

Body composition indices and electromechanical durations in metabolic syndrome

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ABSTRACT

Aim: To examine electromechanical delay (EMD) a predictive of atrial fibrillation (AF) risk, in obese and non-obese metabolic syndrome (MS) patients and to research the relationship between EMD and the new body composition indices.

Method: 118 overweight (body mass index (BMI)>26 kg/m²) individuals with MS meeting the criteria for IDF and ATP III were included in the study. The subjects were divided into two groups: Group 1; 54 obese persons with MS (33 females; mean age 46.2±14.1) while Group 2 included 64 non-obese persons with MS (33 females; mean age 41.4±13.2). In addition to laboratory findings, body composition indices, conventional and tissue Doppler imaging were obtained. Atrial EMD was defined as the time interval from the onset of the P wave on the surface electrocardiogram to the beginning of the late diastolic A wave. Inter, intra and left atrial EMD's were measured from the data obtained.

Results: Inter, intra and left atrial EMDs were significantly greater in the obese group compared to the non-obese group. There was a significant correlation between interatrial EMD and left atrial EMD and all body composition indices except body surface index. The strongest correlation was between body roundness index (BRI) and interatrial and left atrial EMD ($r=0.46$; $p<0.001$, $r=0.39$; $p<0.001$, respectively).

Conclusions: EMD intervals were more prolonged in obese subjects with MS than in non-obese subjects with MS. The parameter most relevant to EMD was BRI. BRI is the body composition index most correlated with increased risk for AF in persons with MS.

Key words: Metabolic syndrome, obesity, electromechanical coupling, body roundness index.

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Introduction

Metabolic Syndrome (MS) is a metabolic function disorder attributable to ectopic lipid accumulation, insulin resistance and obesity [1]. Obesity, one of the components of MS, is one of the key causes of mortality, morbidity

and cardiovascular diseases in adults. Its incidence has been rising in both developed and developing countries [2]. With this growth has come an increase in comorbid diseases [3]. In addition to being an independent risk factor for ischemic heart diseases and heart failure, the obesity in metabolic syndrome increases the risk of supraventricular arrhythmias such as atrial fibrillation [4,5]. Because they can be utilized easily in the assessment of obesity, anthropometric measures are the accepted indicators employed in epidemiological studies. The Body Mass Index (BMI) is acknowledged

as a diagnostic index for general obesity. While they reflect the general distribution of body fat, [6] it is also known that traditional anthropometric indices like BMI and waist circumference (WC) are not successful in making a distinction between fat and body mass [7,8]. Therefore, it has become clear that there is a need for more appropriate anthropometric indices that can measure body type and central obesity and predict disease [9]. Relatively new body composition indices such as a body shape index (ABSI), visceral adiposity index (VAI) and body roundness index (BRI) have been shown to be better correlated with visceral fat tissue, and therefore, better able to predict subclinical cardiovascular disease [10,11].

It is known that a rise in the percent of body fat is associated with a hike in arrhythmogenic risk. It has been reported that for every 5 unit increase in BMI the risk of atrial fibrillation increases by 29% [12]. Atrial fibrillation (AF) is the arrhythmia most commonly seen in clinical practice. Prolonged intra and inter electromechanical conduction times, which are obtained using the tissue Doppler method in transthoracic echocardiograms, are thought to be indicators of the occurrence of AF [13].

This study examined the electromechanical coupling durations, which predict AF risk, in obese subjects with metabolic syndrome and overweight, but non-obese, subjects using the tissue Doppler method. It assessed the degree of obesity by using such conventional indices as BMI and WC, as well as the relatively new body composition indices (BCI), and then evaluated the relation between BCI and EMD.

Materials and methods

Patient selection

118 volunteer patients (58 female, 60 male; mean age 42.7 ± 14.2 years) who came to the Istanbul Training and Research Hospital's

family practice and internal diseases polyclinics with any kind of complaint between April 2018 and December 2018 were included in the study. Patients had given signed consent were included in the study. Patients who had chronic diseases other than Type 2 diabetes, hypertension (HT) and dyslipidemia, such as kidney failure, heart failure, coronary artery disease, AF, malignancy, rheumatological diseases, those with findings of acute infection, a history of acute vascular events, were pregnant, with known genetic diseases, and uncontrolled thyroid function tests, as well as those taking medication that could affect cardiac functions were excluded from the study. Patients meeting the study's criteria were selected at random. A complete patient history was taken from all participants and detailed physical examinations were performed on them. Systolic and diastolic blood pressure and pulse readings were done after having the subject rest for 5 minutes in a seated position. All participants in the study were required to sign an informed consent form. The experimental protocols and the process for obtaining informed consent (in human studies) were approved by the appropriate institutional review committee. The study was begun after having received local Ethic Committee consent (decision no. 1124, dated 06.04.2018).

Definition of metabolic syndrome

We defined metabolic syndrome according to IDF and ATP-III criteria. Among the criteria for MS are the presence of central obesity (in men, the ATP-III-MS criterion is a waist circumference > 102 cm and in women, a waist circumference of > 80 cm), hyperglycemia (a fasting plasma glucose of ≥ 100 mg/dl (5.6 mmol/L)), low HDL-C (HDL-C ≤ 40 mg/dl (1.03 mmol/L) in men and ≤ 50 mg/dl (1.29 mmol/L) in women), hypertriglyceridemia

(fasting plasma triglycerides 150 mg/dl (1.7 mmol/L)) and arterial hypertension (peripheral arterial blood pressure ≥ 140 / ≥ 90 mmHg). Assessed in terms of IDF criteria, MS was defined as central obesity plus the existence of two other components [14]. Assessed in terms of ATP-III criteria, Ma was defined as having three or more components [15].

Body composition indices

The anthropometric measurements were carried out according to the International Society for the Advancement of Kinanthropometry [16]. Height was measured with a stadiometer to the nearest 0.1 without shoes and hair accessories. Weight was measured to the nearest 0.1 kg, in light clothing and without shoes, using a digital scales. Waist circumference was measured using a standard tape measure. Body Mass Index (BMI) was calculated using weight in kilograms and height in meters ($BMI = kg/m^2$). All the subjects were divided into two groups according to these measurements: Group 1, which consists of obese subjects with MS ($BMI > 30 kg/m^2$) (54 subjects, 33 female, 21 male; mean age 46.2 ± 14.1) and group 2, which consists of non-obese subjects with MS ($BMI < 30 kg/m^2$) (64 subjects, 33 female, 21 male; mean age 41.4 ± 13.2 years). All measurements were completed by trained personnel. The indices were calculated using the following formulas.

A Body Shape Index (ABSI) = $WC / (BMI^{2/3} * height^{1/2})$ [17].

Body Roundness Index (BRI) = $365.2 - 365.5 * \sqrt{(1 - ((wc/2\pi))^2) / (0.5 * height^2)}$ [17].

Conicity Index = $(waist\ circumference\ (m)) / (0.109 * \sqrt{body\ weight\ (kg)}) / height\ (m)$ [17].

Visceral adiposity index (VAI) = $WC / 39.68 + (1.88 * BMI) * TG / 1.03 * 1.31 / HDL$ (Males)
 VAI = $WC / 36.58 + (1.89 * BMI) * TG / 0.81 * 1.52 / HDL$ (Females) [18].

Body adiposity index (BAI) = $hip\ circumference\ (cm) / height\ (m)^{1.5} - 18$ [19].

Laboratory parameters

The following laboratory tests were run using blood drawn from a vein after a minimum 8-hour fast: fasting blood sugar (FBS), fasting insulin, tryglericide (TG), total cholesterol (TC), low density cholesterol (LDL), high density cholesterol (HDL) and c-reactive protein (CRP) levels, as well as liver enzymes (aspartate aminotransferase, alanine *transaminase*) and kidney function tests (urea, creatinine). HOMA-IR (Homeostatic Model Assessment for Insulin Resistance) was calculated using the following formula:

HOMA-IR: $(Fasting\ insulin) * (Fasting\ glucose) / 405$ [20]

Transthoracic echocardiography

The transthoracic echocardiography was performed using the "Philips EPIQ" 7 device (Philips healthcare, 3000 Minuteman Road, Andover, MA, USA). M-mod and 2-D measures were made from the parasternal long axis imaging. In the left lateral decubitus position and using a 2.3-3.5 MHz transducer, the sweep speed for m-mod measurements is 50 mm/sec while that of Doppler measurements is 100 mm/sec. Conventional echo parameters, left atrial mechanical function parameters, interatrial coupling parameters, tissue Doppler parameters, myocardial performance parameters, and ejection fraction were obtained for the patients.

Tissue Doppler and electromechanical coupling

Tissue Doppler echocardiography was performed using a 3.5-4.0 MHz transducer, adjusting the spectral pulsed Doppler signal

filter to obtain the Nyquist limit of 15-20 cm/sec and employing the minimal optimal gain setting. The monitor sweep speed was set at 50-100 mm/sec to optimize the spectral display of myocardial velocities. A pulsed wave (PW) Doppler sample volume was placed in an apical four-chamber view at the level of the LV lateral mitral annulus, septal annulus and tricuspid annuluses.

Atrial electromechanical coupling on the surface electrocardiogram was defined as the period from the beginning of the P wave on the surface electrocardiogram until the beginning of the diastolic wave (the Am wave), in other words, the PA interval, and measured from the lateral mitral annulus (PA lateral), the septal mitral annulus (PA septum) and right ventricular tricuspid annulus (PA tricuspid). The difference between the lateral and tricuspid PS intervals was defined as interatrial electromechanical delay, while the difference between the septal and tricuspid PA intervals was defined as intra-atrial electromechanical delay. All PA intervals were calculated as an average of three consecutive beats [21].

Statistical analysis

Statistical analysis was conducted using the SPSS 16.0 computer software program. The Kolmogorov-Simrnov test was performed to determine the degree to which quantitative variables were normally distributed, while the Levene test was done to determine variance homogeneity. Mean, standard deviation, median and minimum-maximum interval were used as descriptive statistics. Categorical variables were expressed as frequency (n) and valid percentage (%). To assess the difference between two groups when testing hypotheses, the Chi square test was used for categorical variables. In cases where the Chi square test did not provide support for hypotheses, the Fisher

Exact test was used. To compare quantitative variables between two independent groups, if the hypotheses were supported, the Student T test was used; if the hypotheses were not supported, the Mann-Whitney U test was used. Rank order correlations between variables was analyzed using Spearman Rho correlation tests. A level of (p) <0.05 was accepted for statistical significants. Conclusions were evaluated on the basis of a 95% confidence interval.

Results

Table 1 contains the demographic characteristics, risk factors, body composition indices, systolic-diastolic blood pressures and heart rates of the two groups. There were more women in Group 1 than in Group 2. Age, systolic-diastolic blood pressures and all body composition indices were higher in Group 1 than in Group 2. Heart rates in the two groups were similar.

Inter, intra and left atrial EMD intervals are significantly more prolonged in the obese group than in the non-obese group (19.7 ± 9.8 vs 9.6 ± 12.4 ; $p < 0.001$, 6.4 ± 7.0 vs 2.8 ± 6.2 ; $p < 0.001$, 13.4 ± 9.2 vs 6.8 ± 12.4 ; $p < 0.001$, respectively) (Table 2).

Table 2 contains the conventional and tissue Doppler parameters for the two groups. Among conventional parameters; interventricular and posterior wall thickness were significantly higher in group 1 compared to group 2. Left ventricular end-systolic, left atrial and aortic diameters were significantly higher in group 1 compared to group 2. Among diastolic functions/E' lateral, E/septal and EDT were significantly higher, E/A ratio was significantly lower in group 1 compared to group 2.

Table 3 contains the Spearman's correlation of inter-atrial and left atrial EMD with body composition indices. There is a significant correlation between them and all indices apart

Table 1. Demographic and laboratory features of the groups.

Parameters	Group 1 (n=54)	Group 2 (n=64)	<i>p</i>
Age (years)	46.2±14.1	41.4±13.2	0.061
Gender (M,n)	33	27	0.041
Smoking (n)	16	20	0.480
Systolic blood pressure (mm Hg)	138.3±19.2	123.1±18.4	<0.001
Diastolic blood pressure (mm Hg)	82.1±11.3	75.7±10.2	<0.001
Heart rate (beat/min)	81.1±11.4	80.5±11.6	0.792
BMI (kg/m ²)	37.3±6.4	25.6±2.9	<0.001
WC (cm)	111.8±12.4	88.2±11.7	<0.001
Waist to Hip ratio	0.94±0.07	0.88±0.09	0.001
BSI	0.079±0.005	0.078±0.006	0.476
BRI	7.83±2.51	3.96±1.35	<0.001
VAI	2.87±1.65	1.58±1.01	<0.001
BAI	40.1±9.00	28.1±4.05	<0.001
CI	1.32±0.09	1.23±0.11	<0.001
Glucose (mg/dL)	129.9±62.5	98.9±31.1	0.001
Total cholesterol (mg/dL)	220.3±49.2	206.5±46.6	0.099
Triglyceride (mg/dL)	169.0±93.8	113.1±60.0	<0.001
LDL cholesterol (mg/dL)	141.2±38.4	132.1±38.3	0.212
HDL cholesterol (mg/dL)	44.6±9.6	52.1±12.1	<0.001
Fasting insulin (IU/ml)	14.4±9.2	7.2±4.1	<0.001
HbA1C (%)	7.23±2.01	6.05±1.20	0.003
HOMA-ir	4.27±4.29	1.70±1.17	<0.001

BMI (body mass index), WC (waist circumference), BSI (body surface index), BRI (body roundness index), VAI (visceral adiposity index), BAI (body adiposity index), CI (conicity index), LDL (low density lipoprotein), HDL (high density lipoprotein), HbA1C (glycated hemoglobin), HOMA IR (Homeostatic Model Assessment of Insulin Resistance).

from BSI. The strongest correlation is between BRI and interatrial and left atrial EMD ($r=0.46$; $p<0.001$, $r=0.39$; $p<0.001$, respectively). Only a weak correlation was found between intra-atrial EMD and the body composition parameters BRI and VAI ($r=0.21$; $p=0.03$, $r=0.36$; $p<0.001$).

In addition, there was a significant correlation between interatrial EMD and fasting blood glucose, fasting insulin, HbA1C and HOMA-IR, and left atrial EMD and fasting insulin, HbA1C and HOMA-IR (interatrial; $r=0.22$; $p=0.018$, $r=0.30$; $p=0.01$, $r=0.32$; $p=0.004$,

$r=0.26$; $p=0.004$, left atrial; $r=0.20$; $p=0.035$, $r=0.24$; $p=0.00435$, $r=0.20$; $p=0.033$ respectively).

Discussion

The key finding of our study is that the risk of atrial fibrillation is higher in obese people with metabolic syndrome than in non-obese persons. The electromechanical delay intervals that indicate AF risk are correlated with body composition parameters. However, while they are more strongly correlated with the body roundness index (BRI) than with conventional

Table 2. Conventional echocardiographic measurements, tissue Doppler and electromechanical durations of the groups.

Parameters	Group 1 (n=54)	Group 2 (n=64)	p
LVEDD (cm)	4.81±0.34	4.73±0.45	0.253
LVESD (cm)	2.85±0.39	2.64±0.41	0.006
IVS (cm)	1.06±0.20	0.93±0.21	0.001
PW (cm)	1.12±0.35	0.89±0.16	0.033
Aortic diameter (cm)	2.67±0.34	2.50±0.35	0.012
Left atrial diameter (cm)	3.56±0.32	3.15±0.47	<0.001
EF (%)	64.9±2.5	66.7±4.5	0.012
Mitral E (cm/s)	0.72±0.17	0.77±0.16	0.188
Mitral A (cm/s)	0.75±0.15	0.66±0.13	0.214
Mitral E/A	0.98±0.29	1.22±0.36	<0.001
Mitral EDT (ms)	199±42	184±32	0.037
E/E' lateral	8.0±3.1	6.5±2.1	0.006
E/E' septal	10.0±3.1	8.9±2.1	0.043
Inter-atrial EMD	19.7±9.8	9.6±12.4	<0.001
Intra-atrial EMD	6.4±7.0	2.8±6.2	<0.001
Left atrial EMD	13.4±9.2	6.8±12.4	<0.001

LVEDD (left ventricle end-diastolic diameter), LVESD (left ventricle end-systolic diameter), IVS (interventricular septum), PW (posterior wall), EF (ejection fraction), EDT (E wave deceleration time), EMD (electromechanical duration).

parameters. The pathophysiology underlying metabolic syndrome is still open to debate. In studies done in previous years, insulin resistance was thought to be the key problem. However, more recent studies have found visceral adiposity is a strong independent predictor of the other parameters seen in MS [22] Visceral body fat, which is a key cause of insulin resistance, is known to be linked to cardiovascular diseases and other side effects in the body. Generally, the abdominal adipose tissue produces various autocrine, paracrine and endocrine activity compounds that can have an impact on metabolism and the cardiovascular system. This aspect of adipose tissue is one of the reasons why it contributes to AF development [23].

Many studies have been conducted on the relationship between AF and both MS and the most important component of MS, obesity.

Chamberlain et al. conducted a study on 15,000 MS subjects, following them for an average of 15.4 years, and discovered that they were at a 67% higher risk of developing AF. The study found that as the number of MS components increased, the risk of AF also rose [24] In another study, Kurt et al. analyzed electromechanical delay in 72 MS subjects and saw that their EMD intervals were significantly more prolonged than those in the control group. However, they did not find that this prolongation of EMD was associated with MS severity [25]. In a study done on 87 patients, Yılmaz et al. found MS had a negative impact not only on EMD but also on P wave dispersion and left atrial mechanical functions [26].

A study done by Erdem et al. on obese subjects showed that electromechanical delay in obese persons was significantly greater than in non-obese persons. It also reported a correlation

Table 3. Spearman’s correlation of inter, intra and left atrial mechanical delays with body composition indices (respectively).

Body composition indices	rho	P
BMI	0.45	<0.001
WC	0.44	<0.001
BSI	0.12	0.193
BRI	0.51	<0.001
AVI	0.44	<0.001
BAI	0.38	0.001
CI	0.29	0.074
VAI	0.36	<0.001
BMI	0.40	<0.001
WC	0.37	<0.001
BSI	0.11	0.246
BRI	0.41	<0.001
AVI	0.36	<0.001
BAI	0.35	<0.001
CI	0.27	0.003
VAI	0.39	<0.001
BMI	0.40	<0.001
WC	0.48	<0.001
BSI	0.30	0.001
BRI	0.51	<0.001
AVI	0.48	<0.001
BAI	0.30	<0.001
CI	0.45	<0.001
VAI	0.41	<0.001

BMI (body mass index), WC (waist circumference), BSI (body surface index), BRI (body roundness index), VAI (visceral adiposity index), BAI (body adiposity index), CI (conicity index).

between prolonged electromechanical delay and BMI indices. When evaluating the study’s results, the relatively small size of the groups included in it (e.g., 35 obese patients) should be borne in mind [27] Temiz et al. reached the same conclusions in a study they conducted on 59 obese children aged 8-18. They found a strong association between prolonged inter and intra-atrial mechanical delay intervals and BMI

[28]. This study is also important in that it demonstrated there was also a tendency towards AF in obese children [28]. Another study, which analyzed the relationship between obesity and atrial EMD and included a total of 80 subjects (40 obese-40 normal weight), also found prolonged electromechanical delay intervals. In the study, the authors linked the prolonged EMD intervals seen in obese subjects to an increase in low-grade inflammation, left atrial delay and diastolic dysfunction [13]

The relationship between obesity and AF when other illnesses are present in various illness populations has been studied. Taşolar et al. analyzed EMD intervals in obese (≥ 30 kg/m²) and thin (≤ 25 kg/m²) patients with polycystic ovary syndrome (PCOS) and discovered that while in thin patients, EMD intervals were more prolonged compared to the control group, the EMD intervals in obese patients were significantly more prolonged than in both PCOS patients and the control group. Moreover, in regression analysis, they showed through HOMA-IR that BMI was an independent determinant of electromechanical delay intervals [29].

Our study also showed that EMDs in obese MS patients were significantly more prolonged than in non-obese MS patients. In addition, there was a significant correlation between EMD intervals and other laboratory components.

While the mechanism of increased AF risk in obesity, which is a major determinant of MS, is not known with certainty, there are many speculations about what underlies the observed relationship. Studies have proposed a number of causes. They include increased fat inflammation in the atrial wall, heightened sympathetic nervous system activity, high inflammatory process, increased oxidative stress, irregular adipokine secretion, and

activation of various profibrotic signal pathways [30,31]. In addition, earlier studies reported LA dilatation and LV diastolic dysfunction in obese cases [32] Increased plasma volume, ventricular diastolic dysfunction and increased neurohormal activation may result in the expansion of the left atrium, which contributes to electrical instability [33].

Although there is a close relationship between MS and obesity, metabolic disorders generally go overlooked in nonoverweight/non-obese people.^[9] Visceral fat plays a critical role in the pathogenesis of MS. Since visceral fat mostly concentrates in the abdomen, for a long time now, central obesity has been identified and managed in these individuals through simple anthropometric indices such as waist circumference (WC) and body mass index (BMI). Because of the weakness these parameters have in revealing fat distribution, new anthropometric indices have been developed [34] Many studies attempting to determine which body composition parameter predicts MS the best have been carried out. For example, while Wu et al. found BMI and WC to be the most relevant indices, [35] Tian et al. reported that WC was the most promising³⁶. In one study Tian et al. conducted, they saw that in a population of women, WC and BRI had better predictive value than BMI and BSI [36]. In another study, Gliszek et al. assessed the value of anthropometric indices where at least one MS component was present. While BMI and WC were better determinants of MS in men, waist-to-height ratio and WC were better determinants in women. Another result of the study was that BSI was not a very good determinant [37] A study was done in China on 2916 nonoverweight/obese adults to see which new body composition indices would identify MS. The research concluded that abdominal

volume index (AVI) was the optimal anthropometric index for identifying MS, and that BRI also played an important role. The authors reported that BSI was a weaker parameter in this regard⁹. Many studies have been conducted not only on AVI but also BRI and BSI; BRI has been shown to be optimal in identifying metabolic components in extremely overweight/obese persons, [38] while a relationship has been demonstrated between BSI and visceral adipose tissue and carotid atherosclerosis [39]

A study done in South Africa looked at the relationship between new BMIs and nutritional status and cardiometabolic risk factors in young adults. It concluded that BRI was associated with dyslipidemia and that conicity index (CI) was associated with insulin resistance, hypertension and dyslipidemia [17]. In another study conducted by Wu et al., the authors examined the value of conventional and new body mass indices in identifying nonadipose cardiometabolic risk. While all indices apart from BAI and BSI were able to identify high cardiometabolic risk, compared to all the other indices, BRI performed better in this respect [40].

In our study, atrial conduction intervals were more prolonged in obese individuals with MS than in non-obese individuals. EMD intervals were significantly associated with such conventional body composition parameters as BMI and waist circumference, as well as with BRI, VAI, BAI, and CI. In addition, considering that increased adipose tissue is one of the fundamental parameters of MS, there was a strong relationship between electromechanical conduction durations and body composition parameters. A relationship was seen between both conventional and new BCIs and the development of atrial fibrillation in these individuals. Because our study

identified BRI as the best parameter for the purpose, we believe that it could be a target parameter in preventing arrhythmias that emerge in individuals with MS.

Our study was a cross-sectional one that included a patient group that met the criteria within a particular period of time. Without a doubt, our findings cannot be generalized to the entire population. Moreover, they may be associated with the ethnic group to which most participants in the study belonged. This should be borne in mind when comparing the findings with other geographical areas and ethnic groups. Another limitation is that the study did not evaluate the group the development of atrial fibrillation prospectively. The participants were not monitored with Holter monitors for the presence of atrial fibrillation. We did not feel it was appropriate to have patients without any complaints related to arrhythmias wear a Holter monitor. The relatively small size of the study population may be another limitation.

Conclusions

Obese individuals with MS had longer EMD intervals than non-obese individuals. The parameter most closely associated with EMD was BRI. The key finding of our study was that BRI was the body composition index having the most power to identify AF development risk in individuals with MS. We believe that it could be a target parameter in the monitoring and treating of AF.

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