Int J Clin Exp Med 2014;7(6):1598-1602
www.ijcem.com /ISSN:1940-5901/IJCEM0000457

Original Article

Evaluation of therelationship between epicardial adipose tissue and myocardial performance (Tei) index

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Received April 8, 2014; Accepted April 18, 2014; Epub June 15, 2014; Published June 30, 2014

Abstract: Aim: Epicardial adipose tissue (EAT) is a tissue around the heart with visceral adipose properties. It can affect the structure and functions of the myocardium and coronary arteries through inflammatory markers and regulators. The myocardial performance (Tei) index is a parameter capable of globally assessing systolic and diastolic heart functions. This study investigated the relation between EAT thickness and the Tei index. Methods: The study population was selected from patients without structural heart disease. EAT thickness was measured with two-dimensional imaging in parasternal long axis view and from the anterior face of the right ventricle. Tei index measurement was calculated with tissue Doppler echocardiography from the mitral lateral annulus. The relation between the Tei index and EAT was assessed using multivariate linear regression analysis. Results: Forty-three patients (36 female, 7 male; mean age 50.2 ± 10.6 years) were included in the study. Mean Tei index was 0.39 ± 0.09, and mean EAT thickness 4.7 ± 2.4 mm. A significant correlation was determined between tissue Doppler Tei index and EAT thickness at correlation analysis (r = 0.522, P < 0.001). EAT thickness was independently correlated with Tei index at multivariate linear regression analysis (Beta = 0.443, t = 3.522, P = 0.001). Conclusion: EAT thickness is independently correlation with Tei index. Increased EAT thickness may therefore be a predictor of left ventricular dysfunction.

Keywords: Epicardial adipose tissue, tissue Doppler echocardiography, Tei index

Introduction

Epicardial adipose tissue (EAT), comprising one part of the visceral pericardial layer, is most densely present in the atrioventricular canal and the coronary arteries [1]. It is directly associated with the coronary artery walls or the myocardium beneath. There is therefore a close metabolic relation between these structures [2]. EAT thickness measured using echocardiography (ECO) is correlated with the thickness of visceral adipose tissue measured using computerized tomography (CT) and magnetic resonance imaging (MRI) [3, 4]. In addition, EAT thickness is closely associated with both the presence and expanse of coronary artery disease [5, 6]. EAT thickness determined with ECO can therefore be used in risk screening for atherosclerotic cardiovascular disease and in predicting the spread of the disease. Despite its close association with coronary artery disease, there are few studies assessing EAT's association with left ventricular (LV) functions.

The standard technique today in the assessment of LV systolic and diastolic functions is ECO. The "myocardial performance (Tei) index" proposed by Tei et al. [7] permits the combined assessment of LV systolic and diastolic functions. This study investigated the correlation between EAT and LV Tei index.

Materials and methods

Patient selection

The study population was drawn from patients undergoing ECO at the Karadeniz Technical University Cardiology Clinic. Patients with non-sinus rhythm, uncontrolled hypertension, connective tissue disease, valve or congenital

Table 1. Demographic characteristics of study population

Age (years)	50.2 ± 10.6
Sex (Female/male)	36/7
Hypertension no (%)	17 (39.5%)
Cigarette use No (%)	14 (32.6%)
BMI (kg/m^2)	28.2 ± 3.2
Systolic BP (mmHg)	121.6 ± 17.2
Diastolic BP (mmHg)	74.4 ± 13.5
Glucose (mg/dl)	96.9 ± 14.1
Creatinine (mg/dl)	0.88 ± 0.14
Total cholesterol (mg/dl)	195 ± 26.5
Triglyceride (mg/dl)	152.8 ± 74.4
LDL-cholesterol (mg/dl)	127.8 ± 22.1
HDL-cholesterol (mg/dl)	41.5 ± 7.6

BMI: body mass index; BP: blood pressure; HDL: high density lipoprotein; LDL: low density lipoprotein.

Table 2. Echocardiographic characteristics of study population

	Mean ± standard deviation
LVSD (mm)	30.2 ± 3.7
LVDD (mm)	47.1 ± 3.2
IVS (mm)	10.3 ± 1.5
PW (mm)	9.7 ± 1.2
LVMI (g/m^2)	85.9 ± 17.7
Ejection fraction (%)	64.4 ± 5.2
E (cm/sn)	70.1 ± 14.7
A (cm/sn)	66.8 ± 16.2
E/A	1.1 ± 0.3
Tei index	0.38 ± 0.08
E'	11.8 ± 3.5
A'	11.6 ± 2.6
S'	10.6 ± 2.9
E'/A'	1.1 ± 0.4
Tei index (tissue Doppler)	0.39 ± 0.09
Epicardial fat (mm)	4.7 ± 2.4

A: late mitral filling velocity; A': mitral annular late diastolic wave; E: early mitral filling velocity; E': mitral annular early diastolic wave; IVS: interventricular septum thickness; LVDD: left ventricular end of diastole diameter; LVMI: left ventricular mass index; LVSD: sol ventricular end of systole diameter; PW: left ventricular posterior wall thickness; S': mitral annular systolic wave.

heart disease, chronic obstructive pulmonary disease, heart failure (EF < 50%) or LV segmental wall motion abnormalities, aortic disease (such as Marfan syndrome or aneurysm), active infection or inflammatory disease, diabetes mellitus (fasting blood sugar > 126 mg/dl or antidiabetic drug use), known malignancy or kidney disease (acute or chronic) were excluded from the study. Presence of blood pressure > 140/90 mmHg or use of hypertensive drugs was regarded as hypertension. Fasting blood sugar, total cholesterol, low density cholesterol, high density cholesterol and triglyceride levels were measured from fasting blood specimens. The study protocol was approved by the local ethical committee.

Epicardial fat measurement

Transthoracic ECO was used to measure epicardial fat thickness. Measurements were made with the patient in the left lateral decubitus position and in long parasternal axis view. The echo free space in the anterior face of the right ventricle was defined as EAT, and measurements were taken using the aortic and mitral annulus as reference [6]. EAT thickness was measured three times perpendicular to the right ventricular free wall, and the mean of these measurements was taken.

Evaluation of myocardial performance index

LV Tei index was obtained from both standard Doppler ECO with transmitral filling velocities and from the lateral mitral annulus using tissue Doppler.

Standard basal and dopler ECO measurements

Basal measurements were performed with M-mode ECO in parasternal long axis view. Systolic and diastolic LV diameters and septum and posterior wall thicknesses were measured. LV mass index was calculated using the Devereux formula [8]. LV systolic and diastolic end volumes were performed in apical four chamber view. Ejection fraction was calculated using the modified Simpson method. Doppler examination was performed in apical four chamber view. Sample volume was placed 1 cm below the mitral valve tips, and early (E) and late (A) mitral filling waves were recorded. Amplitudes and E wave deceleration time were measured. Isovolumetric relaxation time (IVRT) and total diastole duration (a) were measured. Sample volume was positioned at the aorta, and the ejection wave was recorded and its duration measured. Acquisition, Nyquist limit and filter adjustment were performed for tissue

Table 3. Independent determinants of myocardial performance index

	Beta	t	p	95% Confidence interval	
				Lower threshold	Upper threshold
Age	-0.91	-0.701	0.49	-0.003	0.001
Gender	0.108	0.861	0.39	-0.034	0.084
Hypertension	0.503	3.828	< 0.001	0.041	0.135
Epicardial fat tissue	0.443	3.522	0.001	0.007	0.25

Doppler examination. Tissue Doppler evaluation was performed from the lateral mitral annulus. Systolic wave (S'), early (E') and late (A') diastolic wave amplitudes, isovolumetric contraction time (IVCT), IVRT and systolic wave durations were measured.

Myocardial performance index calculation

Myocardial performance index was calculated both from standard Doppler obtained with transmitral filling velocities and also from lateral mitral annulus Doppler specimens using the method recommended by Tei et al. [7].

Tei index = (IVCT + IVRT)/ejection time = (a-b)/b

a: total diastole period; b: ejection time; IVCT: isovolumetric contraction time; IVRT: isovolumetric relaxation time.

Statistical analysis

All constant variables were expressed as mean ± standard deviation. Categoric variables were expressed as percentages. Compatibility with normal distribution was assessed using the Kolmogorov-Smirnov test, and the association between Tei index and EAT and other constant variables was assessed using correlation analysis. At correlation analysis, the Pearson test was used for normal distribution and Spearman's test for non-normal distribution. Linear multivariate regression analysis was performed in order to determine Tei index independent determinants. All statistical analysis was performed on SPSS (13.0, Inc, Chicago, Illinois), and significance was set at $P < 0.05$.

Results

Demographic characteristics of the study population are shown in **Table 1**. This consisted of 43 patients (36 female, 7 male, mean age 50.2 ± 10.6 years). Two-dimensional, M-mode and standard Doppler ECO results are summarized in **Table 2**. Mean Tei index was 0.38 ± 0.08 for standard Doppler and 0.39 ± 0.09 for tissue Doppler. Mean EAT thickness was 4.7 ± 2.4 mm. At correlation analysis, a significant difference was determined between tissue Doppler Tei index and EAT thickness ($r = 0.522$, $P < 0.001$). At multivariate regression analysis, presence of hypertension and EAT thickness were independently correlated with the Tei index (**Table 3**).

Discussion

In this study, the association of EAT thickness determined using ECO was compared with the Tei index, a marker of LV global function. EAT and hypertension were independently correlated with the Tei index.

The myocardial performance index proposed by Tei et al. [7] is a Doppler-based parameter that allows systolic and diastolic functions to be assessed together. Poulsen et al. [9] also reported that it is more sensitive than ejection fraction in predicting LV dysfunction.

EAT is a fat tissue with visceral properties found predominantly around the atrioventricular canal and coronary arteries [1]. The fact that the adjacent myocardium and coronary artery wall have a close anatomical and functional relationship leads to it having a dynamic affect on the working of the heart [1, 2]. It acts as a store for fatty acids to be released when urgently required. It also has a regulatory role on inflammation and endothelial functions by releasing inflammatory mediators. It has also been shown to be associated with various cardiovascular diseases. Natale et al. [10] found that carotid artery intima-media thickness and arterial stiffness were an independent predictor of EAT thickness. Eroğlu et al. [5] showed that EAT thickness is correlated with the presence and severity of coronary artery disease confirmed with coronary angiography. An association with coronary artery disease has also been determined in other studies [5, 6, 12]. Sade et al. [13] determined that EAT thickness was correlated with compromise of coronary flow reserve in female patients with angina symptoms but whose coronary arteries were

angiographically normal. Aydın et al. [14] showed a correlation between EAT thickness and impaired endothelial function in metabolic syndrome patients.

EAT thickness measurement was previously performed using CT and MRG, and then began being assessed using transthoracic ECO by Iacobellis et al. [3] in 2003. Echocardiographic measurement results are largely compatible with those from CT and MRG. In addition, since ECO is a practical, economical and repeatable technique not involving exposure to radiation it quickly became an important method for assessing epicardial and visceral fat tissue [1].

We determined an independent relation between EAT and the Tei index. The literature contains a number of studies examining the relation between cardiac function and EAT. Khawaja et al. [15] reported the presence of lower epicardial fat tissue volume in patients with an ejection fraction less than 55%. Additional analysis in the study established a lower epicardial fat tissue volume in patients with even lower ejection fractions (< 35%). That was attributed to epicardial fat being an energy store for the myocardium and a cytokine regulator. Natale et al. [10] divided patients into two groups on the basis epicardial fat thickness (≤ 7 mm and > 7 mm), and found E/A and E'/A' ratios were lower in the higher EAT thickness group. However, that study did not analyze the relation between diastolic function and epicardial fat tissue thickness using regression analysis.

Our low patient number represents a significant limitation. However, we sought to overcome this through being highly selective in establishing the study population. Another limitation is that no specific patient group was investigated. However, this may be important in permitting generalization of our data to the general population. Inflammation and inflammatory mediators occupy an important place in the cardiovascular effects of EAT [1]. We did not use inflammatory parameters, however. This makes it harder to interpret our study data in terms of etiopathogenesis. Our study data therefore need to be corroborated with studies with wider participation.

In conclusion, the regulation of LV systolic and diastolic function is a dynamic process, and several factors affecting preload and afterload can alter these functions. EAT may affect global LV performance assessed using the Tei index by affecting coronary flow reserve, endothelial dysfunction and the myocardial fatty acid balance.

Disclosure of conflict of interest

None.

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