The relationship between atrial electromechanical delay and P-wave dispersion with the presence and severity of metabolic syndrome

Metabolik sendrom varlığı ve ciddiyeti ile atriyuma ait elektromekanik gecikme ve P dalga dispersiyonu arasındaki ilişki

Mustafa Kurt, M.D., İbrahim Halil Tanboğa, M.D.,# Mehmet Fatih Karakaş, M.D., Eyüp Büyükkaya, M.D., Adnan Burak Akcay, M.D., Nihat Şen, M.D., Emine Bilen, M.D.*

Department of Cardiology, Mustafa Kemal University Faculty of Medicine, Hatay;
#Department of Cardiology, Atatürk University Faculty of Medicine, Erzurum;
*Department of Cardiology, Ankara Atatürk Education and Research Hospital, Ankara

ABSTRACT

Objectives: In this study, we aimed to investigate the association between the presence and severity of metabolic syndrome (MetS) with intra- and inter-atrial electromechanical delay (AEMD) and P-wave dispersion (PWD).

Study design: A total of 144 patients (72 MetS patients and 72 age- and sex-matched control subjects) were included in the study. Patients with MetS were classified into three groups based on the number of MetS criteria as follows: Group 1 (patients with three MetS criteria), Group 2 (patients with four MetS criteria) and Group 3 (patients with five MetS criteria). Intra- and inter-AEMD were measured from parameters of tissue Doppler imaging. PWD was calculated from the 12-lead electrocardiogram.

Results: Both inter-AEMD (22.9±15 *vs.* 11.5±14, p<0.001) and intra-AEMD (23.6±12 *vs.* 8.3±19, p<0.001) were found to be significantly longer in patients with MetS than the control group. Similarly, PWD (49±25 *vs.* 36±24, p=0.001) were found to be significantly longer in the MetS patients than the controls. However, both inter-AEMD and intra-AEMD and P wave measurements were not found to be associated with the severity of MetS. While inter and intra-AEMD were better correlated with LV mass index and LA volume index, PWD correlated better with mitral inflow Doppler parameters. According to multivariate analyses, inter-AEMD, HDL-C, and systolic and diastolic blood pressure were found to be independent predictors, whereas E/A and LDL-C had borderline significance. For the intra-AEMD, systolic and diastolic blood pressure, body mass index and E/A were found to be independent predictors.

Conclusion: In patients with MetS, inter- and intra-AEMD, and P dispersion were found to be lengthened when compared with the controls. However, these parameters were not associated with the severity of MetS.

ÖZET

Amaç: Bu çalışmada, atriyum içi ve atriyumlar arası elektromekanik gecikme (AEMG) ve P dalga dispersiyonu (PDD) ile metabolik sendrom (MetS) varlığı ve şiddeti arasındaki ilişki incelendi.

Çalışma planı: Çalışmaya MetS olan (n=72) ve olmayan (kontrol grubu, n=72) toplam 144 hasta alındı. MetS ciddiyetinin belirlenmesi için hastalar MetS ölçütlerinin sayısına göre üç gruba ayrıldı: Grup 1 (üç ölçütlü hastalar), Grup 2 (dört ölçütlü hastalar) ve Grup 3 (beş ölçütlü hastalar). Hastaların 12 derivasyonlu elekrokardiyografilerinden PDD ve doku Doppler parametrelerinden kulakçıklar arası ve kulakçıklar içi AEMG hesaplandı.

Bulgular: Kulakçılar arası AEMG (22.9±15 ve 11.5±14, p<0.001) ve kulakçık içi AEMG değerleri (23.6±12 ve 8.3±19, p<0.001) MetS'li hastalarda, kontrol grubuna göre anlamlı olarak daha uzun bulundu. Benzer şekilde, PDD değerleri kontrol grubu ile karşılaştırıldığında MetS'li hastalarda anlamlı olarak daha uzun bulundu (49±25 ve 36±24, p=0.001). Ancak, kulakçıklar arası ve içi AEMG ve PDD'nin MetS şiddeti ile ilişkisi gösterilemedi. Korelasyon analizinde, atriyumlar arası AEMG ve atriyum içi AEMG daha çok sol ventrikül kitle indeksi ve sol atriyum hacim indeksi ile, P dalga dispersiyonu ise daha çok mitral Doppler parametreleri ile ilişkili bulundu. Çoklu değişken analizi sonucu, atriyumlar arası AEMD için, HDL-K, sistolik ve diyastolik kan basıncı bağımsız öngördürücüler olarak bulunurken; E/A ve LDL için bu değerler istatistiksel anlamlılık sınırında kaldı. Kulakçık içi AEMD için ise sistolik ve diyastolik kan basıncı, beden kitle indeksi ve E/A bağımsız öngördürücüler olarak bulundu.

Sonuç: MetS'li hastalarda kulakçıklar arası ve kulakçık içi AEMG ve PDD, kontrol grubuna kıyasla daha uzundur. Fakat bu uzamanın MetS ciddiyeti ile ilişkisi yoktur.

Received: May 28, 2012 *Accepted:* August 09, 2012

Correspondence: Dr. Mustafa Kurt. Tayfur Sökmen Kampüsü Tıp Fakültesi Sağlık Uygulama ve Araştırma Hastanesi, Serinyol, Antakya.
Tel: +90 326 - 229 10 00 e-mail: drmustafakurt@yahoo.com

Metabolic syndrome (MetS) is defined as a clinical entity which starts with insulin resistance and includes atherosclerotic risk factors such as abdominal obesity, glucose intolerance or diabetes mellitus, dyslipidemia and hypertension. MetS is highly prevalent in the general population, affecting about 22% of adults.[1] There is an association of MetS and MetS components separately with atrial fibrillation (AF), which is known to be associated with increased cardiovascular mortality including heart failure and ischemic stroke.[2-5] Heterogeneous spread of sinus impulses is shown to be related to atrial arrhythmias. Indices of the spread of sinus impulses are assessed non-invasively by ECG and color tissue Doppler imaging. The intra-atrial and inter-atrial electromechanical coupling intervals and P-wave dispersion (PWD) have been found to be prolonged in cases with paroxysmal AF in some studies.[6]

In the present study, we aimed to evaluate the association between the presence and severity of MetS with intra-atrial and inter-atrial electromechanical coupling intervals and PWD.

PATIENTS AND METHODS

Seventy-two patients who presented to the cardiology outpatient clinic from March 1, 2011 to March 15, 2011 and had a diagnosis of MetS, and 72 age- and sex-matched controls without MetS and admitted to the outpatient clinic, were included in the study. Patients with a previous history of myocardial infarction, AF, left ventricular systolic dysfunction, moderate to severe valvular heart disease, chronic obstructive pulmonary disease, pre-excitation syndromes, atrioventricular conduction abnormalities, left bundle branch block, thyroid diseases or previously implanted cardiac pacemakers were excluded. The protocol was approved by the local ethical committee, and all enrolled subjects gave informed written consent.

Definition of metabolic syndrome

MetS was diagnosed according to the National Cholesterol Education Program Adult Treatment Panel III criteria 3 (NCEP ATP 3). These criteria require the presence of three or more of the following: (1) abdominal obesity (waist circumference (WC) >102 cm in men and >88 cm in women); (2) a high triglyceride (TG) level (>150 mg/dl); (3) a low high-density lipoprotein (HDL) cholesterol level (<40 mg/dl for men and <50 mg/dl for women); (4) a high blood pressure (BP) (systolic ≥130 mmHg or diastolic ≥85 mmHg, or use of an antihypertensive medication); and (5) a high fasting plasma glucose (FBG) concentration (>100 mg/dl). Patients with MetS were classified into three groups based on the number of MetS criteria they displayed: Group 1 (patients with three MetS criteria), Group 2 (patients with four MetS criteria) and Group 3 (patients with five MetS criteria).[7]

Abbreviations:

AEMD	*Atrial electromechanical coupling delay*
AF	*Atrial fibrillation*
EF	*Ejection fraction*
MetS	*Metabolic syndrome*
TDI	*Tissue Doppler imaging*
PWD	*P-wave dispersion*

Electrocardiographic measurements

Twelve-lead electrocardiograms (ECG) were obtained from each subject in the supine position at a paper speed of 50 mm/s and signal size of 10 mm/mV standardization. P wave duration was measured manually with the use of a caliper by two cardiologists who were blind to the results of echocardiography and clinical data. Subjects with measurable P waves in nine or fewer electrocardiographic leads were excluded from the study. P wave duration was measured in all leads, with the beginning of the P wave defined as the point where the first atrial deflection crossed the isoelectric line. The end of the P wave was defined as a point where the atrial deflection returned to the isoelectric line. The difference between maximum and minimum P wave duration (Pmax and Pmin) was defined as the PWD.[8] If the onset and termination of the P wave could not be identified in a particular lead, this lead was excluded from analysis.

Echocardiographic measurements

Echocardiography was performed using a GE Vivid 7 system (GE Vingmed Ultrasound AS, Norten, Norway) with a 2.5 MHz phased-array transducer. All measurements were made by two investigators blinded to the clinical data of the subjects. The left ventricular ejection fraction (EF) was calculated by the biplane Simpson's method. Interventricular septum, posterior wall thickness, left ventricular end-diastolic diameter, and left atrial volume index were also measured. The mitral inflow velocity pattern was recorded from the apical four chamber view with the pulsed-wave Doppler sample volume positioned at the tips of the mitral leaflets during diastole. Peak early diastolic

Table 1. Baseline clinical characteristics of control and MetS groups

	Control (n=72)		MetS (n=72)		*p*
	%	Mean±SD	%	Mean±SD	
Age (year)		50±12		50±10	0.983
Sex (male)	37		31		0.381
Smoking	44		51		0.404
Systolic blood pressure (mmHg)		126±17		137±16	**<0.001**
Diastolic blood pressure (mmHg)		80±8.4		87±8.1	**<0.001**
Body mass index (kg/m^2)		24.3±3.5		30.0±5.0	**<0.001**
Waist circumference (cm)		87.9±9.9		100.9±8.6	**<0.001**
Fasting blood glucose (mg/dl)		92±14		115±29	**<0.001**
HDL-C (mg/dl)		45.2±8.9		36.0±8.1	**<0.001**
LDL-C (mg/dl)		99.8±29.8		116.8±27.1	**<0.001**
Triglyceride (mg/dl)		127.5±61.1		231.1±114.2	**<0.001**

MetS: Metabolic syndrome; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol.

velocity (E), peak late diastolic velocity (A), deceleration time and isovolumetric relaxation time were measured. The early diastolic annular velocity (E') was measured by means of tissue Doppler imaging (TDI) at the septal border of the mitral annulus.

TDI was performed in the apical four-chamber view with a 2.5-mm sample volume. The LV lateral mitral annulus, septal mitral annulus, and tricuspid lateral annulus were evaluated by TDI, and the myocardial velocity curves were constructed from digitized images in the apical four-chamber view for the assessment of atrial electromechanical coupling delay (AEMD). AEMD (the time interval from the onset of the P wave on the ECG to the beginning of the late diastolic wave [Am wave]) was calculated from the lateral mitral annulus, septal mitral annulus, and lateral tricuspid annulus and named as an interval from the onset of the P wave to the onset of the Am wave (PA) lateral, PA septum, and PA tricuspid, respectively. Values were averaged over three consecutive beats. The difference between the PA lateral and PA tricuspid (PA lateral - PA tricuspid) was defined as the inter-AEMD, and the difference between the PA septum and PA tricuspid (PA septum - PA tricuspid) was defined as the intra-AEMD.[9]

Statistics

Continuous variables are expressed as mean±SD. Categorical variables are expressed as percentages. To compare parametric continuous variables, the Student's t test or analysis of variance were used. In order to compare nonparametric continuous variables, the Mann-Whitney U or Kruskall-Wallis tests were used. To compare categorical variables the chi-square test was used. Correlations between variables were tested by the Pearson correlation test for normally distributed variables and with Spearman correlation tests for the non-normally distributed variables. In order to determine the independent predictors of inter- and intra-AEM, uni- and multivariate analysis were performed. The parameters that were found to have significance ($p<0.10$) in the univariate analysis were evaluated by stepwise logistic regression analysis. Ninety five percent confidence interval and Odds ratios (OR) were presented together. Two-tailed *p* values <0.05 were considered to indicate statistical significance. Statistical analyses were performed using SPSS, version 15.0 for Windows.

RESULTS

The study population comprised of 72 MetS patients (50±10, 31% male) and 72 controls (50±12, 37% male). Baseline differences between the MetS group and the control group are demonstrated in Table 1 and Table 2. Both inter-AEMD (22.9±15 *vs.* 11.5±14, $p<0.001$) and intra-AEMD (23.6±12 *vs.* 8.3±19, $p<0.001$) were found to be significantly increased in patients with MetS when compared to the control group (Fig. 1). Similarly, Pmax (112±26 *vs.* 96±24, $p<0.001$) and PWD (49±25 *vs.* 36±24, $p=0.001$) were

Table 2. Baseline echocardiographic and electrocardiographic characteristics of Control and MetS groups

	Control (n=72)	MetS (n=72)	*p*
E/A ratio	1.50±0.74	1.18±0.57	0.005
E/Em ratio	12.5±4.9	15.3±9.5	0.027
LV-Mass-Index (gr/m^2)	105±20	130±24	**<0.001**
LA-Volume-Index (ml/m^2)	22±5.9	33±6.5	**<0.001**
LV-Ejection fraction (%)	63±4.6	61±5.4	0.05
PA-Lateral (ms)	53±13	74±12	**<0.001**
PA-Septal (ms)	44±10	51±10	**<0.001**
PA-Right (ms)	33±10	28±10	0.003
Inter-AEMD (ms)	11.5±14	22.9±15	**<0.001**
Intra-AEMD (ms)	8.3±19	23.6±12	**<0.001**
Maximum P wave (ms)	96±24	112±26	**<0.001**
Minimum P wave (ms)	60±19	62±23	0.518
P-wave dispersion (ms)	36±24	49±25	**0.001**

MetS: Metabolic syndrome; AEMD: Atrial electromechanical delay; LA: Left atrium; LV: Left ventricle.

found to be significantly increased in the MetS patients when compared to controls. Subgroup analysis revealed that both inter- and intra-AEMD were not found to be associated with the severity of MetS. Similarly, P-max, P-min, and PWD were not found to be associated with the severity of MetS (Table 3).

The correlation analysis for E/A, E/E`, left atrial volume index, and left ventricular mass index with inter- and intra-AEMD, Pmax, Pmin, and P dispersion are shown in Table 4. The correlation analysis revealed that while inter- and intra-AEMD were better correlated with left ventricular mass index and left atrial volume index (Fig. 2 and Fig. 3), PWD had a better correlation with mitral inflow Doppler parameters (Fig. 4).

In order to determine the independent predictors of inter- and intra-AEMD, uni- and multivariate analysis were performed. According to those analyses, for inter-AEMD, HDL, systolic and diastolic BP were

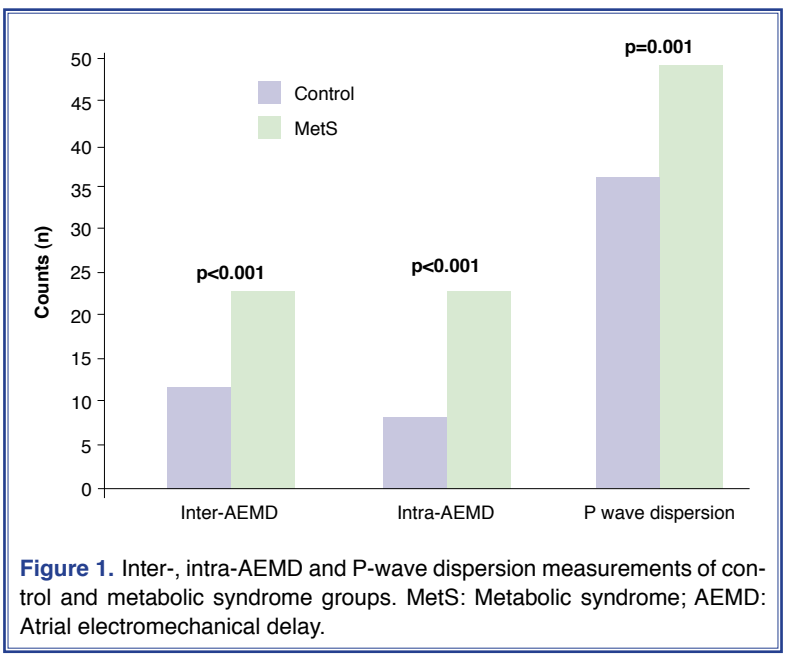

Figure 1. Inter-, intra-AEMD and P-wave dispersion measurements of control and metabolic syndrome groups. MetS: Metabolic syndrome; AEMD: Atrial electromechanical delay.

Table 3. AEMD and P wave measurements of MetS subgroups

	Group 1	Group 2	Group 3	*p*
Inter-AEMD	20±16	21±17	27±10	0.148
Intra-AEMD	19±13	22±12	28±11	0.103
P-maximum	114±32	107±25	116±24	0.419
P-minimum	67±24	55±22	68±23	0.077
P-dispersion	48±33	52±23	48±21	0.770

AEMD: Atrial electromechanical delay; MetS: Metabolic syndrome.

Table 4. Correlation analysis of inter- and intra-AEMD and P-wave dispersion with E/A, E/E`, left atrial volume index and left ventricular volume index

	Inter-AEMD		Intra-AEMD		P-dispersion	
	r	*p*	r	*p*	r	*p*
E/A	0.05	0.58	-0.17	0.04	-0.19	0.02
E/E`	0.04	0.60	0.02	0.82	0.08	0.35
Left atrial volume index	0.26	0.002	0.15	0.06	0.03	0.71
Left ventricular volume index	0.26	0.002	0.35	<0.001	0.11	0.16

AEMD: Atrial electromechanical delay; MetS: Metabolic syndrome.

found to be independent predictors whereas, E/A and LDL were borderline significant. The analyses for intra-AEMD and systolic and diastolic BP showed that body mass index and E/A were found to be independent predictors (Table 5).

DISCUSSION

The major findings of the present study were a) MetS patients without atrial arrhythmia displayed an increased PWD and significant intra- and inter-atrial electromechanical delay which was assessed by tissue Doppler echocardiography and b) these prolonged intra- and inter-atrial electromechanical delays and increased PWD were not associated with the severity of MetS which was defined by the number of MetS components.

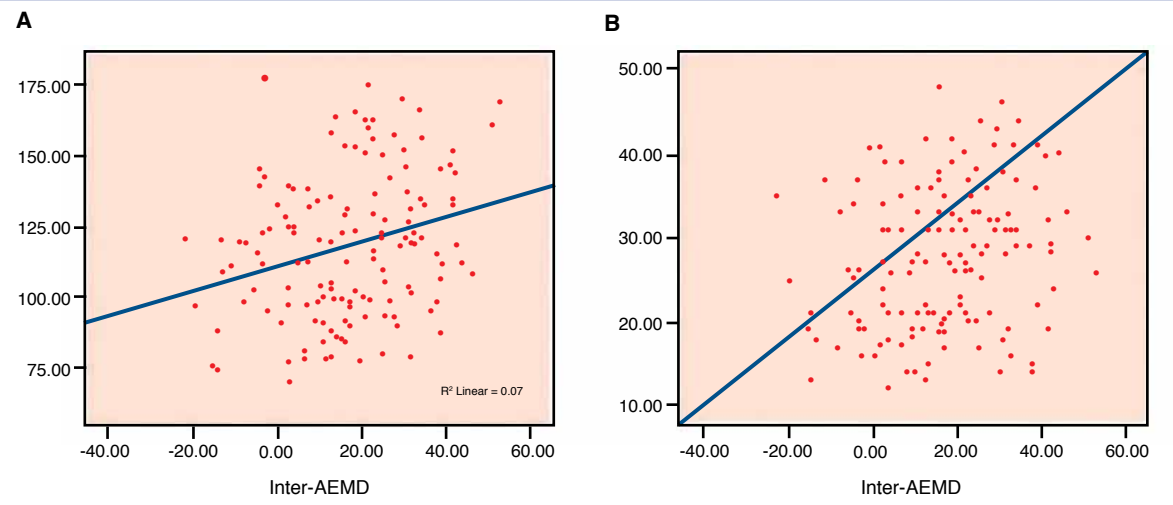

Figure 2. Correlation plot for inter-AEMD with **(A)** left atrial volume index and **(B)** left ventricular volume index.

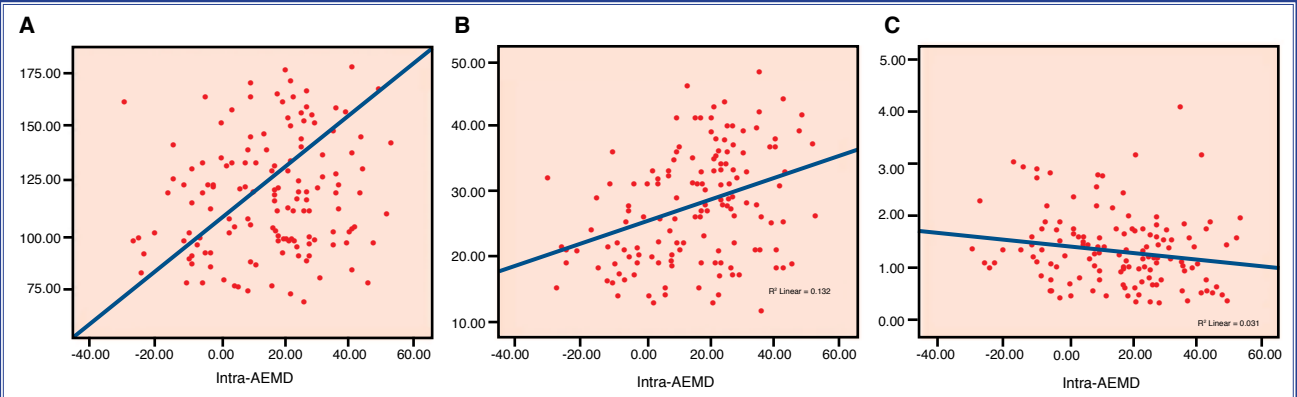

Figure 3. Correlation plot for intra-AEMD with **(A)** left atrial volume index, **(B)** left ventricular volume index and **(C)** E/A.

The incidence of AF is increased in patients with MetS. In the ARIC study, after a follow-up period of 19 years, patients with MetS were found to have a 67% increase in the incidence of AF. The increase in the AF risk was correlated with all MetS components, and the most powerful correlation was found to be with hypertension.[10] In another study, all MetS components were independently correlated with the increase in the AF risk. However, in this study, the most powerful correlation was found to be with high plasma glucose whereas the least important correlation occurred with central obesity.[11] Left atrial tension and dilation, autonomic nervous system activation, inflammation and oxidative stress have been reported as the responsible factors for the development of AF.[12-15] Obesity might cause this dysrhythmia through left atrial dilation resulting from increased blood volume, ventricular diastolic dysfunction and neurohormonal activation or increased oxidative stress.[16-18] A possible explanation for the diabetes mellitus to cause predisposition to AF may be left ventricular hypertrophy, myocardial ischemia or fibrosis.[19] Also, an explanation for the elevated levels of HDL-C, cholesterol and triglycerides to cause predisposition to AF may be inflammation and oxidative stress. HT is one of the most frequently seen etiological factors causing AF. Left ventricular hypertrophy and diastolic dysfunction may cause LA dilation and AF.[20] These findings demonstrate that MetS may result in electrical and structural remodeling in the atria.

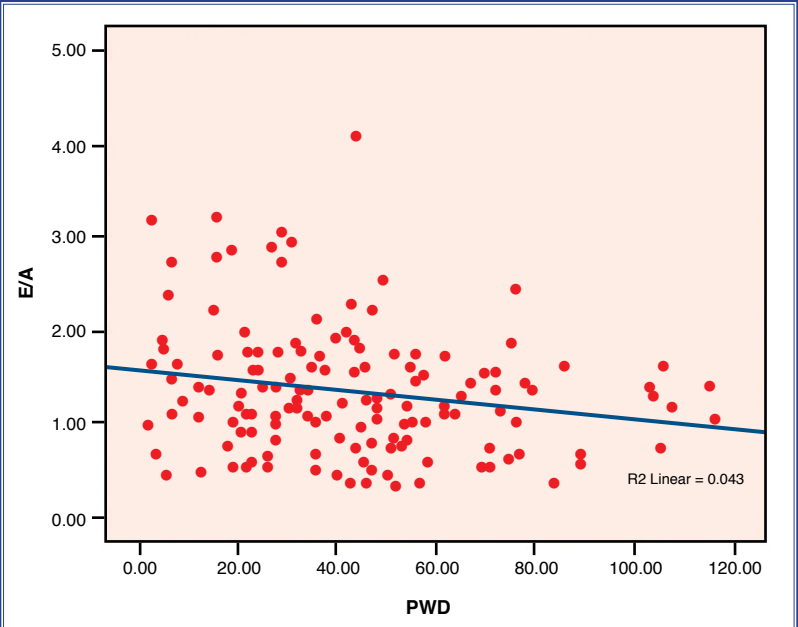

Figure 4. Correlation plot for P-wave dispersion with E/A.

The main electrophysiological features that lead to predisposition to AF are intra- and inter-AEMD and the heterogeneous spread of the sinus impulses.[21] Prolongation of inter- and intra-atrial conduction times in patients with paroxysmal AF were reported to be greater compared to the controls in previous studies. In our study, we demonstrated that PA-lateral, PA-septal, and PA-tricuspid atrial electromechanical coupling times, and intra- and inter-atrial electromechanical delay were lengthened in patients with MetS. However, the severity of MetS, defined with the number of MetS components, was not correlated with atrial dyssynchrony. Vyssoulis et al.[11] reported that the prevalence of AF was progressively increasing with the

Table 5. Independent predictors of inter- and intra-AEMD in multiple linear regression analysis

	Inter-AEMD			Intra-AEMD		
	ß coefficient ± SE	ß	*p*	ß coefficient ± SE	ß	*p*
Systolic BP (mmHg)	-0.31±0.12	-0.37	0.013	0.42±0.14	0.43	0.004
Diastolic BP (mmHg)	0.70±0.24	0.42	0.004	-0.71±0.29	-0.36	0.016
E/A ratio	3.67±1.93	0.16	0.060	-4.7±2.3	-0.18	0.043
Body mass index (kg/m^2)	–	–	–	0.85±0.41	0.25	0.038
LDL-C (mg/dl)	0.09±0.05	0.18	0.051	–	–	–
HDL-C (mg/dl)	-0.32±0.14	-0.21	0.024	–	–	–

AEMD: Atrial electromechanical delay; SE: Standard error; BP: Blood pressure; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol.

severity of MetS. In addition, we found that the left ventricular diastolic functional parameters such as mitral E/A, E/E', LA volume index, and left ventricular mass index were deteriorated in the MetS group when compared to the control group. Ultimately, increased left ventricular filling pressures may cause atrial fibrosis.[22] This situation is a contributing factor that leads to the lengthening of atrial activation time. In a study where atrial synchronization was evaluated in MetS patients with insulin resistance, only the HOMA index was found to be an independent predictor of deteriorated right-left atrium and inter-atrial dyssynchrony in multivariate analysis.[23] Subgroup analysis revealed that, there was no difference in the atrial synchrony in MetS patients with regard to the presence of HT. They concluded that hemodynamics may not play a major role in atrial electrical remodeling in MetS. Differently, we found that left ventricular filling pressure, left atrial volume index, which reflects long term deteriorated left ventricular diastolic function, and left ventricular mass index were increased in our patient population. The left ventricular diastolic dysfunction and an increase in the LA volume might change the geometry of atrial fibrils, which in turn can lead to intra- and inter-atrial dyssynchrony.

In addition to the increased intra- and inter-atrial electromechanical delay in MetS, we found that PWD was increased as well. PWD is defined as the difference between maximum and minimum p wave duration and it is shown to be a simple and non-invasive indicator for atrial arrhythmias.[24] In our study, we found an increase in PWD in MetS patients; however this increase was not associated with the severity of MetS. There are few studies regarding PWD in MetS. Yasar et al.[25] reported that PWD was increased in MetS patients when compared to controls. Similarly, Hanci et al.[26] reported that MetS patients had higher PWD values in the preoperative period.

Limitations

The major limitation of the present study is the absence of a prospective follow-up for arrhythmic incidents. For this reason, it is unknown whether intra- and inter-atrial dyssynchrony would predict atrial arrhythmia in the patients. One other limitation of the study is the lack of quantitative data about insulin resistance such as HOMA and fasting insulin level. ATPIII criteria were used in the selection of MetS patients; therefore, measuring the insulin resistance separately may be more appropriate.

Conclusion

In the current study we found that in MetS patients without atrial arrhythmia, both intra- and inter-atrial synchrony was impaired and this was not related to the severity of MetS. We proposed that this atrial electrical heterogeneity may be a result of hemodynamic disturbances which are related to left ventricular diastolic functions.

Conflict-of-interest issues regarding the authorship or article: None declared

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Key words: Arrhythmias, cardiac; electrocardiography; heart conduction system; metabolic syndrome X.

Anahtar sözcükler: Aritmi, kardiyak; elektrokardiyografi; kalp iletim sistemi; metabolik sendrom X.