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ORIGINAL RESEARCH

Epicardial Adipose Tissue Thickness is Higher in Right Ventricular Outflow Tract Tachycardia

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ABSTRACT

Introduction: Idiopathic ventricular arrhythmias, which occur in the absence of structural heart disease, are commonly originating from the outflow tract, and 80% of the them arise from the right ventricle. Epicardial adipose tissue (EAT), which originates from the splanchnopleuric mesoderm, has been shown to be an important source of inflammatory mediators and plays an important role in cardiac autonomic function by epicardial ganglionated plexuses. EAT may potentially contribute to the pathophysiology of idiopathic right ventricular outflow tract (RVOT) tachycardia by different mechanisms. In this study, we aimed to investigate the relationship between EAT thickness and RVOT tachycardia. **Methods:** This study included 55 patients (32 male, 23 female) with RVOT tachycardia and 60 control subjects (38 male, 22 female). Patients who had more than three consecutive ventricular beats over 100 bpm with specific morphological features on the electrocardiogram (ECG) were diagnosed with RVOT tachycardia. EAT thickness was measured by transthoracic echocardiography. **Results:** EAT thickness was significantly higher in the RVOT tachycardia group ($p < 0.05$). Ejection fraction (EF), and the thickness of the posterior wall of the left ventricle and of the interventricular septum were significantly lower, and left ventricular end-diastolic diameter, left ventricular end-systolic diameter, and left atrial diameter were significantly higher in patients who had RVOT tachycardia compared to normal subjects ($p < 0.05$). **Conclusion:** Patients who were diagnosed with RVOT tachycardia had increased EAT thickness compared to normal subjects. The underlying mechanism of the condition could be mechanical, metabolic, infiltrative, or autonomic effects of the EAT.

Keywords: epicardial adipose tissue, ventricular tachycardia, right ventricular outflow

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INTRODUCTION

Idiopathic ventricular arrhythmias, which occur in the absence of structural heart disease or ion channelopathies, are commonly originating from the outflow tract, and 80% of them arise from the right ventricle.[1–3] Right ventricular outflow tract (RVOT) tachycardias are generally observed in adolescents or young adults, and cyclic adenosine monophosphate (c-AMP)-mediated delayed afterdepolarizations and triggered activity is thought to be the underlying pathophysiology of this condition.[1,2] Outflow tract tachycardia is usually adrenergic-mediated and occurs during excercise.[4] ECG findings of RVOT tachycardia include a wide range of QRS complex, a left bundle branch block morphology, and an inferior QRS axis.[5]

Epicardial adipose tissue (EAT), which originates from the splanchnopleuric mesoderm, has been shown to be an important source of inflammatory mediators, effecting the adjacent myocardium through paracrine or vasocrine secretion.[6,7] A fat-cell migration occurs from the EAT to the myocardium due to the absence of fascial boundary between them.[8] In addition, it has been shown that EAT plays a substantial role in cardiac autonomic function.[9]

EAT may potentially contribute to the pathophysiology of idiopathic RVOT tachycardia by different mechanisms. In this study, we aimed to investigate the relationship between EAT thickness and RVOT tachycardia.

METHODS

STUDY POPULATION

This cross-sectional study was conducted between January 2015 and March 2016. The study included 55 patients (32 male, 23 female) with RVOT tachycardia and 60 healthy subjects (38 male, 22 female).

Demographic features, family history of sudden cardiac death, and physical examination were recorded. Peripheral venous blood samples were drawn to analyze the complete blood count, liver and kidney function, electrolytes, thyroid stimulating hormone, HbA1c, and lipid profile from all the patients and healthy subjects. Transthoracic echocardiography, surface 12-lead electrocardiography, and 24-hour Holter monitorization were performed for each study subject.

Ventricular tachycardia (VT) was diagnosed according to Vereckei and Brugada's criteria.[10–12] Specific morphological features of the ECG were used to determine the location of the arrhythmia. RVOT tachycardia was diagnosed according to the following criteria: more than three consecutive ventricular beats over 100 bpm with specific morphological features (wide range QRS complex, a left bundle branch block, an inferior QRS axis, and transition of R dominance after lead V3) on the ECG. Patients who presented with exercise-induced tachycardia underwent a treadmill stress test revealing tachycardia.

EXCLUSION CRITERIA

Patients who had diabetes mellitus (DM), hypertension, low hemoglobin level (<12 g/dL), thyroid disorders, renal dysfunction, hepatic disorders, coronary artery disease (CAD), structural heart disease, electrolyte imbalance, family history of sudden cardiac death, long/short QT distance or Brugada pattern on surface ECG, left and right bundle branch block morphology on resting ECG were excluded from the study. DM was defined as a fasting plasma glucose >126 mg/dL, HbA1c >6.5%, or current medication with hypoglycemic agents. Hypertension was defined as current medication with antihypertensive agents or repeated measurements of blood pressure >140/90 mmHg. Thyroid disorders were defined as serum TSH >5.4 µIU/mL, TSH <0.5 µIU/mL, or current treatment with thyroid replacement therapy or antithyroid agents. Hepatic disorders were defined as a 2.5-fold increase in serum hepatic enzymes.

Invasive coronary angiography was performed in patients who had CAD risk factors or anginal signs and symptoms. Coronary artery lesion causing >50% luminal narrowing or previous coronary artery stent implantation or coronary artery bypass surgery was defined as CAD.

TRANSTHORACIC ECHOCARDIOGRAPHY

Transthoracic echocardiography (Epiq 7; Philips) was evaluated by the same practitioner by using a standard protocol in all patients. Left ventricular ejection fraction (EF) was calculated with the modified Simpson's formula from the apical four-chamber view. Parasternal longitudinal view was used to measure the EAT thickness on the right ventricular free wall. An echo-free space between the outer wall of the myocardium and the visceral layer of the pericardium was defined as epicardial fat tissue. EAT thickness was measured perpendicularly on the free wall of the right ventricle at the end-systole in three cardiac cycles according to a predefined method.[13]

DATA ANALYSIS

All statistical calculations were performed with SPSS 23.0 (SPSS for Windows, Chicago, IL, USA). All continuous

TABLE 1. Clinical and demographic findings of the patients

		VT group (n = 55)	Control group (n = 60)	p value
Gender	Male, n (%)	32 (58%)	38 (63%)	0.6
	Female, n (%)	23 (42%)	22 (37%)	
Age (years)		40.58 ± 12.11	37.57 ± 9.93	0.2
BMI		26.63 ± 4.09	25.09 ± 4.55	0.05

BMI, body mass index; VT, ventricular tachycardia

variables were expressed as mean ± standard deviation; categoric variables were defined as percentages (%). Categorical parameters were compared with the Chi square test and Fischer's exact test. Normal distribution was determined by histogram and the Kolmogorov-Smirnov test. Mean values of continuous variables were compared between the groups using the Mann-Whitney U test. All tests were applied as two tailed; the statistical significance level was p <0.05.

ETHICS

The study was approved by the ethics committee of the Şevket Yılmaz Medical and Research Hospital, Bursa, Turkey, and the study was conducted according to the principles stipulated in the Declaration of Helsinki.

RESULTS

Baseline characteristics of the study population are summarized in Table 1. There was no difference between groups in terms of mean age and gender. The body mass index (BMI) was higher in the VT-group (p <0.05). The existence of a relationship between EAT and BMI was examined with Pearson's correlation coefficient, and a statistically significant, positive, weak correlation was found between them (r = 0.38, p = 0.001).

Conventional echocardiographic findings and EAT thickness for both groups are shown in Table 2. EAT thickness was significantly higher in the RVOT tachycardia group (p <0.05). EF, and the thickness of the posterior wall of the left ventricle and of the interventricular septum were significantly lower, while the left ventricular end-diastolic diameter, left ventricular end-systolic diameter, and left atrial diameter were significantly higher in patients who had RVOT tachycardia compared to normal subjects (p <0.05).

Complete blood counts and biochemical parameters are presented in Table 3 and Table 4, respectively. Neutrophil count and sodium levels were lower, whereas red blood cell distribution width (RDW), creatinine and potassium levels were higher in the RVOT tachycardia group than in the control group.

DISCUSSIONS

The results of this study showed that the EAT thickness was significantly associated with the presence of RVOT

TABLE 2. Echocardiographic findings in both groups

	VT group (n = 55)	Control group (n = 60)	p value
EAT (cm)	0.914 ± 0.19	0.488 ± 0.16	<0.001
EF (%)	55.16 ± 7.85	64.58 ± 3.82	<0.001
LVEDD (cm)	5.01 ± 0.32	4.65 ± 0.28	<0.001
LVESD (cm)	3.4 ± 0.45	2.89 ± 0.25	<0.001
IVS (cm)	0.91 ± 0.08	0.96 ± 0.09	<0.001
PW (cm)	0.82 ± 0.08	0.88 ± 0.09	<0.001
LA (cm)	3.77 ± 0.35	3.39 ± 0.35	<0.001

EAT, epicardial adipose tissue; EF, ejection fraction; IVS, interventricular septum; LA, left atrium; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; PW, posterior wall; VT, ventricular tachycardia

TABLE 3. Complete blood count measurements for the two groups

	VT group (n = 55)	Control group (n = 60)	p value
WBC (1/µL)	7,978.55 ± 1485.67	8,923.33 ± 2,387.20	0.1
Hg (g/dL)	14.18 ± 1.48	14.08 ± 1.41	0.7
PLT (1/µL)	248.60 ± 63.04	254.06 ± 53.22	0.6
MPV (fL)	8.69 ± 1.08	8.33 ± 0.98	0.7
RDW (%)	14.26 ± 2.35	13.62 ± 0.93	0.06
NEU (1/µL)	4,458.91 ± 1,101	5,315.67 ± 1,452	0.01
LYM (1/µL)	2,642 ± 610.48	2,652.33 ± 1125.25	0.9

EAT, epicardial adipose tissue; EF, ejection fraction; IVS, interventricular septum; LA, left atrium; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; PW, posterior wall; VT, ventricular tachycardia

TABLE 4. Biochemical parameters and thyroid-stimulating hormone measurements for the two groups

	VT group (n = 55)	Control group (n = 60)	p value
Urea (mg/dL)	13.32 ± 5.08	12.93 ± 4.24	0.6
Creatinine (mg/dL)	0.84 ± 0.18	0.759 ± 0.15	< 0.001
AST (U/L)	24.04 ± 10.36	22.83 ± 4.54	0.4
ALT (U/L)	23.78 ± 14.55	24.63 ± 12.62	0.7
TSH (mlU/L)	1.70 ± 1.08	1.56 ± 1.06	0.5
Sodium (mEq/L)	139.51 ± 1.84	141 ± 1.94	<0.001
Potassium (mEq/L)	4.48 ± 0.38	4.15 ± 0.37	<0.001
Glucose (mg/dL)	88.31 ± 11.5	91.5 ± 10.05	0.1

AST, aspartate aminotransferase; ALT, alanine aminotransferase; TSH, thyroid-stimulating hormone; VT, ventricular tachycardia

tachycardia. EAT, which is a metabolically active organ, plays a substantial role in lipid metabolism and energy homeostasis, secretes pro- and anti-inflammatory cytokines, and provides mechanical support in physiological conditions.[6,7,14,15] On the other hand, when an increase in epicardial fat tissue thickness is observed, it causes an increase in the secretion of proinflammatory cytokines and loses its protective properties.[6,7]

It has been shown that increased free fatty acids due to sympathetic discharge in the early stage of myocardial infarction causes intramyocardial fat accumulation, which may result in ventricular arrhythmias.[17] Baroldi *et al.*[16] have found intense fat cell infiltration in the scar area that had developed after myocardial infarction. They argued that adipocytes in the scar area were formed as a result of metaplasia migration from the epicardium or perivascular area to the myocardium. Additionally, a positive correlation was determined between macroscopic EAT thickness and microscopic fatty infiltrative degree of the myocardium.[8,18,19] Moreover, fatty infiltration leads to separation of intercellular cardiomyocyte communication, which directly results in conduction slowing and/or anisotropy.[20-22] Lipomatous metaplasia causes low bipolar and unipolar amplitudes that may trigger ventricular arrythmia.[23] Increased EAT thickness may play a role in the formation of RVOT tachycardia with similar mechanisms.

It has been shown that EAT is an important source of inflammatory mediators such as tumor necrosis factor alpha, interleukin 1 beta, and interleukin 6.[6,7] Proinflammatory cytokines that act with paracrine and vasocrine secretions have been shown to cause various structural changes in the myocardium, including fibrosis.[6,7,24] Excessive inflammatory mediators caused by increased EAT thickness trigger ventricular arrhythmias by causing structural, ultrastructural, and electrical remodeling of the heart.[24] Increased EAT thickness can result in myocardial fibrosis, and the electrical remodeling of the heart may be a substrate for RVOT tachycardia.

Cardiac ganglion plexuses (GPs) are localized in the EAT and are associated with the intrinsic and extrinsic autonomic nervous system of the heart.[9] In a study in lambs, GPs were mostly localized in the medial part of the superior vena cava, around the left azygous vein, and between the aorta and the pulmonary truncus.[25] GPs between the aorta and pulmonary truncus may be implicated in triggering ventricular arrhythmias due to their close neighborhood to the right ventricular outlet. It has been found that EAT shows its effect on the autonomic function of the heart by interfering with GP function in the epicardial fat pads.[9] Activation of the GPs leads to an increase in calcium ion flux and a shorter duration of action potential, resulting in delayed afterdepolarizations and triggered activity.[26] An increase in the level of inflammatory cytokines is observed when EAT thickness increases, which may play a role in the development of ventricular arrhythmias by causing impaired function of the GPs.

Common causes of cardiomyopathy due to tachycardia include arrhythmias such as atrial fibrillation, atrial flutter, supraventricular tachyarrhythmias, and ventricular tachycardias.[27-31] Tachycardia-induced cardiomyopathy depends on the ventricular rate rather than the origin of the arrhythmia. It has been also shown that tachycardia originating from the RVOT causes tachycardia-induced cardiomyopathy.[32] In this study, we found that the EF was significantly lower in the RVOT tachycardia group compared to the control group, while the end-diastolic diameter was higher. As in previous studies, in our study there was a weak relationship between EAT thickness and BMI.[6,33]

According to our study, serum creatinine, potassium, and sodium levels were within normal range in both groups. However, serum creatinine and potassium levels were significantly higher, and sodium levels were significantly lower in the RVOT tachycardia group.

In our study, we aimed to investigate the relation between EAT thickness and RVOT tachycardia, and we found that there was a significant correlation. This may be owing to mechanical, metabolic, infiltrative, or autonomic effects of the EAT.

LIMITATIONS OF THE STUDY

Our study has several limitations. Firstly, we did not evaluate plasma inflammatory markers and inflammatory activity of the EAT. Secondly, our study could not detect

the locations of cardiac ganglionated plexuses. Thirdly, we chose 2D echocardiography for the evaluation of EAT thickness; however, cardiac magnetic resonance imaging and cardiac computed tomography are the methods of choice for imaging visceral fat.

CONCLUSION

Patients who were diagnosed with RVOT tachycardia had increased EAT thickness compared to normal subjects. The underlying mechanism of the condition could be mechanical, metabolic, infiltrative, or autonomic effects of the EAT.

CONFLICT OF INTEREST

Nothing to declare.

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