Journal of Cardiology 67 (2016) 378–383

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Assessment of atrial electromechanical delay and P-wave dispersion in patients with type 2 diabetes mellitus

Kenan Demir (M.D.)[a,*], Ahmet Avci (M.D.)[a], Zeynettin Kaya (M.D.)[b], Kamile Marakoglu (M.D.)[c], Esra Ceylan (M.D.)[c], Ahmet Yilmaz (M.D.)[a], Ahmet Ersecgin (M.D.)[a], Mustafa Armutlukuyu (M.D.)[c], Bulent Behlul Altunkeser (M.D.)[a]

[a] Selcuk University Faculty of Medicine, Cardiology Department, Konya, Turkey
[b] Mevlana University Faculty of Medicine, Cardiology Department, Konya, Turkey
[c] Selcuk University Faculty of Medicine, Department of Family Medicine, Konya, Turkey

ARTICLE INFO

Article history:
Received 31 March 2015
Received in revised form 4 June 2015
Accepted 7 June 2015
Available online 9 July 2015

Keywords:
Diabetes mellitus
Tissue Doppler imaging
Atrial electromechanical delay
P-wave dispersion

ABSTRACT

Objectives: Diabetes mellitus is an independent and strong risk factor for development of atrial fibrillation (AF). Electrophysiologic and electromechanical abnormalities are associated with a higher risk of AF. In this study we aimed to determine the correlation of atrial conduction abnormalities between the surface electrocardiographic and tissue Doppler echocardiographic measurements in type 2 diabetes mellitus (T2DM) patients.

Methods: A total of 88 consecutive T2DM patients and 49 age-, gender-, and body mass index-matched healthy volunteers were included in the present study. Baseline characteristics were recorded and 24-hour ambulatory blood pressure monitoring, transthoracic echocardiography, and 12-lead surface electrocardiography were performed for all study participants. Atrial electromechanical delay (EMD) intervals were measured.

Results: Maximum P-wave duration and P-wave dispersion (Pd) were significantly higher in patients with T2DM (105.7 ± 10.2 ms vs. 102.2 ± 7.5 ms, $p = 0.02$; 40.6 ± 7.6 ms vs. 33.6 ± 5.9 ms, $p < 0.001$, respectively). Interatrial, intraatrial, and intraleft atrial EMD were significantly higher in the T2DM patients when compared with the controls (16.5 ± 7.8 ms vs.11.2 ± 4.4 ms, $p < 0.001$; 9.0 ± 7.3 ms vs. 6.0 ± 3.8 ms, $p = 0.002$, and 7.4 ± 5.2 ms vs. 5.1 ± 3.2 ms, $p = 0.002$ respectively). Correlation analysis showed a positive correlation between interatrial EMD and Pd ($r = 0.429$, $p < 0.001$) and left atrial volume ($r = 0.428$, $p < 0.001$).

Conclusions: In this study, there was significant EMD and Pd in patients with T2DM as compared with healthy volunteers. Additionally, interatrial EMD was correlated with Pd and left atrial volume index.

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder and a major risk factor for cardiovascular disease. The frequency of cardiovascular disease, including coronary artery disease, systolic and diastolic heart failure, arrhythmia, and thromboembolism, is increased in diabetic patients. Atrial fibrillation (AF) is one of the most frequently sustained cardiac arrhythmias seen in clinical practice, and is associated with an increased risk of ischemic stroke, heart failure, and overall mortality. Many risk factors have been reported for the development of AF [1]. It was shown that DM is an independent and strong risk factor for development of AF. The frequency of AF development is 1.4- to 2.1-fold higher in cases with DM than cases without DM [2]. Inflammation and oxidative stress have been implicated in the pathogenesis of both the DM and AF [1].

Electrophysiologic and electromechanical (excitation–contraction coupling) abnormalities resulting from intraatrial and interatrial conduction disorders are associated with a higher risk of AF. The prolongation of intraatrial and interatrial electromechanical delay (EMD) and the inhomogeneous propagation of sinus impulses are well-known electrophysiological characteristics of the atria prone to fibrillation [3]. Atrial conduction abnormalities were evaluated with noninvasive techniques by using electrocardiography (ECG) and tissue Doppler imaging (TDI) in previous studies [4,5].

To our knowledge, there is no study that evaluated atrial conduction abnormalities using both TDI and ECG in patients with

* Corresponding author at: Selcuk University Faculty of Medicine, Cardiology Department, 42075 Konya, Turkey. Tel.: +90 505 506 1334; fax: +90 332 241 2151.
E-mail address: drkenan76@yahoo.com (K. Demir).

http://dx.doi.org/10.1016/j.jjcc.2015.06.003
0914-5087/

type 2 diabetes mellitus (T2DM). In this study we aimed to determine the correlation of atrial conduction abnormalities between the surface electrocardiographic and TDI measurements in T2DM patients.

Methods

Study population

A total of 88 consecutive T2DM patients (45 males, 43 females; mean age, 49.5 ± 7.9 years) and 49 age-, gender-, and body mass index-matched healthy volunteers (19 males, 30 females; mean age, 47.3 ± 9.2 years) were included between January 2014 and June 2014 to the present study. The study protocol was approved by our local ethics committee, and all participants gave their written informed consent to participate in the study.

The diagnosis of T2DM was made based on the criteria of the American Diabetes Association [6]. Medical history was obtained and physical examination was performed in all patients. All participants were evaluated with 24-hour blood pressure monitoring for diagnosing arterial hypertension. Patients and controls with a history of coronary artery disease, arterial hypertension, left ventricular (LV) wall motion, LV ejection fraction (EF) less than 50%, primary cardiomyopathy, valvular heart disease, paroxysmal AF, dysrhythmia, bundle branch block, atrioventricular conduction abnormalities on ECG, thyroid dysfunction, anemia, electrolyte imbalance, renal failure, pulmonary disease, and poor quality echocardiographic and ECG imaging were excluded from the study. All participants were in sinus rhythm and those who had been taking medications such as antiarrhythmics, tricyclic antidepressants, antihistaminics, and antipsychotics were excluded from the study.

Using standard laboratory methods, blood samples were drawn after an overnight 12-h fasting to determine levels of hemogram and biochemical parameters.

Echocardiography

All echocardiographic examinations (1.5–4.6 MHz phased array transducer, Vivid E9; GE, Horten, Norway) were performed by a cardiologist who was blinded to the clinical details and results of the other investigations of each patient and control. ECG (DII) was recorded continuously during echocardiography. M-mode measurements and conventional Doppler echocardiographic examinations were performed according to the criteria of the American Society of Echocardiography guidelines [7]. Three consecutive cycles were averaged for every parameter. Left atrium (LA) dimension and LV end-systolic and end-diastolic diameters were measured. LV ejection fraction was estimated by modified Simpson's rule. LV mass was calculated with Devereux formula and indexed to body surface area. LA volume was calculated at end systole of the LV in the apical four- and two-chamber views using the methods of disks (Simpson's rule). LA volume was indexed to the body surface area.

Transmitral pulsed-wave Doppler velocities were recorded from the apical four-chamber view with the Doppler sample placed between the tips of the mitral leaflets. Early (E) and late (A) wave velocities, E/A ratio, E deceleration time (DT), and isovolumetric relaxation time (IVRT), isovolumetric contraction time (IVCT), and ejection time (ET) were measured from the mitral inflow profile.

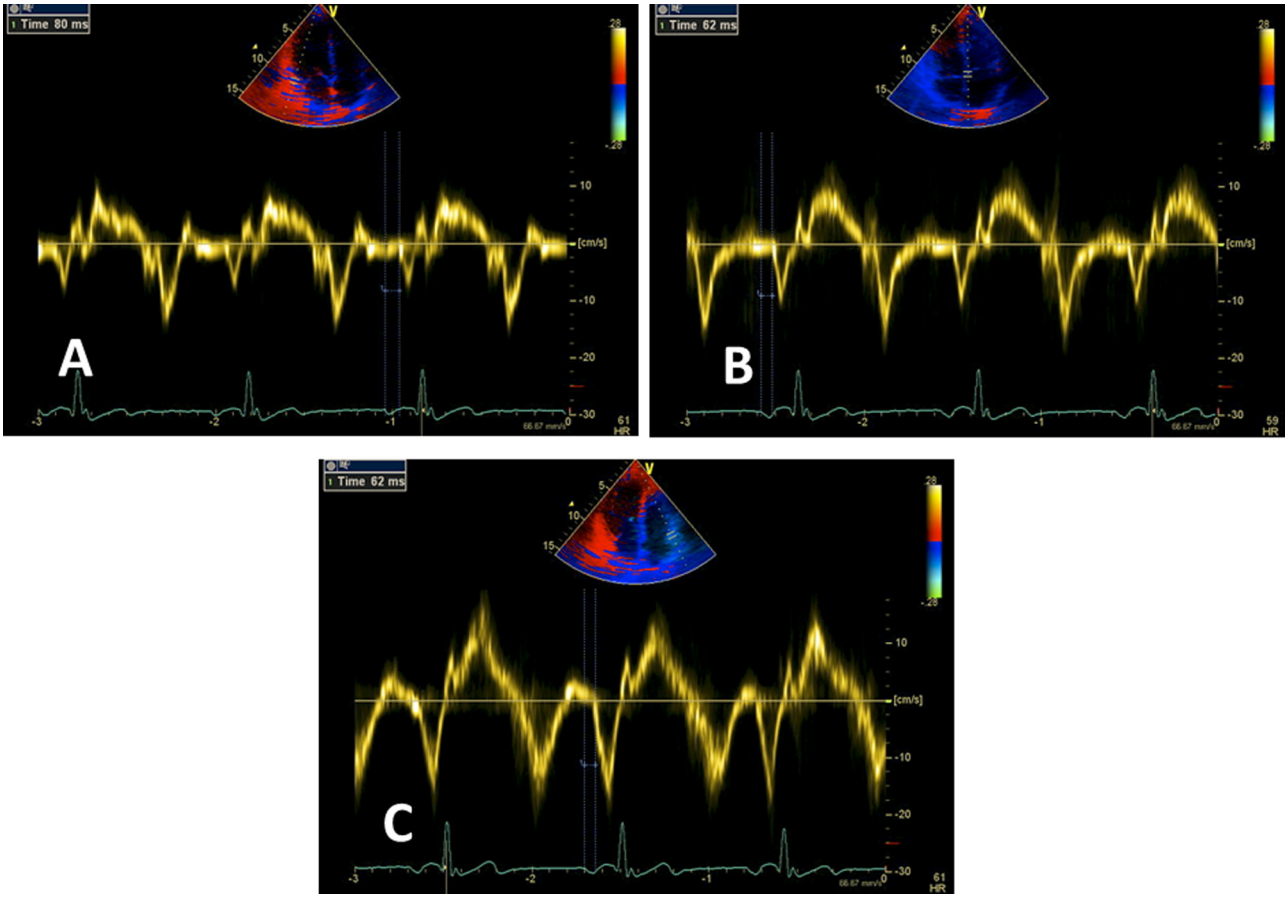

Fig. 1. Measurement of time interval from the onset of P-wave on surface electrocardiogram to the beginning of Am wave (PA) interval with tissue Doppler echocardiography. (A) Lateral PA; (B) Septal PA; (C) Tricuspid PA.