dysfunction. Evidence for abnormal left ventricular relaxation filling, diastolic distensibility and diastolic stiffness

can be present in coronary artery disease at rest with or without previous myocardial infarction, and during acute ischaemia. Diastolic heart failure patients with congestive heart failure have a better prognosis than those with systolic heart failure.² So, early recognition of diastolic dysfunction or failure is very important.

Left ventricular diastolic dysfunction in ischemic ventricle results in an increase in left ventricular end-diastolic pressure (LVEDP) and in left atrial dimensions. In LVDD, maintenance of sinus rhythm and atrial contractions are vital for stability of cardiac output. If atrial fibrillation (AF) occurs,

atrial output decreases considerably and results in an increase of LVDD and progression of diastolic heart failure, which worsens the patient's clinical condition.³ The increase in left atrial dimensions as a result of rising intra-atrial pressure changes the geometry of atrial fibrils; this, in combination with nonhomogenous fibrosis of the left atrial wall, interrupts the conduction of sinus impulses. As a result, reentry focuses appear, which can start AF. If AF occurs, the loss of atrial kick, which accounts for 40% of atrial output, results in an increase of LVDD and in progression of diastolic heart failure.⁴

Two-dimensional echocardiography with Doppler is the best noninvasive tool to confirm the diagnosis of LVDD. Among other noninvasive tool, 12-lead electrocardiogram (ECG) is a commonly used tool to assess left atrial enlargement, which is a marker of left ventricular diastolic dysfunction⁵.

P wave dispersion (PWD) is defined as the difference between maximum and minimum P wave duration measured from surface ECG. P wave dispersion is related to the nonhomogenous and interrupted conduction of sinus impulses intra and interatrially. Maximum P wave duration and PWD are non-invasive markers that show the heterogeneous and unstable distribution of stimulations originating from the sinus node on the atrium wall.^{6,7}

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. Several ECG indicators have been investigated to predict the occurrence of arrhythmia in patients with left ventricular dysfunction.⁸

Increase in LVEDP and left atrial dimensions results in increase P wave dispersion. P wave dispersion is described also as a noninvasive indicator of AF risk and can predict left ventricular diastolic dysfunction.⁹ As it is a very cheap, easily available tool, it can help us predicting left ventricular diastolic dysfunction without echocardiography.

Methods:

This cross sectional analytical study conducted in the Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka from January

2013 to December 2013. The main objective of the study was to assess the relationship between left ventricular diastolic dysfunction and P wave dispersion in patients with ischemic heart diseases. A total of 100 patients with IHD were included in the study. They were grouped according to the presence or absence of diastolic dysfunction and group I were patients with IHD having LVDD and group II were patients with IHD not having LVDD. Then P wave dispersion in both groups was measured from surface ECG. Patients with hypertension, diabetes, atrial fibrillation, valvular heart disease, cardiomyopathy, electrolyte imbalance, pericardial disease, endocarditis, myocarditis were excluded from the study.

Echocardiographic assessment of left ventricular function:

GE Vivid S5 echocardiography machine was used for performing echocardiography of all the patients by this investigator. Left ventricular systolic functions were determined by Simpson's method. Pulsed Doppler mitral flow velocity was obtained by placing the sample volume between the tips of the mitral leaflets in the apical four-chamber view. The following variables were measured: E, A, E/A ratio, deceleration time of early filling (DT). Mitral inflow patterns were identified by the mitral E/A ratio and DT. They included normal, impaired LV relaxation, pseudo normal LV filling (PNF), and restrictive LV filling. PW tissue Doppler imaging (DTI) was performed in the apical views to acquire mitral annular velocities. The sample volume was positioned at or 1 cm within the septal and lateral insertion sites of the mitral leaflets and adjusted as necessary (usually 5-10 mm) to cover the longitudinal excursion of the mitral annulus in diastole. Primary measurements include the systolic (S), early diastolic and late diastolic velocities. The early diastolic (E2) lengthening velocities were considered to be sensitive measures of LV diastolic function. The subdivision and cutoff values were predefined and based on previous studies in normal subjects and previous combined echocardiographic Doppler studies.¹⁰

ECG assessment of P wave parameters:

12 lead resting ECG was done at a paper speed of 25 mm/s and 10mm standardization. Twelve-lead ECG at rest, with 10 mV amplitude and 25 mm/sec rate, were obtained. The beginning of the P

wave defined was the point where the initial deflection of the P wave crossed the isoelectric line, and the end of the P wave was defined as the point where the final deflection of the P wave crossed the isoelectric line. Patients whose measurements could be performed in at least 3 leads 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. P wave dispersion was calculated by subtracting the minimum P wave duration from the maximum P wave duration. Measurement of P wave parameters was done by using slide calipers and scale.

Results:

The mean age of the studied patients was 49.4 ± 7.9 years ranging from 32 to 70 years. The mean age of the group I patients was 50.0 ± 7.3 years ranging from 39 to 65 years and the mean age of the group II patients was 48.8 ± 8.6 years ranging from 32 to 70 years. The mean age of group I was higher than group II which was not statistically significant ($p=0.47$). Male patients were 44% and 68% in group I and group II respectively. On the contrary female patients were 56% and 32% in group I and group II respectively.

Table-I
Biochemical status of the study subjects (n=100).

Biochemical parameters	Group I (n= 50)		Group II (n=50)		p value
	Mean \pm SD		Mean \pm SD		
Random blood sugar (mmol/L)	6.3 \pm 0.6		6.8 \pm 1.7		0.02
Serum Creatinine mg/dl	1.0 \pm 0.1		1.0 \pm 0.2		0.66
Serum Na ⁺ meq/L	139.4 \pm 3.9		140.3 \pm 3.9		0.27
Serum K ⁺ meq/L	4.1 \pm 0.4		4.1 \pm 0.4		0.30

Table-II
IHD status of the study Population (n=100).

Diagnosis	Group I (n= 50)		Group II (n=50)		Total(n=100)		p value
	Number	%	Number	%	Number	%	
Chronic Stable Angina	13	26.0	15	30.0	28	28.0	0.65
Unstable Angina	9	18.0	10	20.0	19	19.0	0.79
Acute ST-Segment Elevated Myocardial Infarction	16	32.0	12	24.0	28	28.0	0.37
Acute Non ST-Segment Elevated Myocardial Infarction	8	16.0	9	18.0	17	17.0	0.79
Old Myocardial Infarction (Anterior)	2	4.0	2	4.0	4	4.0	1.00
Old Myocardial Infarction (Inferior)	2	4.0	2	4.0	4	4.0	1.00

Table-III
Comparison of P Wave Parameters in Study Population (n=100).

Characteristics	Group I (n= 50)		Group II (n=50)		p value
	Mean \pm SD		Mean \pm SD		
Maximum P wave duration (ms)	119.60 \pm 8.2		114.0 \pm 6.4		0.001
Minimum P wave duration (ms)	62.70 \pm 7.4		72.6 \pm 7.5		0.001
P wave dispersion (ms)	56.6 \pm 6.3		41.5 \pm 5.2		0.001

Table-IV
Comparison of P Wave Parameters in Different Stages of LVDD (N=50).

Characteristics	Grade I: Prolonged	Grade II:	Grade III:	P value
	Relaxation (n=18) Mean \pm SD	Pseudonormalization (n=25) Mean \pm SD	Restrictive Pattern (n=7) Mean \pm SD	
Maximum P wave duration (ms)	115.83 \pm 8.1	121.2 \pm 8.2	123.6 \pm 5.5	0.03
Minimum P wave duration (ms)	59.44 \pm 5.5	65.0 \pm 5.9	62.8 \pm 6.3	0.04
P wave dispersion (ms)	55.8 \pm 5.2	55.9 \pm 7.0	61.4 \pm 4.7	0.09

Table-V
Comparison of Left Atrial Dimension of the Study Population (n=100).

Characteristics	Group I (n= 50)	Group II (n=50)	p value
	Mean \pm SD	Mean \pm SD	
Left atrial dimension (mm)	41.4 \pm 3.8	31.6 \pm 2.9	0.001

Left atrial dimension (mm) was observed significantly ($p=0.001$) higher in group I than group II (41.4 \pm 3.8mm vs 31.6 \pm 2.9mm).

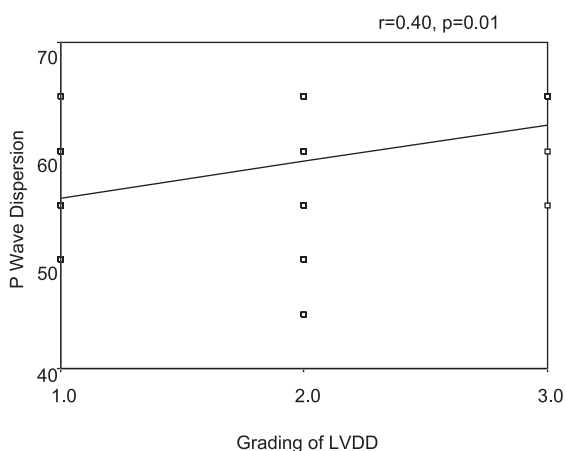


Fig.-1: Correlation between P Wave Dispersion and LVDD Grading.

Table I shows biochemical status of the study patients. The mean random blood sugar was 6.3 \pm 0.6 mmol/L in group I and 6.8 \pm 1.7 mmol/L in group II. The difference was statistically significant ($p=0.02$). It was also observed that the mean electrolyte and serum creatinine levels of the two groups of the study patients were normal.

Table II shows that IHD status among the studied patients was almost identical in both the groups. There was no significant difference between two groups in terms of IHD status.

Maximum P wave duration was observed significantly ($p=0.001$) higher in group I (119.60 \pm 8.2ms vs 114.0 \pm 6.4ms). Minimum P wave duration (ms) was observed significantly ($p=0.001$) higher in group II (72.6 \pm 7.5ms vs 62.70 \pm 7.4ms). P wave dispersion was observed significantly ($p=0.001$) higher in group I than group II (56.6 \pm 6.3ms vs 41.5 \pm 5.2ms).

Maximum P wave duration was observed to increase significantly and sequentially increased ($p=0.03$) among 3 stages of LVDD (115.83 \pm 8.1ms vs 121.2 \pm 8.2ms vs 123.6 \pm 5.5ms). Minimum P wave duration (ms) was observed in the sequence of I, II and III grades of LVDD with significant ($p=0.04$) differences (59.44 \pm 5.5ms vs 62.8 \pm 6.3ms vs 65.0 \pm 5.9). P wave dispersion (ms) was observed sequentially increased among 3 stages of LVDD (55.8 \pm 5.2ms vs 55.9 \pm 7.0ms vs 61.4 \pm 4.7) but the differences were not statistically significant ($p=0.09$).

The figure shows that there is a positive correlation between P wave dispersion and LVDD grading ($r=0.40$). The figure indicates that PWD increases progressively as the grading of LVDD increases. It was observed that the Spearman's correlation was statistically significant ($p=0.01$).

Discussion:

Thirty to forty percent of patients who showed clinical signs of heart failure have normal systolic function but LVDD. 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, treatment, and prognosis evaluation in heart failure patients.¹¹

Because of increased end-diastolic pressure in LVDD, the maintenance of sinus rhythm and atrial contractions is vital for the stability of cardiac output. If AF occurs, the loss of atrial kick, which accounts for 40% of atrial output, results in an increase of LVDD and in progression of diastolic heart failure.¹²

Hypertension and ischemic heart disease are among the most important causes of AF. Left ventricular diastolic dysfunction in a hypertrophic or ischemic ventricle results in an increase in LVEDP and left atrial dimensions. The increase in left atrial dimensions as a result of rising intra-atrial pressure changes the geometry of atrial fibrils; this, in combination with nonhomogenous fibrosis of the left atrial wall, interrupts the conduction of sinus impulses. As a result, reentry focuses appear, which can start atrial fibrillation.¹³

PWD is related to the nonhomogenous and interrupted conduction of sinus impulses intra- and interatrially. Currently, PWD is described as a noninvasive indicator of atrial fibrillation risk, which can be calculated easily on a 12-lead surface ECG.

In this study, maximum P wave duration was observed significantly higher in group I. Minimum P wave duration was observed significantly higher in group II. P wave dispersion was observed significantly higher in group I. P wave dispersion was observed sequentially increased among 3 grades of LVDD but the differences were not statistically significant.

Study result showed that maximum P wave duration, P wave dispersion and left atrial diameter were significantly higher in the patients with LVDD when compared with those values in the control group. Therefore, it can be said that the presence of LVDD is an important factor affecting PWD.

It is known that PWD increases in ischemic heart disease. Therefore, an increase in PWD is expected in patients whose LVDD is associated with ischemic heart disease. As a matter of fact, in our study, the PWD in ischemic heart disease patients

was 56.6 ± 6.3 milliseconds, values that were significantly higher than those for the control group. Left atrial dimension (mm) was observed significantly ($p=0.001$) higher in patients with LVDD.

Another study conducted by Yilmaz et al.¹⁴ showed maximum P wave duration and P wave dispersion are increased, and atrial fibrillation occurrence risk is higher in patients with pseudonormal/restrictive filling pattern after first acute anterior myocardial infarction. The grade of diastolic dysfunction is an independent predictor of P wave measurements and AF occurrence.

Study conducted by Polychronis et al.¹⁵ showed that PWD dispersion showed higher values during the anginal episode in patients with left ventricular dysfunction, independently of the presence of a previous myocardial infarction. Atrial conduction abnormalities, as estimated with P maximum and particularly P dispersion, are significantly influenced by myocardial ischemia in patients with CAD and spontaneous angina.

Study conducted by Necati et al.¹⁶ showed high blood pressure, left ventricular hypertrophy, diastolic dysfunction and increased left atrium diameter and volume shows parallelism in hypertensive cases.

These physio pathological changes may cause different and heterogeneous atrial electrical conduction. This led to a marked increase in maximum P wave and PWD and can be used as a non-invasive marker of target organ damage in the hypertension population.

Study conducted by Tsai et al. showed increased brachial artery pulse wave velocity (baPWV) and PWDC were correlated with high E/Ea and LVDD.⁵ The addition of baPWV and PWDC to a clinical model improved the prediction of high E/Ea and LVDD. Screening patients by means of baPWV and PWDC might help identify the high risk group of elevated left ventricular filling pressure and LVDD.

Study conducted by Dogan et al.¹⁷ showed that impaired LV relaxation contributes to the heterogeneous atrial conduction in hypertensive patients and results in increased PWD.

Conclusion:

From this study it may be concluded that, left ventricular diastolic dysfunction is associated with

increased P wave dispersion in patients with ischemic heart disease. There is a positive correlation between P wave dispersion and LVDD. So, keen observation of P wave dispersion in surface ECG is needed which is a cheap, easily available tool, can help us predicting left ventricular diastolic dysfunction in patients with ischemic heart disease.

Study Limitations

Although the results of this study support the hypothesis, there are some facts to be considered which might affect the results. It was a single centre study and of number of study patients was limited. Measurement of P wave parameters was conducted manually by slide calipers and scale instead of digital method.

Recommendations

Left ventricular diastolic dysfunction is associated with increased P wave dispersion in patients with ischemic heart disease. There is a positive correlation between P wave dispersion and LVDD. P wave dispersion in surface ECG which is a cheap, easily available tool can help us predicting left ventricular diastolic dysfunction in patients with ischemic heart disease and can help in early recognition and treatment of such ailment in ischemic patients. Large population based and multicentre study is needed to establish P wave dispersion in ECG as a predictor of left ventricular diastolic dysfunction.

Conflict of Interest - None.

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