Assessment of atrial conduction times in prediabetic patients with coronary artery disease

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Abstract

Objective: Prediabetes is a dysglycemic state and is associated with subtle myocardial injury and dysfunction. We evaluated atrial conduction times (ACTs) and atrial electromechanical delays (EMDs) in prediabetic patients with coronary artery disease (CAD).

Methods: In the present study, we recruited 128 consecutive patient candidates (40 euglycemic, 48 prediabetic, and 40 diabetic patients) for coronary artery bypass grafting. ACTs were measured using tissue Doppler imaging (TDI). The time intervals between the beginning of the P wave in the surface electrocardiogram and the peak of the a' wave in TDI (PA) in the septal and lateral mitral annuli and the lateral tricuspid annulus were measured and termed as "septal PA," "lateral PA," and "right ventricular (RV) PA," respectively. The differences between lateral and septal PA, septal and RV PA, and lateral and RV PA were termed as "left intra-atrial EMD," "right intra-atrial EMD," and "inter-atrial EMD" respectively.

Results: Septal PA, lateral PA, RV PA, left and right intra-atrial EMDs, and inter-atrial EMD were not statistically different between these three groups. Furthermore, multivariable linear regression models, adjusted for potential confounders, showed that glycemic state was not associated with ACTs, left and right intra-atrial EMDs, and inter-atrial EMD.

Conclusion: There were no significant differences between the euglycemic, prediabetic, and diabetic patients with CAD regarding ACTs and atrial EMDs. *(Anatol J Cardiol 2017; 17: 374-80)*

Keywords: prediabetes, tissue Doppler echocardiography, atrial conduction time, coronary artery disease

Introduction

Prediabetes is a dysglycemic state where there is abnormal sugar metabolism, but it does not fulfill the criteria for diabetes mellitus (1). The American Diabetes Association has defined prediabetes as fasting plasma glucose levels between 100 mg/dL and 125 mg/dL or 2 hour plasma glucose after 75 g of oral glucose intake between 140 mg/dL and 199 mg/dL or hemoglobin A1c (HbA1c) between 5.7% and 6.4% (2). It has been estimated that by the year 2035, approximately 471 million people in the world will be affected by prediabetes (3). The conversion rate from prediabetes to diabetes has been reported to range between 4% and 19% (4). Prediabetes is associated with cardiovascular morbidity and mortality (5, 6). Furthermore, it has been shown that prediabetes is allied to subtle myocardial injury and dysfunction (7). Recently, left atrial (LA) and right atrial (RA) mechanical dysfunction has been demonstrated in prediabetes (8).

In patients with diabetes, the prevalence of atrial fibrillation is about 15%, and the hazard ratio of its occurrence is 1.5 compared with nondiabetic subjects (9, 10). Atrial conduction times (ACTs) evaluated using tissue Doppler imaging (TDI) are used as a noninvasive, helpful method for the evaluation of electrical and structural atrial remodeling (11–13). ACTs can also predict the occurrence of atrial fibrillation (14). ACTs measured using TDI have been evaluated in several conditions, such as diabetes mellitus (15–17), metabolic syndrome and insulin resistance state (18, 19), and obesity (20). The association between ACTs and inflammation and subclinical atherosclerosis in patients with type 2 diabetes has been demonstrated (16), whereas studies on ACTs in prediabetic patients are very limited (21). To the best of our knowledge, there are currently no studies on ACTs in patients with coronary artery disease (CAD) based on their glycemic state. On the basis of this evidence, we hypothesized that an aggravation in glycemic state is in tandem with prolonged ACTs in patients with significant CAD.

Methods

Study population

Between November 2015 and February 2016, we enrolled 128 consecutive patients admitted for coronary artery bypass graft-

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ing in our hospital. Sample venous blood was obtained after 12 hours of overnight fasting. These samples were analyzed for complete blood count and biochemistry tests. In total, 612 patients were excluded from our study. The exclusion criteria comprised history of myocardial infarction in the preceding 4 weeks, rhythms other than sinus rhythm, bundle branch block, cardiomyopathies, valvular stenosis of any degree, valvular regurgitation > mild degree, history of thyroid disease, left ventricular (LV) diastolic dysfunction > mild degree, LV ejection fraction <45%, history of cancer, HbA1c >11%, creatinine >1.5 mg/dL, history of hepatic diseases, type 1 diabetes mellitus, estimated systolic pulmonary artery pressure on echocardiography >35 mm Hg, and poor echocardiography window. Diabetes was defined as fasting blood sugar >126 mg/dL in two samples or using oral antidiabetic agents or insulin. Prediabetes was defined as HbA1c between 5.7% and 6.4%. Normal patients were defined as HbA1c <5.7%. The laboratory staff was blind to the echocardiography data. CAD burden was evaluated via a method previously described by Gensini et al. (22). In summary, 40 patients were allocated to the diabetic group, 48 patients to the prediabetic group, and 40 patients to the euglycemic group. The study was approved by the Institutional Review Board, and an informed consent was obtained from the patients.

Echocardiography

All echocardiographic examinations were performed and analyzed by an experienced echocardiologist, who was blind to the patients' glycemic state. Echocardiography was performed in the left lateral position by recording one lead of the electrocardiogram (ECG; Lead II). The gain of the ECG lead was adjusted to obtain the highest P wave. A commercial echocardiography setting (GE Medical Systems S5, U.S.A., Wauwatosa, WI, 2–4 MHz probe) was used for echocardiography. LV end-systolic and end-diastolic diameters and volumes, septal and posterior wall thickness, LA diameters, mean maximum LA volume in the 2- and 4-chamber views, biplane LV ejection fraction, mid-cavity (RV) diameter, tricuspid annular systolic plane excursion, maximum RA volume and LV mass, peak velocity of the mitral and tricuspid flow in early and late diastole (E wave and A wave, respectively), deceleration time of mitral E wave, peak systolic and diastolic waves of the pulmonary vein (S and D, respectively), peak velocities of the myocardium in systole and in early and late diastole (s′, e′, and a′) in the tricuspid annulus, and the septal and lateral sides of the mitral annulus were measured using pulsed-wave TDI according to the recommendations of the American Society of Echocardiography (ASE) (23, 24). All Doppler measurements in the left chambers were averaged in three cardiac cycles, and those in the right chambers were averaged in five cardiac cycles. Maximal effort was applied to achieve maximal alignment to the mentioned cardiac walls. The velocity scale of recording was between –20 cm/s and 20 cm/s. The horizontal sweep at analysis time was set at 67–100 mm/s. The gain was adjusted to obtain the least possible noise in the recording. The time interval between the beginning of the P wave in the surface ECG and the peak of the a′ wave (PA) was adopted as ACT (Fig. 1). ACTs at the tricuspid annulus and the septal and lateral mitral annuli were measured and termed as "RV PA," "septal PA," and "lateral PA," respectively. The differences between septal PA and RV PA, lateral and septal PA, and lateral and RV PA were calculated and termed as "right intra-atrial electromechanical delay (EMD)," "left intra-atrial EMD," and "inter-atrial EMD," respectively. The mean value of three consecutive cardiac cycles was used in the analysis. The average of e′ (septal and lateral) was computed, and E/e′ for LV with the peak velocity of the mitral E wave and the averaged e′ was calculated. E/e′ for RV was computed with the peak velocity of the tricuspid E wave and the e′ of the tricuspid annulus.

Statistical analysis

The categorical data are presented as frequencies and percentages. The continuous data are presented as means and standard deviations (SDs), if normally distributed; otherwise, they are presented as median and interquartile (25^{th}–75^{th}) ranges. The continuous data were compared between the euglycemic, prediabetic, and diabetic patients using the one-way analysis of variance or the nonparametric Kruskal–Wallis H test, as appropriate. The Bonferroni-adjusted method was employed for pairwise comparisons if the omnibus test was significant. Additionally, the categorical data were compared between the above-mentioned three groups using the χ^2 test or the Fisher exact test, as appropriate. Correlations were evaluated using the Pearson correlation coefficient. Because left and right intra-atrial EMDs and inter-atrial EMD were not normally distributed, we logarithmically transformed these variables for the purposes of normalization. Multivariable linear regression models were employed to determine the association of the glycemic state groups with ACTs, left and right intra-atrial EMDs, and inter-atrial EMD adjusted for heart rate, body mass index, cigarette smoking, RV diameter, averaged e′, and LV E/e′ ratio as the potential confounders. Data analysis was performed using IBM Statistical

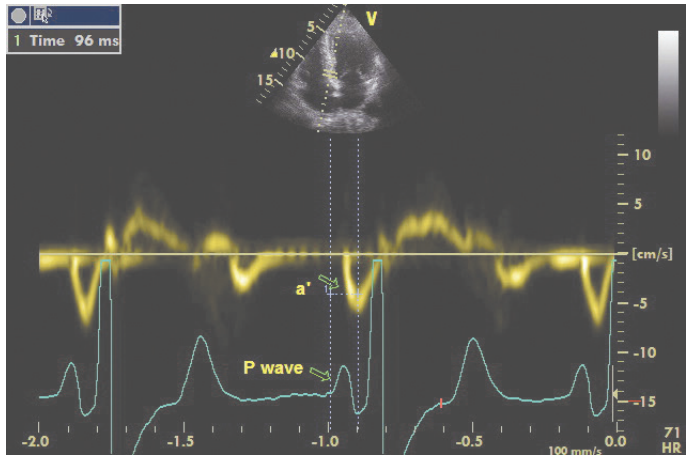

Figure 1. Measurement of atrial conduction times from the beginning of the *P* wave on the surface electrocardiogram to the peak of the a′ wave (PA) using tissue Doppler imaging

Table 1. Demographic, clinical, and laboratory data of the study groups categorized according to their glycemic state

Group variables	Euglycemic patients (n=40)	Prediabetic patients (n=48)	Diabetic patients (n=40)	*P*
Sex, male, n (%)	34 (85%)	38 (79%)	27 (68%)	0.162
Age, y	59.7±1.2	61.1±8.2	60.9±7.0	0.695
SBP, mm Hg	119.9±12.7	117.6±13.5	122.8±17.0	0.246
DBP, mm Hg	75.3±7.8	73.1±8.3	75.1±8.7	0.402
Heart rate, bpm	70.5±12.5	65.4±11.3	74.3±11.6	0.002*
BMI, kg/m^2	26.4±2.3	27.6±3.9	28.6±4.7	0.034**
Body surface area, m^2	1.8±0.1	1.8±0.2	1.8±0.2	0.814
NYHA class				
I, n (%)	3 (8%)	8 (17%)	6 (15%)	0.419
II, n (%)	31 (78%)	30 (63%)	26 (65%)	0.288
III, n (%)	6 (15%)	10 (21%)	8 (20%)	0.761
Hypertension, n (%)	16 (40%)	27 (56%)	24 (60%)	0.159
Dyslipidemia, n (%)	12 (30%)	20 (42%)	20 (50%)	0.187
Cigarette smoking, n (%)	9 (23%)	20 (42%)	21 (53%)	0.020
FH, n (%)	9 (23%)	18 (38%)	14 (35%)	0.288
ACE/ARB use, n (%)	24 (60%)	32 (67%)	28 (70%)	0.630
Calcium blocker use, n (%)	8 (20%)	5 (10%)	4 (10%)	0.319
Beta-blocker use, n (%)	18 (45%)	11 (23%)	12 (30%)	0.082
Statin use, n (%)	34 (85%)	37 (77%)	25 (63%)	0.061
Diuretic use, n (%)	3 (8%)	3 (6%)	7 (18%)	0.258
Nitrate use, n (%)	29 (73%)	35 (73%)	28 (70%)	0.950
Aspirin use, n (%)	23 (58%)	20 (42%)	19 (48%)	0.331
Number of diseased vessel				
One, n (%)	2 (5%)	4 (8%)	3 (8%)	0.911
Two, n (%)	8 (20%)	7 (15%)	7 (18%)	0.797
Three, n (%)	30 (75%)	37 (77%)	30 (75%)	0.965
LAD, n (%)	40 (100%)	48 (100%)	40 (100%)	>0.999
LCX, n (%)	36 (90%)	42 (88%)	33 (83%)	0.601
RCA, n (%)	32 (80%)	39 (81%)	34 (85%)	0.831
Gensini score	58.8 (40.3–91.3)	68.0 (48.0–99.0)	64.0 (48.4–85.3)	0.529
Hemoglobin level, mg/dL	14.8±1.6	14.7±1.5	14.0±1.5	0.070
FBS level, mg/dL	87.0±8.1	96.4±10.4	135.9±42.4	<0.001***
HbA1c, %	5.5±0.2	6.1±0.3	7.4±1.1	<0.001****
Serum triglyceride level, mg/dL	130.0 (91.3–173.0)	114.0 (90.5–183.5)	137.5 (94.3–252.8)	0.396
Serum cholesterol level, mg/dL	142.6±32.0	150.8±36.7	151.4±35.3	0.445
Serum HDL level, mg/dL	37.8±9.1	36.9±7.9	36.1±7.6	0.651
Serum LDL level, mg/dL	86.9±28.9	95.5±32.7	92.2±29.7	0.416
Serum urea level, mg/dL	35.8±10.9	35.6±8.5	37.7±10.8	0.591
Serum creatinine level, mg/dL	0.9±0.2	0.9±0.1	0.8±0.2	0.329

Data are presented as means±standard deviations for the normally distributed continuous variables, median and interquartile (25^{th}–75^{th}) ranges for the continuous variables with a skewed distribution, and frequencies (%) for the categorical variables. To compare the continuous data, we used the one-way analysis of variance or the nonparametric Kruskal–Wallis H test, as appropriate. The Bonferroni-adjusted method was employed for pairwise comparisons if the omnibus test was significant (defined in the following table with symbols). To compare the categorical data, we used the χ^2 test or the Fisher exact test, as appropriate. * - prediabetes vs. diabetes, *P* value =0.002; ** - euglycemia vs. diabetes, *P* value =0.029; *** - euglycemia vs. diabetes, *P* value <0.001; prediabetes vs. diabetes, *P* value <0.001; **** - euglycemia vs. diabetes, *P* value <0.001; prediabetes vs. diabetes, *P* value <0.001; euglycemia vs. diabetes; *P* value <0.001. ACEI/ARB - angiotensin-converting enzyme inhibitor/Angiotensin receptor blocker; BMI - body mass index; DBP - diastolic blood pressure; FBS - fasting blood sugar; FH - family history of coronary artery disease; HbA1c - glycated hemoglobin; HDL - high-density lipoprotein; LAD - left anterior descending artery; LCX - left circumflex artery; LDL - low-density lipoprotein; NYHA - New York Heart Association; RCA - right coronary artery; SBP - systolic blood pressure

Table 2. Standard echocardiography data of the study groups categorized according to their glycemic state

Group variables	Euglycemic patients (n=40)	Prediabetic patients (n=48)	Diabetic patients (n=40)	*P*
LVEDV index, mm^3/m^2	42.1±10.1	43.3±10.3	41.0±9.4	0.568
LVESV index, mm^3/m^2	18.3±4.9	18.1±5.5	18.6±5.3	0.925
LVEF, %	56.4±5.8	58.2±7.1	55.0±7.2	0.090
LA diameter, mm	36.5±3.7	35.8±3.7	35.8±3.5	0.607
Posterior wall thickness, mm	9.0±1.1	9.3±1.3	9.5±1.3	0.164
Interventricular septal thickness, mm	9.2±1.2	9.3±1.5	9.6±1.5	0.355
LV mass, g	154.0±39.3	151.4±45.6	157.4±42.6	0.808
LV mass index, g/m^2	84.3±20.5	83.4±22.4	87.6±22.6	0.645
RV diameter, mm	29.1±2.5	28.7±3.4	27.4±3.2	0.040*
TAPSE, mm	21.7±3.2	21.7±2.5	22.0±3.6	0.900
LAV index, mm^3/m^2	24.9±7.0	25.4±7.1	25.4±5.7	0.934
RAV index, mm^3/m^2	16.8 (14.4–22.8)	17.0 (13.6–21.8)	14.3 (10.6–20.1)	0.093
Mitral E, cm/s	63.8±16.9	60.1±14.3	63.9±17.0	0.438
Mitral A, cm/s	69.8±18.7	70.1±19.4	75.0±19.3	0.388
Mitral DT, ms	237.5±57.5	232.5±62.8	243.8±58.6	0.683
Mitral E/A ratio	0.86 (0.70–1.16)	0.82 (0.72–0.94)	0.82 (0.67–1.03)	0.587
S, cm/s	52.3±13.4	47.9±9.0	49.5±7.8	0.148
D, cm/s	36.8±10.2	34.4±8.0	33.1±7.0	0.162
S/D ratio	1.5±0.3	1.4±0.3	1.5±0.3	0.296
Tricuspid E, cm/s	37.0±9.7	35.4±6.8	36.3±7.8	0.653
Tricuspid A, cm/s	35.9±9.4	36.5±9.0	39.2±12.8	0.322
Tricuspid E/A ratio	1.1±0.2	1.0±0.3	1.0±0.2	0.392
SPAP, mm Hg	27.6±4.5	27.6±4.3	25.8±3.8	0.095
Septal s′, cm/s	7.4±1.1	7.0±1.3	6.8±1.1	0.092
Septal e′, cm/s	7.2±1.8	6.7±1.8	6.2±1.5	0.044**
Septal a′, cm/s	9.6±1.8	8.9±1.5	9.1±1.3	0.106
Septal e′/a′ ratio	0.8±0.3	0.8±0.2	0.7±0.2	0.166
Lateral s′, cm/s	8.4±2.3	7.7±2.0	8.1±1.5	0.286
Lateral e′, cm/s	9.5±1.9	8.9±2.2	8.2±1.8	0.015***
Lateral a′, cm/s	9.8±2.6	10.0±2.5	10.4±2.2	0.519
Lateral e′/a′ ratio	1.0 (0.8–1.1)	0.9 (0.7–1.1)	0.8 (0.7–1.0)	0.012****
Averaged septal and lateral e′, cm/s	8.3±1.6	7.8±1.8	7.2±1.5	0.010†
RV s′, cm/s	12.2±2.4	11.4±2.2	11.9±2.5	0.285
RV e′, cm/s	8.6±2.4	8.2±2.8	7.8±2.1	0.384
RV a′, cm/s	14.7±4.0	13.3±3.4	13.6±3.2	0.166
RV e′/a′ ratio	0.6 (0.5–0.7)	0.6 (0.5–0.8)	0.6 (0.5–0.7)	0.782
LV E/e′ ratio	7.8±2.1	8.0±2.3	9.0±1.8	0.024††
RV E/e′ ratio	4.2 (3.5–5.5)	4.6 (3.3–5.9)	4.4 (3.9–5.7)	0.642

Data are presented as means±standard deviations for the normally distributed continuous variables and median and interquartile (25^{th}–75^{th}) ranges for the continuous variables with a skewed distribution. To compare the continuous data, we used the one-way analysis of variance or the nonparametric Kruskal–Wallis H test, as appropriate. The Bonferroni-adjusted method was employed for pairwise comparisons if the omnibus test was significant (defined below the table with symbols). * - euglycemia vs. diabetes, *P* value =0.050; ** - euglycemia vs. diabetes, *P* value = 0.039; *** - euglycemia vs. diabetes, *P* value =0.012; **** euglycemia vs. diabetes, *P* value =0.007; † - euglycemia vs. diabetes, *P* value =0.012; †† - euglycemia vs. diabetes, *P* value =0.038. DT - deceleration time; LA - left atrium; LAV - left atrial volume; LV - left ventricle; LVEDV - left ventricular end-diastolic volume; LVEF - left ventricular ejection fraction; LVESV - left ventricular end-systolic volume; RAV - right atrial volume; RV - right ventricle; SPAP - systolic pulmonary artery pressure; TAPSE - tricuspid annular plane excursion

Package for the Social Sciences (SPSS) for Windows (version 23.0) (Armonk, NY: IBM Corp.).

Results

The clinical, laboratory, and demographic characteristics of our study patients are depicted in Table 1. These groups were significantly different in terms of body mass index, heart rate at the time of echocardiography, and cigarette smoking. The heart rate of prediabetic patients was higher than that of diabetic patients, and the body mass index of euglycemic patients was less than that of diabetic patients. The diabetes duration was 3.3 (0.5–7.0) years, and 31 (78%) diabetic patients used insulin or oral antidiabetic agents. The frequency of patients with 1-, 2-, and 3-vessel disease and the Gensini score as a marker of CAD burden were not significantly different between the groups. The baseline echocardiographic data are presented in Table 2. RV diameter, e′ septal, e′ lateral, e′/a′ ratio lateral, average of e′ septal and lateral, and LV E/e′ ratio were significantly different between the euglycemic and diabetic groups. There were no significant differences concerning septal PA, lateral PA, RV PA, left and right intra-atrial EMDs, and inter-atrial EMD (Table 3). Our multivariable analysis adjusted for potential confounders (Table 4) showed that glycemic state was not associated with septal PA, lateral PA, RV PA, right intra-atrial EMD, and inter-atrial EMD. The multivariable analysis was not significant regarding left intra-atrial EMD.

Discussion

Our study showed no significant differences apropos ACTs, left and right intra-atrial EMDs, and inter-atrial EMD according to the glycemic state in patients with CAD even after adjusting for potential confounders.

There have been several studies on ACTs evaluated using TDI in diabetic patients (15–17) and one study on ACTs in patients with impaired fasting glucose (21). Nevertheless, CAD was considered an exclusion criterion in these studies. There are, therefore, no data on ACTs evaluated using TDI in CAD patients with or without diabetes. In these studies, CAD was excluded only by the presence of a history of CAD, and the control groups comprised euglycemic and prediabetic patients. It has been

Table 3. Atrial conduction times, left and right intra-atrial electromechanical delays, and inter-atrial electromechanical delay of the study groups according to their glycemic state

Group variables	Euglycemic patients (n=40)	Prediabetic patients (n=48)	Diabetic patients (n=40)	*P*
Septal PA, ms	88.7±21.8	92.0±16.7	84.7±20.3	0.223
Lateral PA, ms	106.0±20.5	110.9±15.9	104.1±20.0	0.212
RV PA, ms	74.5±24.1	73.4±18.0	70.3±22.4	0.663
Lateral PA-Septal PA, ms	16.2 (10.3–22.3)	18.3 (12.3–23.9)	20.0 (12.0–24.9)	0.582
Septal PA-RV PA, ms	10.7 (5.0–18.8)	16.2 (10.1–26.2)	12.5 (5.5–22.3)	0.250
Lateral PA-RV PA, ms	31.8 (22.1–42.8)	36.7 (26.4–48.0)	32.0 (21.0–44.3)	0.183

Data are presented as means±standard deviations for the normally distributed continuous variables and median and interquartile (25^{th}–75^{th}) ranges for the continuous variables with a skewed distribution. To compare the continuous data, we used the one-way analysis of variance or the nonparametric Kruskal–Wallis H test, as appropriate. PA - the time interval from the onset of the *P* wave on the surface ECG to the peak of a′ in tissue Doppler imaging; RV - right ventricle

Table 4. Adjusted association between glycemic state, atrial conduction times, right intra-atrial electromechanical delay, and inter-atrial electromechanical delay

Variables	Septal PA		Lateral PA		RV PA		Log (Septal PA-RA PA)		Log (Lateral PA-RV PA)	
	β	*P*	β	*P*	β	*P*	β	*P*	β	*P*
Diabetes	-0.11	0.289	-0.07	0.497	-0.05	0.648	-0.12	0.249	-0.02	0.839
Prediabetes	0.01	0.920	0.04	0.722	-0. 10	0.352	0.14	0.172	0.20	0.065
Averaged e′	-0.45	<0.001	-0.45	<0.001	-0.24	0.017	-0.28	0.005	-0.22	0.034
LV E/e′	-0.27	0.006	-0.34	<0.001	-0.28	0.006	0.02	0.818	-0.01	0.899
Heart rate	-0.15	0.101	-0.22	0.016	-0.31	0.001	0.27	0.003	0.19	0.054
Body mass index	0.01	0.893	0.15	0.067	0.01	0.896	0.02	0.816	0.15	0.088
Cigarette smoking	-0.09	0.299	-0.16	0.061	-0.12	0.177	0.09	0.327	-0.03	0.722
RV diameter	0.04	0.675	-0.02	0.842	-0.12	0.187	0.23	0.012	0.09	0.368

Multivariable linear regression was used to determine the association between the glycemic state groups and ACTs and left and right intra-atrial electromechanical delays and inter-atrial electromechanical delay adjusted for the potential confounders. LV - left ventricle; PA - the time interval from the onset of the *P* wave on the surface electrocardiogram to the peak of a′ in tissue Doppler imaging; RV - right ventricle

shown that atrial size is correlated with ACTs (25), but RA size in these studies has not been reported. On the strength of such evidence, our study is the first of its kind to evaluate ACTs in patients having documented CAD with prediabetes compared with euglycemic and diabetic patients with CAD.

Akyel et al. (15) showed that septal PA, lateral PA, left and right intra-atrial EMDs, and inter-atrial EMD were prolonged in patients with type 2 diabetes compared with nondiabetic patients, while RV PA was similar. Most of the diabetic patients and control subjects had no LV diastolic dysfunction. As previously mentioned, patients with CAD were excluded by history taking; however, it is highly probable that such patients were indeed included in that study. Furthermore, the control patients comprised prediabetic and euglycemic patients, and the authors failed to mention the duration of diabetes and RA size.

Demir et al. (17) demonstrated that ACTs, left and right intra-EMDs, and inter-atrial EMD were prolonged in patients with type 2 diabetes. In their study, CAD was excluded only by history taking. The control subjects comprised prediabetic and euglycemic subjects. LA size was different between the two groups. Although the researchers tried to adjust ACTs according to LA size, they selected linear atrial diameters for adjustment. It would have been preferable had they adjusted ACTs according to the absolute LA volume. It seems that LV diastolic function was normal in most of the study patients. The authors failed to measure RA size.

Ayhan et al. (21) compared ACTs between a subgroup of patients with prediabetes (impaired fasting glucose) and euglycemic subjects and reported that septal PA, lateral PA, left intra-atrial EMD, and inter-atrial EMD were prolonged in the patients with impaired fasting glucose compared with the euglycemic subjects. CAD was excluded if there was a history of cardiovascular events. The authors did not clearly present the LV diastolic function of the subjects nor did they report RA size.

The differences between our results and those in the aforementioned studies can be explained by the following facts. First, all of our study patients had documented significant CAD, while the previous studies (15, 17, 21) have tried to exclude these patients, with the exclusion seeming unsatisfactory. In a recent study, it was demonstrated that LA mechanical function was not different between CAD patients with and without diabetes (26), which provides evidence in favor of our findings. Consequently, it can be hypothesized that the presence of an ischemic milieu modulates the effects of a dysglycemic state on ACTs, such that the difference due to dysglycemia cannot manifest itself. The interaction between an ischemic milieu and glycemic state vis-à-vis ACTs should be further evaluated by future studies. Second, while LV diastolic function in previous studies seems to be normal for most of the subjects, in our study, all of the patients had mild LV diastolic dysfunction. The interaction between diastolic function and glycemic state concerning ACTs merits in-depth analysis in future investigations. Third, in the aforesaid studies, ACTs were measured from the beginning of the P wave in the surface ECG to the beginning of the a′ wave in TDI, whereas we measured ACTs from the beginning of the P wave to the peak of a′ in TDI. The correlation between these two methods has not been studied yet. Furthermore, according to a report, the correlation between ACTs measured using TDI and invasive electrophysiology study is moderate (27).

Study limitations

The main limitation of our study is its small sample size. Also, it was not possible for us to follow up the patients apropos the occurrence of postoperative atrial fibrillation. We assessed ACTs using TDI, but the gold standard for the evaluation of ACTs is invasive electrophysiology study.

Conclusions

Our study showed that septal PA, lateral PA, RV PA, left and right intra-atrial EMDs, and inter-atrial EMD measured via pulsed-wave TDI were not significantly different among CAD patients categorized according to their glycemic state even after adjustment for potential confounders. These findings can be due to the presence of an ischemic milieu or LV diastolic dysfunction in all of our study groups. Larger studies for the evaluation of interaction between glycemic state, ischemia, and LV diastolic function are needed in the future for further clarification of these issues.

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