Assessment of atrial conduction time and P-wave dispersion in patients with gestational diabetes mellitus

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Abstract

Purpose: We aimed to assess atrial conduction time and P wave dispersion in Gestational Diabetes Mellitus (GDM) patients. **Methods:** 30 patients with GDM and 30 healthy pregnant women were included to the study. Atrial conduction time times (PA) were calculated by the time interval from the onset of the P wave on the electrocardiography (ECG) to the onset of the late diastolic flow (Am wave) on echocardiography; from the lateral mitral annulus (PA lateral), septal mitral annulus (PA septum), and right ventricular tricuspid annulus (PA tricuspid). **Results:** Mean PWD were higher in GDM (52.7±5.1 ms vs. 28.9±4.2 ms, p<0.001). PA lateral, PA septal and PA tricuspid were significantly higher in the GDM patients compared to the control group (65.7±4.2ms vs. 47.7±4.7 ms, p<0.001; 56.1±3.4 ms vs. 40.8±3.7 ms, p<0.001 and 48.4±3.9 ms vs. 36.0±3.6 ms, p<0.001 respectively). **Conclusion:** We suggest that patients with GDM has higher PWD and higher atrial conduction and EMD times compared to otherwise healthy pregnant control group.

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Methods: 30 patients with GDM and 30 healthy pregnant women were included to the study. Atrial conduction time times (PA) were calculated by the time interval from the onset of the P wave on the electrocardiography (ECG) to the onset of the late diastolic flow (Am wave) on echocardiography; from the lateral mitral annulus (PA lateral), septal mitral annulus (PA septum), and right ventricular tricuspid annulus (PA tricuspid).

Results: Mean PWD were higher in GDM (52.7±5.1 ms vs. 28.9±4.2 ms, $p<0.001$). PA lateral, PA septal and PA tricuspid were significantly higher in the GDM patients compared to the control group (65.7±4.2ms vs. 47.7+-4.7 ms, $p<0.001$; 56.1+-3.4 ms vs. 40.8+-3.7 ms, $p<0.001$ and 48.4+-3.9 ms vs. 36.0+-3.6 ms, $p<0.001$ respectively).

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Keywords: Gestational diabetes mellitus, atrial conduction time, P wave dispersion, electromechanical delay

INTRODUCTION

Gestational diabetes mellitus (GDM) is variable degree of glucose intolerance that begins or is recognized during pregnancy [1]. Pregnant women with GDM have an increased risk of developing type II diabetes mellitus (DM) and cardiovascular disease in long-term follow-ups. Moreover, children of mothers with a history of GDM are at risk of developing obesity and metabolic syndrome in long-term follow-up [2]. In addition, some studies have shown subclinical atherosclerosis[3], endothelial dysfunction [4], and increased risk of cardiovascular disease in women with a history of GDM[5,6]. Diabetes also increases arrhythmic events by causing an imbalance in autonomic tone, silent ischemia, heterogeneity in atrial and ventricular repolarization, and direct myocardial damage through increased arrhythmogenic substrates. Atrial fibrillation (AF) is a common arrhythmia in diabetic individuals [7]. Electrophysiological and electromechanical delays from intraatrial and interatrial conduction disturbances are associated with an increased risk of AF [8,9]. The difference between the maximum P-wave duration (P max) and the minimum P-wave duration (P min) measured noninvasively from a twelve-lead surface electrocardiogram (ECG) is called P-wave dispersion (PWD), and PWD has been found to be closely associated with the development of paroxysmal AF [10]. In recent years, the tissue Doppler method has been used to evaluate intraatrial and interatrial conduction times. Atrial EMD measured by this method have been shown to be prolonged in patients with paroxysmal AF[11]. It has been suggested that atrial conduction times may be disordered in diabetic patients [12]. However, as far as we know, atrial

conduction times has not been studied in GDM patients. The aim of our study is to investigate PWD and atrial conduction times in patients with GDM.

METHODS

After obtaining approval from the local ethics committee (2018/177), we enrolled 30 patients with GDM aged between 18 and 45 years and 30 healthy pregnant women without GDM matched with age and gestational weeks.

All pregnant women were screened for GDM with a laboratory-based screening test using blood glucose levels. Screening for GDM is generally performed at 24–28 weeks of gestation [13]. GDM was determined by 50 g and 100 g oral glucose challenge tests (OGTT) according to the current guidelines [14]. Exclusion criteria included chronic conditions (hypertension, diabetes before pregnancy), eclampsia or pre-eclampsia, ECG rhythm other than sinus rhythm (atrial fibrillation, atrial flutter, atrioventricular (AV) blocks, complete bundle branch block) history of myocardial infarction, chronic obstructive pulmonary disease, asthma, obstructive sleep apnea syndrome, heart failure (ischemic, dilated, hypertrophic), moderate to severe valvular insufficiency or stenosis, history of paroxysmal atrial fibrillation, history of percutaneous coronary intervention (PCI), drug use which may affect cardiac electrical conduction, chronic renal failure, rheumatic and endocrine disease and insufficient echocardiographic image. Medical history, drugs used, pregnancy week, smoking status, laboratory findings (hb, glucose values), and blood pressure values were noted. Body mass index (BMI) was calculated (with the following formula: BMI: weight (kg)/ square of height (in meters)). ECG and echocardiography records of the participants were taken.

The ECGs of the patients were recorded at a rate of 50 mm/sec and amplitude standardization of 20 mm/mV (Nihon Kohden Cardiofax; Japan). While recordings, subjects were permit breathe normally, forbid to hold their breath or speak. ECG analyses were made by Tomax brand digital caliper (150 mm). P wave measurement calculated by averaging the 3 P waves examined in all leads. The beginning of the P wave was considered as the point where the P wave separated from the isoelectric line of the first deviation, and the end of the P wave was taken as the point where it intersects with the isoelectric line again. The PWD was calculated as the difference between the longest and shortest P-wave times in the 12 leads. ECG parameters were measured by the same processor.

Echocardiography was performed on all subjects using a Philips EPIQ 7 device (Philips Medical Systems, Bothell, WA, USA). The patients were examined in the left supine position. Parasternal long axis, short axis, apical four-chamber, and two-chamber images were taken and evaluated using M-mode, 2-D, continuous wave Doppler, pulsed wave Doppler, and tissue Doppler methods according to the criteria of the American Society of Echocardiography [15]. All echocardiography was performed by the same processor. One-lead ECG recordings were taken continuously during the echocardiography procedure. Tissue Doppler echocardiography was performed using a transducer with a frequency of 2.5 MHz's. The pulsed Doppler sample volume was placed in the septal mitral annulus, lateral mitral annulus, and right ventricular tricuspid annulus in 4-chamber view. Systolic wave (Sm) amplitude, early diastolic wave (Em) amplitude, and late diastolic wave (Am) amplitude were determined. Atrial conduction time times (PA) were calculated by the time interval from the onset of the P wave on the ECG to the onset of the late diastolic flow (Am wave) on echocardiography (Figure.1); from the lateral mitral annulus (PA lateral), septal mitral annulus (PA septum), and right ventricular tricuspid annulus (PA tricuspid). The difference between (PA lateral-PA tricuspid) was defined as interatrial electromechanical delay (EMD); the difference between (PA septum-PA tricuspid) was defined as intraatrial EMD, and the difference between (PA lateral-PA septal) was defined as intraleft atrial EMD.

Statistical analyses were performed using SPSS version 20.0 (IBM, Chicago, USA). The conformity of the variables to the normal distribution was examined using Shapiro–Wilk test. Normally distributed numerical variables were compared by independent -T test, and the non-normally distributed variables were compared using Mann–Whitney U test. Categorical variables were compared using the Chi-square test. Spearman's

correlation (for non-normally distributed data) and the Pearson correlation test (for normally distributed data) were used for correlation analysis. Comparisons with a p value below 0.05 were considered statistically significant.

RESULTS

A total of 60 pregnant women were included in the study (GDM=30; controls=30). The BMI (30.9+-4.5 kg/m^2 vs. 28.0+-4.1 kg/m^2, p=0.01) and systolic blood pressure of the GDM (115+-7 mmHg vs 109+-8 mmHg, p=0.006) were higher than control group. Median fasting glucose level was 95 mg/dL in the GDM group and 84 mg/dL in the control group p<0.001. Other clinical and laboratory findings were similar (Table 1).

P max duration and PWD were significantly higher in the GDM group (119.0+-6.6 ms vs. 99.2+-4.1 ms, p<0.001 and 52.7+-5.1 ms, vs. 28.9+-4.2 ms, p<0.001, respectively). Whereas P min duration was significantly lower in in the GDM group (66.2+-3.1 ms vs. 70.3+-3.7 ms, p<0.001) (Table 2).

Among echocardiographic findings IVS diameter and PW thickness were significantly higher in the GDM group (9.9 +- 0.8 mm vs. 9.2 +- 0.8 mm, p=0.001; 9.3 +- 0.8 mm vs. 8.6 +- 1.0 mm, p=0.003). Other echocardiographic findings were similar (Table 3).

PA lateral, PA septal and PA tricuspid were significantly higher in the GDM patients when compared with the controls (65.7+-4.2ms vs. 47.7+-4.7 ms, p<0.001; 56.1+-3.4 ms vs. 40.8+-3.7 ms, p<0.001 and 48.4+-3.9 ms vs. 36.0+-3.6 ms, p<0.001, respectively). Moreover, median values of interatrial EMD, intraleft atrial EMD and intraatrial EMD were also significantly higher in the GDM group than control group (18 ms vs. 12 ms, p<0.001; 10 ms vs.7.5 ms, p<0.001 and 8 ms vs. 4 ms, p=0.001, respectively) (Table 4).

Correlation analysis showed a positive correlation between PWD and age (r=0.36, p=0.04) (Figure 1). Correlations of PWD with other clinical characteristics including BMI, systolic BP and gestation duration were not significant. Among EMDs only intra-atrial EMD was significantly correlated with PWD (r=0.39, p=0.03).

DISCUSSION

The main finding of our study; PWD and atrial conduction and EMD times were significantly higher in the GDM group than otherwise healthy pregnant women. Additionally, positive significant correlation was found between intraatrial EMD and PWD.

GDM is observed in approximately 7 % of all pregnancies[16]. In most affected women, blood glucose returns to normal levels in the postpartum period. However, there is a wealth of evidence for the development of DM later in life in women with a history of GDM [17,18]. However, whether there is a relationship between GDM and cardiovascular diseases is still a research topic. GDM has also been associated with cardiovascular diseases[19]. DM is associated with increased AF risk[20]. Whether GDM may predispose to increased AF risk at long-term is not known. For this reason, in our study, we searched PWD and atrial EMDs, which may be early predictors of future AF risk, in patients with GDM.

Since PWD is a non-invasive simple method, its relationship with atrial arrhythmias in both cardiovascular and other diseases has been widely investigated. Among the subjects of these studies are type II diabetes[21], pre-diabetes[22], hypertension[11], hyperthyroidism[23], rheumatoid arthritis [24], systemic lupus erythematosus[25], ankylosing spondylitis [26], familial Mediterranean fever[27] , scleroderma[28], obesity[29] , inflammatory bowel disease[30], metabolic syndrome [31], obstructive sleep apnea syndrome [32], and hemodialysis[33]. Susceptibility to atrial arrhythmias increases in all these diseases and it has been found that PWD is prolonged in these situations. To our knowledge, PWD and atrial conduction time have not been investigated in in GDM

patients. Clinical value of our finding of increased PWD in GDM patients in the present study requires long-term follow-up.

Several studies have been conducted in patients with type II DM using similar methods with our study. Akyel et al., 40 patients with type II DM and 40 members of the control group were evaluated, and PA mitral and PA septal times were found to be higher in the diabetic group[34]. Demir et al. evaluated 88 type II DM patients and 49 age- and sex-matched controls. They reported that PWD was higher in DM patients than in the controls. Moreover, they found that interatrial, intraatrial, and intraleft atrial EMDs were higher in type II DM patients, and they observed a positive correlation between inter-atrial EMD and PWD [21]. Our findings are in accordance with those reports.

PWD reflects heterogeneous atrial conduction with the detection of abnormal atrial conduction in different ECG leads. The mechanism underlying the increase in PWD in diabetic patients is not clearly known, but the structural and electrophysiological changes caused by diabetes in the atrium are thought to play an important role. Chronic hyperglycemia causes structural and functional disorders by changing the chemical structure of the proteins in the cell membrane. In addition, interstitial fibrosis of the myocardium and extracellular protein deposition cause variability in atrial conduction velocity and atrial refractoriness, resulting in an increase in PWD. Therefore, it is suggested that PWD can be used in the diagnosis of high-risk patients for AF [21,31,35,36].

Similar results have been reported in animal studies. Fu et al. found that atrial electromechanical properties were significantly impaired in diabetic rabbits [37]. Kato et al. reported that intra-atrial activation time and atrial fibrotic storage increased in diabetic animal models [38]. Watanabe et al. evaluated the hearts of diabetic rats with the optical mapping method and stated that the atrial conduction velocity slowed [39]. In an animal study conducted by Li et al. in 2016, the relationship between DM and the P wave was examined at the molecular level. The researchers found that longer P-wave duration in rats with type II DM was not dependent on left atrial enlargement. Rather, it was associated with fibrosis and expression of gap junction proteins such as Cx40 and Cx43, which are important for the progression of impulses in the heart[40]. The increase in PWD, which is one of the main findings of our study, may be due to changes at the cellular level.

PWD and atrial EMD have also been reported in patients with pre-diabetes. Mahfouz et al. stated that as well as PA lateral, PA septal, and PA tricuspid durations, intra-atrial and inter-atrial EMDs were higher in pre-diabetic patients [41]. It was suggested that, atrial cell damage starting in the pre-diabetic phase which includes alterations in the chemical composition of proteins such as collagen I and elastin, may result in changes in atrial conduction times [42]. The physio pathological mechanisms underlying PWD and prolonged atrial conduction times may be similar in GDM patients.

This was a small numbered, cross-sectional, single-center study. Long-term follow up to observe prospective atrial arrhythmic events is lacking. Another limitation of our study was that body mass indexes were not equal in the two groups. However, higher BMI itself is associated with GDM and is a risk factor for GDM. Although there was no significant difference in diastolic BP between GDM and control groups, it was not similar in terms of systolic BP. Nevertheless, all the patients had normal blood pressures. Manual calculation of ECG P-wave durations and PWD is another limitation. Atrial conduction times were measured by tissue Doppler echocardiography, not by the invasive electrophysiological study which is the gold standard.

CONCLUSION

PWD and atrial conduction and EMD times were increased in GDM patients. Considering the findings, it can be said that susceptibility to atrial arrhythmias may be increased in GDM patients. Therefore, it may be convenient to monitor GDM patients for increased PWD and atrial conduction and delay times. Long-term follow-up and large-scale prospective studies will be more instructive to better understand the effects of PWD and atrial EMDs on atrial arrhythmias and atrial fibrillation in patients with GDM.

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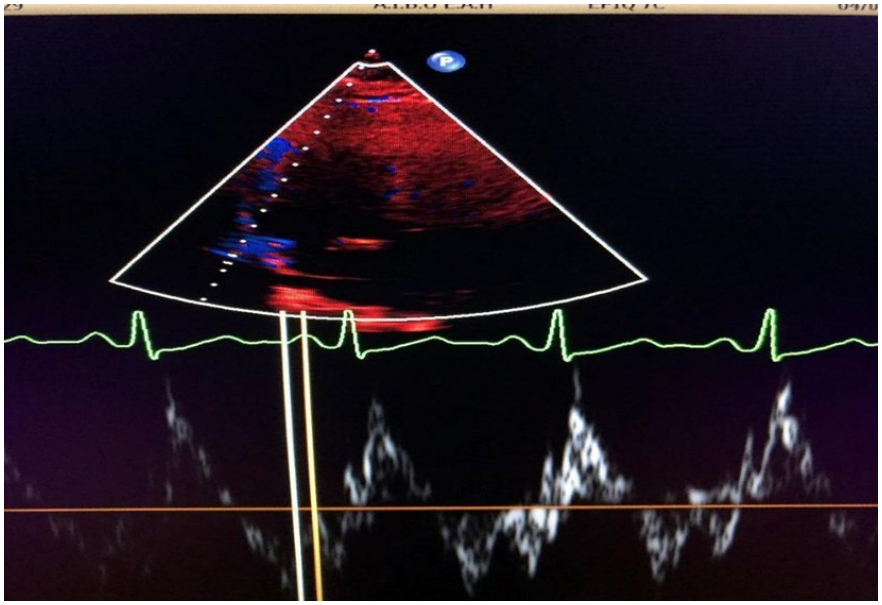
Figure Legends

Figure 1. Atrial conduction time time(PA)



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