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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Atrial electromechanical delay analysed by tissue Doppler echocardiography is prolonged in patients with generalised anxiety disorders

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Abstract

Background: It has been shown that psychological status is associated with the likelihood of atrial fibrillation (AF). Prolongation of the duration of atrial electromechanical delay (AEMD) is known to be a precursor for AF development.

Aim: Therefore, we aimed to evaluate AEMD in patients with anxiety disorder.

Methods: In this prospective study, a total of 82 anxiety disorder and 80 healthy subjects were enrolled. Symptoms of anxiety were evaluated by using the Hamilton Anxiety Rating Scale (HAM-A). P-wave dispersion (PWD) was measured on a 12-lead electrocardiogram. Both intra- and inter-AEMD were measured with tissue Doppler imaging.

Results: Basal characteristics were similar between the two groups. PWD, inter- and right intra-AEMD were significantly prolonged in patients with anxiety disorders, compared to the control group ($p < 0.05$). In the correlation analysis, HAM-A was significantly and moderately correlated with right intra- and inter-AEMD, and PWD.

Conclusions: Patients suffering from anxiety disorders are characterised by prolonged AEMD, which can provide significant contributions to evaluate the risk for AF development in this group.

Key words: atrial fibrillation, atrial electromechanical delay, tissue Doppler imaging, anxiety disorders

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INTRODUCTION

Atrial fibrillation (AF) is the most commonly sustained arrhythmia globally and results in significantly increased morbidity and mortality, including a fivefold risk of stroke [1]. Recent studies have shown that stress, anxiety, sadness, and anger each increase the likelihood of AF development, whereas happiness is protective [2]. Although the exact pathophysiological mechanism between anxiety disorders and AF is not well defined, it may be associated with increased atrial automaticity and atrial electromechanical delay (AEMD) in patients with anxiety disorders [3].

Prolonged intra- and inter-AEMD measured by tissue Doppler imaging (TDI) has been shown to be a precursor for AF development [4]. It is well correlated with invasive electrophysiological measurements, and hence this method can be a useful technique to assess atrial conduction times [5]. To our knowledge, there is no study in the literature evaluating the intra- and inter-AEMD in patients with anxiety disorders.

Because it is well-known that AF has numerous devastating complications, early recognition of risk factors for AF is very important for prevention and treatment of AF. Therefore, we aimed to investigate AEMD in patients with anxiety disorders.

METHODS

Study population

Eighty-two newly diagnosed anxiety disorder patients and 80 age- and sex-matched healthy volunteers were prospectively enrolled to the study. Patients were involved in the

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study from an out-patient psychiatry clinic. We used the criteria listed in the diagnostic and statistical manual of mental disorders (DSM-5), published by the American Psychiatric Association, to diagnose patients with generalised anxiety disorder. We performed an anxiety rating scale to measure generalised anxiety. The control group consisted of healthy volunteers who were normal according to the result of psychological evaluation.

In total 28 subjects were excluded from study due to the following criteria. Subjects with right bundle or left bundle branch block (n = 0), AF (n = 0), supraventricular tachycardia (n = 0), ventricular arrhythmia (n = 0), pacemaker implantation (n = 0), hypertension (n = 1), diabetes mellitus (n = 7), coronary artery disease (CAD) (n = 2), heart failure (n = 2), moderate or severe valvular heart disease (n = 4), peripheral artery disease (n = 0), chronic obstructive pulmonary disease (n = 4), obstructive sleep apnoea syndrome (n = 3), malignancies (n = 2), renal or liver disease (n = 0), any rheumatological or endocrinological disease (n = 0), using beta-blockers, digitalis, or anti-arrhythmic drugs (n = 3), and using medication for anxiety disorders or any other psychiatric diseases (n = 0) were excluded. The presence of supraventricular and/or ventricular arrhythmia were excluded with the history and electrocardiographic examination of the patients.

Demographic and clinical information, and psychiatric examination was recorded on the day of echocardiographic and electrocardiographic evaluation. Body weight (kg) and height (m) were determined, and body mass index (BMI) was calculated. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured for all study participants.

Symptoms of anxiety were evaluated by using the Hamilton Anxiety Rating Scale (HAM-A) [6]. Symptoms of anxiety were measured using 14 anxiety questions scored 0–4 based on recent symptoms. The results of each question were totalled to measure generalised anxiety, with higher scores linked to greater anxiety (range 0–56). Anxiety severity was classified as mild (HAM-A: 0–17), mild to moderate (HAM-A: 18–24), and moderate to severe (HAM-A: 25–30).

Using standard laboratory methods, blood samples were drawn after a 12-h fasting period to determine levels of biochemistry and cholesterol panels and fasting plasma glucose concentrations.

Transthoracic echocardiography

Tissue Doppler echocardiography (Vivid 7 pro) was performed with transducer frequencies of 3.5–4.0 MHz by adjusting the spectral pulsed Doppler signal filters until a Nyquist limit of 15–20 cm/s was reached and using the minimal optimal gain. Echocardiographic examination was performed using a GE Vingmed Vivid 7 (GE Vingmed Ultrasound, Horten, Norway) echocardiography device and a GE Vingmed Vivid 3 system (GE Vingmed Ultrasound AS) with a 3.5–4.0 MHz frequency transducer by a single cardiologist blind to the patient characteristics and clinical data. In all examinations, patients were placed in the left lateral decubitus position. Parasternal long- and short-axis views and apical views were used as classical echocardiographic windows. Dimensions of the cardiac chambers were performed on M-mode traces recorded from the parasternal long-axis view according to the criteria of the American Society of Echocardiography guidelines [7]. Ejection fraction was estimated by Simpson's rule. During echocardiography, a one-lead electrocardiogram (ECG) was recorded continuously. All echocardiographic images contained at least three consecutive beats, and they were digitally stored for further analysis. All echocardiographic images were analysed by an experienced cardiologist who was blinded to the patients' characteristics. The early (E) and late (A) wave velocities and E/A ratio were measured with pulsed wave Doppler from the mitral inflow profile in apical four-chamber view. TDI was performed with transducer frequencies of 3.5–4.0 MHz by adjusting the spectral pulsed Doppler signal filters until a Nyquist limit of 15–20 cm/s was reached, and by using the minimal optimal gain. In the apical four-chamber view, the pulsed Doppler sample volume was placed at the level of the left ventricular (LV) lateral mitral annulus, septal mitral annulus, and right ventricular (RV) tricuspid annulus. AEMD was defined as the time delay beginning from the initial point of the P wave on the surface ECG to the initial point of the A wave, and designated as the PA interval (Fig. 1). This time interval was obtained from the lateral mitral annulus (lateral PA), septal mitral annulus (septal PA), and lateral tricuspid annulus (tricuspid PA), respectively. Lateral PA to tricuspid PA interval was defined as inter-AEMD; lateral PA to septal PA interval was defined as left intra-AEMD; and septal PA to tricuspid PA interval was defined as right intra-AEMD.

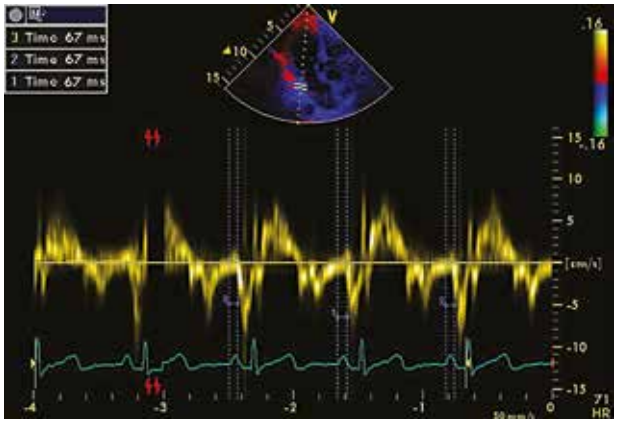

Figure 1. Measurement of time interval from the onset of P-wave on surface electrocardiogram to the beginning of A-wave interval with tissue Doppler imaging

Intra-observer variability was evaluated in 20 randomly selected participants, including 10 from the anxiety disorders

Table 1. Basal characteristics of study participants

	Control group (n = 80)	Anxiety disorders group (n = 82)	p
Age [years]	41.6 ± 9	39 ± 8.4	0.169
Gender (female)	22 (53%)	31 (75%)	0.064
Body mass index [kg/m^2]	29.2 ± 4.3	30.5 ± 4.2	0.193
Systolic BP [mm Hg]	112 ± 11	113 ± 10	0.693
Diastolic BP [mm Hg]	72.5 ± 7.1	72 ± 6.8	0.937
Heart rate [bpm]	77 ± 9	77 ± 8	0.941
Haemoglobin [g/dL]	14.3 ± 1.5	14.1 ± 1.4	0.408
Creatinine [mg/dL]	0.84 ± 1.4	0.91 ± 1.5	0.055
Fasting plasma glucose [mg/dL]	100 ± 11.4	101 ± 7.7	0.973
Total cholesterol [mg/dL]	197.3 ± 49.8	186.8 ± 29.6	0.237
LDL-cholesterol [mg/dL]	114.5 ± 40.5	109.9 ± 29.5	0.559
HDL-cholesterol [mg/dL]	43.6 ± 4.8	44.3 ± 3.5	0.503
Triglyceride [mg/dL]	141 ± 79.5	123.1 ± 62.4	0.263
HAM-A	–	12.9 ± 5.4	–

BP — blood pressure; HDL — high-density lipoprotein; LDL — low-density lipoprotein; HAM-A — Hamilton Anxiety Rating Scale

group and 10 from control group, by repeating the measurements under similar basal conditions as for the anxiety disorders. Intra-observer variability was 3.8% for lateral PA, 3.6% for septal PA, and 4% for tricuspid PA, respectively.

Electrocardiography

Twelve-lead surface ECGs were obtained for each subject in the supine position at a paper speed of 50 mm/s and voltage of 20 mm/mV. It was used to measure the maximum (Pmax) and minimum (Pmin) P-wave durations. Mean values for at least three complexes were calculated in each lead. The difference between the Pmax and the Pmin was calculated and defined as P-wave dispersion (PWD) (PWD = Pmax – Pmin). The PWD was measured manually.

The study protocol was approved by the local Ethics Committee of Ankara Education and Research Hospital, and written informed consent was obtained from study participants.

Statistical analysis

All statistical studies were carried out using the SPSS software package (version 17.0; SPSS, Chicago, Illinois, USA). As the result of post-hoc power calculation using the link http://clincalc.com/Stats/Power.aspx, the power of our study was 82.5% (α = 0.5 and β = 0.1). Distribution properties of the data were performed using the Kolmogorov-Smirnov test. Normally distributed data were shown as mean ± standard deviation. Comparisons of continuous values between two groups were performed by Independent-Samples T test. Categorical variables were compared using the χ^2 test or Fisher's exact test. Results were shown as a percentage. Correlation was examined using Pearson's correlation. Intra-observer agreement was assessed with correlation coefficient, and with the average difference between readings, corrected for their mean (variability). A p value less than 0.05 was considered statistically significant.

RESULTS

The basal characteristics and laboratory parameters of the anxiety disorder and control patients are summarised in Table 1. Age, gender, BMI, heart rate, laboratory parameters, SBP, and DBP were similar in both groups.

The basal echocardiographic parameters of the two groups were similar (Table 2). Biventricular and valvular functions were also in normal reference ranges for both study groups. The findings of pulsed wave Doppler, atrial electromechanical conduction times measured by TDI and electrocardiography are represented in Table 3. Both the mean lateral PA interval (64.6 ± 6.8 vs. 69.9 ± 5.6 ms, p < 0.001) and mean septal PA interval (49.4 ± 6.4 vs. 53.1 ± 6.7 ms, p < 0.012) were significantly prolonged in patients with anxiety disorder compared to healthy control subjects (Table 3). However, the tricuspid PA interval was similar between the study groups (p = 0.418). PWD was also significantly higher in anxiety disorder patients (p < 0.001) (Table 3). As presented in Figure 2, inter-AEMD was significantly higher in the patient group. Similarly, right intra-AEMD was prolonged in patients with anxiety disorder (Fig. 2). However, left intra-AEMD was similar between the study groups (p = 0.135) (Fig. 2).

Table 2. Echocardiographic findings of study participants

Characteristic	Control group (n = 80)	Anxiety disorders group (n = 82)	p
LVEF [%]	64.3 ± 3.4	64.8 ± 3.2	0.575
LVEDD [cm]	4.39 ± 0.33	4.34 ± 0.29	0.529
Left atrial size [cm]	3.09 ± 0.30	3.14 ± 0.33	0.460
LVSWT [cm]	0.94 ± 0.11	0.96 ± 0.10	0.267
LVPWT [cm]	0.87 ± 0.11	0.89 ± 0.08	0.321

LVEF — left ventricular ejection fraction; LVEDD — left ventricular end-diastolic diameter; LVSWT — left ventricular septal wall thickness; LVPWT — left ventricular posterior wall thickness

Table 3. Echocardiographic pulsed wave, tissue Doppler, and electrocardiographic variables findings of study participants

	Control group (n = 80)	Anxiety disorders group (n = 82)	p
E-wave velocity [m/s]	0.82 ± 0.19	0.79 ± 0.12	0.415
A-wave velocity [m/s]	0.80 ± 0.11	0.77 ± 0.13	0.174
E'-wave velocity [cm/s]	10.4 ± 1.2	10.7 ± 1.4	0.317
A'-wave velocity [cm/s]	9.1 ± 1.8	9.2 ± 1.4	0.679
E/A	1.03 ± 0.27	1.06 ± 0.23	0.710
E/E'	7.9 ± 2.1	7.5 ± 1.6	0.274
Mitral PA interval [ms]	64.6 ± 6.8	69.9 ± 5.6	< 0.001
Septal PA interval [ms]	49.4 ± 6.4	53.1 ± 6.7	0.012
Tricuspid PA interval [ms]	41.6 ± 6.1	40.3 ± 7.3	0.418
Pmax [ms]	101.7 ± 6.9	108 ± 6.3	< 0.001
Pmin [ms]	69.7 ± 6.6	64 ± 5.2	< 0.001
P-wave dispersion [ms]	31.9 ± 6.2	44 ± 7.3	< 0.001

PA — time interval from the onset of P-wave on surface electrocardiogram to the beginning of A-wave interval with tissue Doppler echocardiography

Correlation analysis

The correlation analysis of inter- and right intra-AEMD with HAM-A is shown in Figure 3. There was a significant and moderate correlation between HAM-A and inter-, right intra--AEMD ($r = 0.483$, $p < 0.0001$; $r = 0.429$, $p < 0.0001$, respectively). Thus, PWD was positively and moderately correlated with HAM-A ($r = 0.328$, $p < 0.0001$) (Fig. 3).

DISCUSSION

This study demonstrated that patients with anxiety disorders had prolonged AEMD and increased PWD compared to age- and gender-matched controls. To our knowledge, this is the first study in the literature demonstrating an association between anxiety disorders and AEMD, which can be defined as a precursor for AF development.

Anxiety disorder is one of the most commonly encountered chronic diseases in clinical course [8]. Several studies have indicated an association between anxiety and cardiovascular risk factors and CAD [9]. Anxiety seems to be an adequate predictor of CAD in conjunction with depression [10]. Phobic anxiety was found to be significantly related with a nearly fourfold increase in the relative risk of fatal CAD [11, 12]. Chronic emotional distress, especially type-D personality pattern, characterised by increased negative affectivity and social inhibition, was found to be predictive of worse outcomes in patients with CAD [13]. In addition to CAD, acute and chronic stress is also associated with fatal arrhythmias and sudden cardiac death [14, 15]. Lown and DeSilva [16] suggested that stress was a precipitant of ventricular premature contractions with ventricular fibrillation, and Lampert et al. [17] demonstrated that emotions could precipitate ventricular arrhythmias. In the literature there are studies demonstrating a link between anxiety disorders and atrial arrhythmias. Tully et al. [18] demonstrated that anxiety symptoms in the postoperative period were associated with AF. Lampert et al. [2] showed that stress, anxiety, sadness, and anger each increased the likelihood of AF, whereas happiness was protective. However, there is little information about the link between anxiety disorders and AF development.

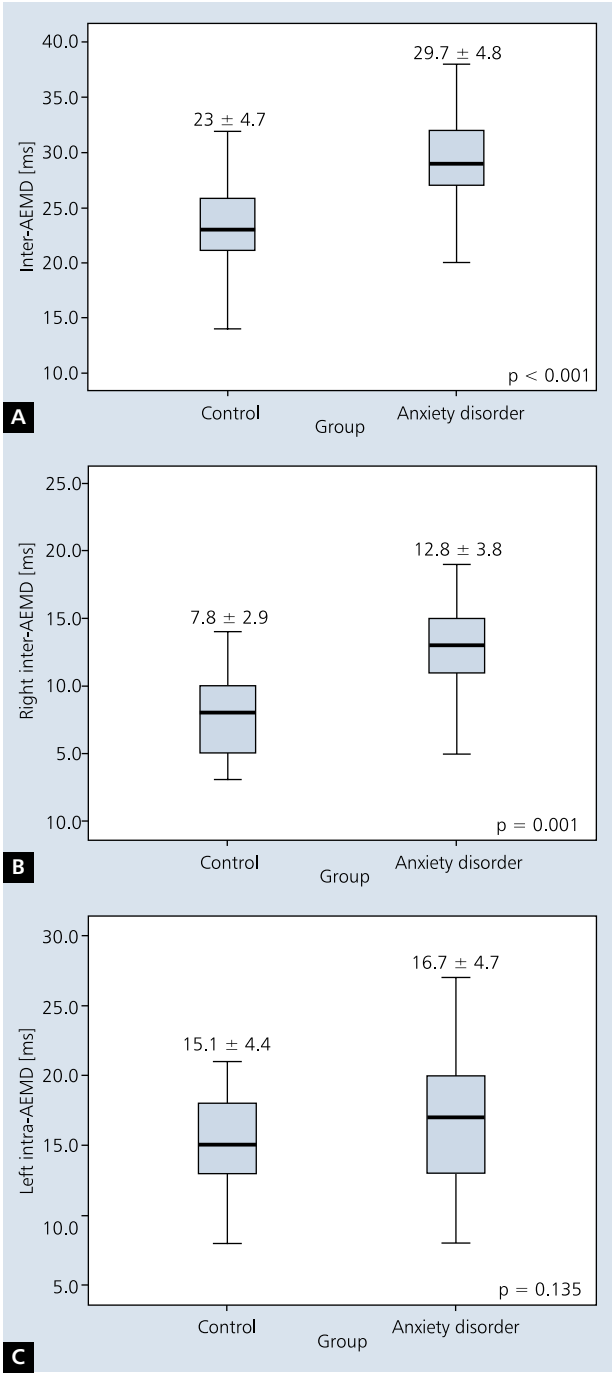

Figure 2. **A.** Inter-atrial electromechanical delay (AEMD) time of anxiety disorder group versus control group; **B.** Right intra-AEMD time of anxiety disorder group versus control group; **C.** Left Intra-AEMD time of anxiety disorder group versus control group

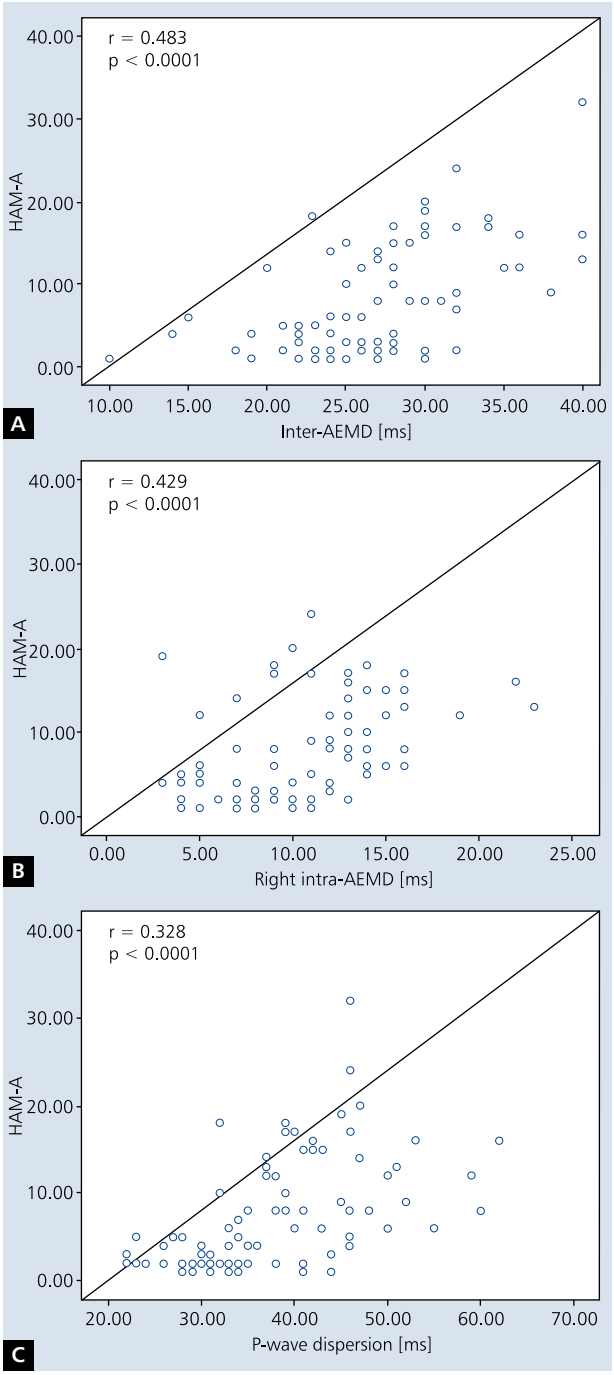

Figure 3. **A.** Positive correlation between inter-atrial electromechanical delay (AEMD) and Hamilton Anxiety Rating scale (HAM-A), between right intra-AEMD and HAM-A (**B**), and between P-wave dispersion and HAM-A (**C**)

Atrial electromechanical delay with TDI echocardiography is a non-invasive method that is an alternative to invasive electrophysiological studies [4]. Prolongation in inter- and intra-AEMD times and non-homogeneous distribution of sinus impulses are well-known features of the AF-vulnerable atrium [4, 19]. This method can be used to predict paroxysmal AF. The distance between the two atria is a significant determinative factor for atrial conduction, and inter-nodal pathways may play a major role in inter-AEMD [20]. Previous studies demonstrated that AEMD durations were significantly

prolonged in patients with paroxysmal AF [4, 19]. Roshanali et al. [21] found that the atrial electromechanical interval was a predictor of AF emerging after coronary artery bypass graft (CABG) and showed that the preoperative administration of amiodarone to patients having a longer atrial electromechanical interval decreased the postoperative AF incidence. These studies showed that a prolonged electromechanical interval seemed to reflect atrial remodelling for an arrhythmogenic substrate [19, 21].

This is the first study demonstrating increased AEMD in patients with anxiety disorder, which in turn may cause AF development in the future. Although the exact mechanism of prolongation in AEMD duration is not known, the dysregulation of the sympathetic nervous system demonstrated in patients with anxiety disorder might be a possible reason for the prolongation of AEMD durations [18, 22]. It is well known that a consistent endocrine system plays a significant role in the overall coherence and normal functioning of the cardiovascular system. Stressful experiences and emotions impress the heart directly through the autonomic nervous system and indirectly through neuroendocrine pathways [12]. Psychological stress results in hypothalamic-adrenocortical and sympathoadrenal hyperactivity [23]. This situation results in an increase in corticosteroid and catecholamine levels leading to adrenergic hyperactivity with a resulting increase in myocardial irritability and facilitation of the stimulation pro-arrhythmic effect [23]. Specifically, an increased sympathetic nervous-system response as a result of weak vagal tone can increase catecholamine levels, and thus disrupt refractoriness or local reentry atrial wavelets, directly or indirectly initiating AF during emotional or mental stress [18].

In addition, we have also demonstrated that PWD is prolonged in patients with anxiety disorder, and this was positively related with the anxiety score of the patients. P-wave duration and PWD are important predictors of atrial arrhythmias, including AF [24]. Yavuzkir et al. [25] showed that PWD on ECG was increased among patients with panic disorder, compared with a control group, as a predictor of AF after CABG [18].

Limitations of the study

The present study has certain limitations. First, the participant number is relatively low. Second, we lacked follow-up data for future arrhythmic events in our study population. Further studies with greater sample size and a longer follow-up period are needed to clear our findings. Third, our study is limited by the lack of invasive correlation for prolongation in AEMD durations. Fourth, we excluded arrhythmia by using ECG recordings and history of the study participants instead of using rhythm Holter monitoring. And finally, we did not measure blood corticosteroid levels to monitor adrenergic hyperactivity. Further studies may aim to investigate the association between anxiety disorders and atrial wall abnormalities.

CONCLUSIONS

In the present study, prolonged intra- and inter-AEMD and increased PWD duration were demonstrated in patients with anxiety disorders. Moreover, these abnormalities were positively correlated with HAM-A, which indicated classified severity of the disease. Our results could contribute to identify patients at high risk for AF development. Thus, further investigation and closer clinical follow-up can be performed to identify paroxysmal AF in this high-risk group of patients.

Conflict of interest: *none declared*

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Zwiększenie przedsionkowego opóźnienia elektromechanicznego analizowanego za pomocą tkankowej echokardiografii doplerowskiej u chorych z uogólnionymi zaburzeniami lękowymi

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Streszczenie

Wstęp: Wykazano, że stan psychiczny chorych wiąże się z ryzykiem wystąpienia migotania przedsionków (AF). Wydłużenie czasu przedsionkowego opóźnienia elektromechanicznego (AEMD) jest uważane za czynnik poprzedzający rozwój AF.

Cel: Celem niniejszej pracy była ocena AEMD u chorych z zaburzeniami lękowymi.

Metody: Do prospektywnego badania włączono 82 pacjentów z zaburzeniami lękowymi i 80 osób zdrowych. Objawy lęku oceniano za pomocą skali Hamiltona (HAM-A). Dyspersję załamka P (PWD) mierzono na 12-odprowadzeniowym elektrokardiogramie, a między- i wewnątrzprzedsionkowe opóźnienie elektromechaniczne — za pomocą doplera tkankowego.

Wyniki: Początkowe parametry były podobne w obu grupach. PWD, opóźnienie elektromechaniczne międzyprzedsionkowe i wewnątrz prawego przedsionka było istotnie większe u chorych z zaburzeniami lękowymi niż u osób z grupy kontrolnej ($p < 0,05$). W analizie korelacji ocena w skali HAM-A była istotnie i umiarkowanie związana z wewnątrz- i międzyprzedsionkowym opóźnieniem elektromechanicznym oraz z PWD.

Wnioski: Chorzy z zaburzeniami lękowymi charakteryzują się wydłużonym AEMD. Ta obserwacja może istotnie przyczynić się do oceny ryzyka rozwoju AF w tej grupie pacjentów.

Słowa kluczowe: migotanie przedsionków, przedsionkowe opóźnienie elektromechaniczne, obrazowanie metodą doplera tkankowego, zaburzenia lękowe

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