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Epicardial Fat Thickness as a Predictor of Ventricular Extrasystoles

Aim	Epicardial adipose tissue (EAT) is a layer between the myocardium and the epicardium, similar to the intra-abdominal adipose tissue. Many cardiovascular diseases have been associated with increased EAT. Limited proof exists that EAT contributes to ventricular extrasystoles (VES). In this study, we aimed to examine the role of EAT on VES.
Material and methods	266 subjects were included in this prospective study between April 2022 and March 2023. They underwent a 12-lead electrocardiogram, 24-hour Holter monitoring, and echocardiography. The subjects were divided into two groups: the VES Group (n=134) (>60 VES/hr) and the non-VES Group (n=132) (<10 VES/hr) group. In addition, severe VES were defined as ≥10.000 VES/24-hr. EAT and other variables were compared between the non-VES and VES groups. Logistic regression analysis was performed to find the factors affecting VES, and an ROC analysis was used to determine the cut-off values of the variables.
Results	EAT was higher in the VES group (p<0.001). In pairwise comparisons, higher EAT in the VES group was independent of ventricular frequency (p=0.552). Variables affecting the presence of VES were left ventricular mass index (p=0.031), QT dispersion (p=0.010), and EAT (p<0.001). The EAT predicted the presence of VES at a cut-off value of 4.05 with a sensitivity of 54.5% and a specificity of 81.3%.
Conclusion	This research indicated that increased EAT might be an independent predictor of VES.
Keywords	Epicardial fat thickness; ventricular extrasystole; QT dispersion; arrhythmia
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Introduction

Epicardial adipose tissue (EAT) is located around the heart and is formed by pathophysiological processes like those that produce intra-abdominal adipose tissue [1]. EAT has a protective effect against atherogenic inflammation. However, excessive EAT causes hypertrophy of epicardial adipocyte cells and increased thickness of epicardial fat appears to be associated with elevated levels and generation of proinflammatory cytokines [2]. In addition, increased EAT can have local effects, e.g., increased beta-adrenergic stimulation, development of coronary artery plaque, and systemic inflammation [3]. In obesity, adipose accumulation is observed in certain parts of the body, and an increase in EAT thickness is typical in obese individuals. It has been suggested that excess adiposity may cause many cardiovascular diseases by increasing proinflammatory mediators and decreasing anti-inflammatory factors [4]. In addition, EAT may act as a barrier to myocardial electrical conduction and, thus, may play a role in the etiology of arrhythmias. Epicardial adipose tissue (EAT) is a specialized type of visceral fat positioned between the myocardium and the pericardium. It serves to protect the adjacent myocardium through its brown fat-like thermogenic capabilities. Simultaneously, it impacts the surrounding myocardial cells by releasing inflammatory and profibrotic factors via its paracrine function. The infiltration of adipose tissue into the myocardium poses a structural barrier to cardiac excitation, causing delays in activation and an increased susceptibility to arrhythmias. Furthermore, the intercellular electrical coupling between cardiomyocytes and epicardial adipose tissue (EAT) can contribute to a further slowdown in conduction, raising the risk of blockages and promoting re-entry, thereby enhancing the likelihood of arrhythmias [5]. Although sudden cardiac death due to malignant arrhythmias is frequently seen in structural heart disease, the etiology of malignant arrhythmias in individuals without structural heart disease has not been fully elucidated.

Ventricular extrasystoles (VESs) are irregular contractions in the heart, resulting from ventricle foci that do not utilize the physiological conduction system of the heart. Instances of idiopathic ventricular tachycardia (VT) or frequent VESs

originating from the right ventricular outflow tract typically manifest in the absence of structural cardiac abnormalities [6]. While VES often results from metabolic or coronary artery disease, VES may also be idiopathic. Frequent VES may increase the likelihood of developing heart failure. In addition, they may predispose to attacks of ventricular tachycardia that result in sudden cardiac death [7]. However, the etiology of VES and its association with major cardiac events is not completely clear. Kırış A. et al found increased EAT in individuals with frequent, idiopathic VES [8]. However, it included a smaller number of patients than our study. We used echocardiography as in this study and, unlike this study, we also examined ventricular repolarization parameters. Shen J et al. recently demonstrated a relationship between the volume and density of EAT and VES. Computed tomography was used to measure the volume and density of EAT. Only patients with frequent VES who underwent radiofrequency ablation were included in this study [5]. Increased EAT may initiate arrhythmia cycling and lead to failure of medical and ablation therapies. Another study reported a correlation between EAT and ablation failure in individuals with high arrhythmia burden [9]. Studies on the effect of EAT on severe ventricular arrhythmias and ventricular repolarization parameters are limited, although some studies have shown a correlation between EAT with atrial fibrillation (AF). They have been suggested that epicardial adipocytes may possess arrhythmogenic characteristics through the production of inflammatory cytokines, adipocytokines, and the initiation of interactions between adipocytes and myocytes. These interactions are believed to play a role in contributing to atrial fibrillation (AF). Additionally, the proximity of epicardial fat to the ostia of the pulmonary veins, which are recognized as having implications in the pathogenesis of AF, is thought to result in a local impact [10]. In the present study, we have investigated the role of EAT in the occurrence of VES using echocardiography.

Material and methods

A prospective study from the two centers (Kahramanmaras Sutcu Imam University Faculty of Medicine and Necip Fazıl City Hospital) was conducted on 266 participants, including 134 control and 132 VES patients, who were examined between April 2022 and March 2023. All the participants' demographic, electrocardiographic, echocardiographic, and laboratory data were recorded. In addition to standard parameters in the echocardiogram, EAT was measured from echocardiographic images. Patients with poor echocardiographic images or with a lack of precise evaluation of EAT were excluded from the study. Three-channel digital during 24-hour Holter monitoring was applied to all individuals. The number of VES per hour is calculated by dividing the total number of ventricular extrasystoles per day by 24. Individuals with <10 VES /hr during 24-hour Holter monitoring were taken as the non-VES (Control) group. Exclusion criteria were as follows: structural heart disease (dilated and hypertrophic cardiomyopathy), heart failure with ventricular ejection fraction (LVEF) <50%, electrolyte imbalance, known pulmonary disease, known coronary artery disease, known AF, patients under antiarrhythmic therapy, hyper- or hypothyroidism, moderate and severe valvular diseases, ST and T changes or branch block in electrocardiogram (ECG). The parameters of the VES and non-VES groups were compared. The study was designed and performed in accordance with the principles of the Helsinki Declaration, and the protocol was approved by the ethics committee of Kahramanmaras Sutcu Imam University Faculty of Medicine (approval date: 06.04.2022; decision no: 05). All participants provided written consent.

Definitions

The number of VES per hour is calculated by dividing the total number of ventricular extrasystoles per day by 24. *Non-VES (Control) Group:* <10 VES/hr during the 24-hour Holter monitoring. *Moderate VES Group:* >60 VES/hr during 24-hour Holter monitoring (regardless of the origin of VES). *Severe VES Group:* ≥10.000 VES/ 24 hr. Hypertension (HT) and diabetes mellitus (DM) were defined according to the 2020 ESC and 2022 ADA guidelines [11, 12].

Echocardiographic and ECG parameters

ECGs of all participants were taken after 10 min of rest at a speed of 50 m/s and an amplitude of 10 mm/mV. The ECGs were examined by two cardiologists with the help of magnification. Heart rate, QT, and Tp-e interval data were recorded. For the QT interval, the distance between the onset of the QRS and the end of the T wave was measured. QT interval measurement was made usually from lead V5. However, in case of an imprecise QT measurement in lead V5, lead V4 was used. Maximum and minimum QT values of all leads were noted. The difference is these two values was evaluated as the QT dispersion (QTd). The heart rate-adjusted QT value (cQT) was computed using Bazzet's formula. The Tp-e interval was defined as the distance between the crest of the T wave and the endpoint of the T wave.

All participants underwent echocardiography according to standard European and American Imaging Society guidelines. Parasternal (short and long dimensions) and apical (four-chamber and two-chamber view) views were obtained in the left lateral and decubitus positions. Cardiac measurements (left ventricular end-diastolic diameter, LVEDD; interventricular septal thickness, IVST; posterior wall thickness, PWT) were made via M-mode. The LVEF was calculated using Simpson's method. Mitral flow diastolic patterns were recorded by pulse Doppler echocardiography. The EAT was defined as an area without an echo in the free wall of the right ventricle between the myocardium and pericardium [10]. The right ventricular myocardium was measured in an orthogonal direction during

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three consecutive cardiac cycles and at the end of diastole. An average value was obtained. The aortic ring was the anatomical reference for the measurements. The mostellar formula was used to calculate body surface area. The left ventricular mass index (LVMI) was computed by the Devereux formula:

$0.8 \times \{1.04 \times [[LVEDD + IVSD + PWD]^3 - LVEDD^3]\} + 0.6\,g$

was obtained by dividing the left ventricular mass (computed using the Devereux formula) by the body surface area. It is common to express left ventricular mass in relation to body size to account for variations in body size among individuals.

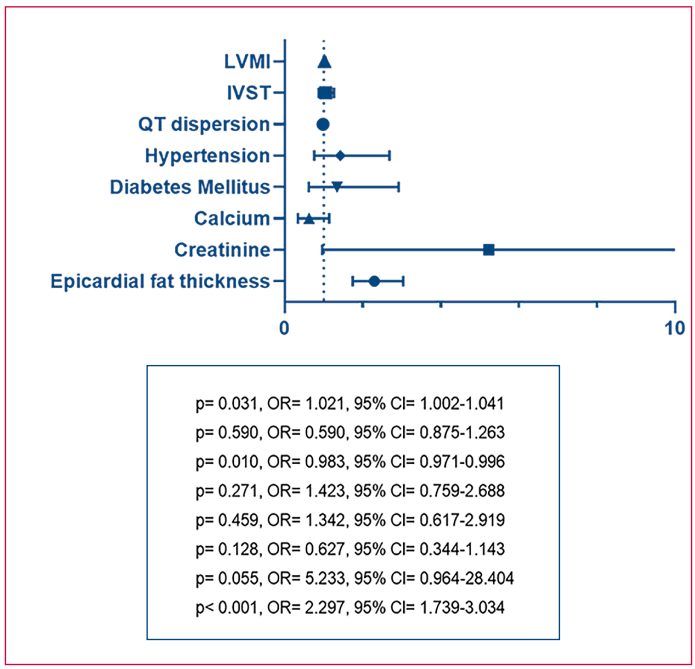
24-hour Holter monitoring

Holter ECG (CardioDay v.2.4 software, Getemed AG, Berlin, Germany) was applied to all participants for 24 hr [13]. The participants were asked to continue their daily work routine and to avoid abnormal exercises while the Holter ECG device was attached. Those records with interference or insufficient recording time were excluded from the study. Total heart rate, atrial beats, and VES amount during 24 hr were calculated automatically. Two cardiologists reviewed all records. Rhythm disturbances misdiagnosed due to interference were corrected, and the total number of beats was recalculated. A VES was defined as a QRS complex >120 ms absent a P wave and followed by an inverted T wave.

Statistical analysis

All data were input into SPSS 22 (SPSS Inc., Chicago, IL, USA), and the variables were analyzed in three groups (non-VES, moderate VES, severe VES). Categorical data is expressed as mean (percentage). Numerical data with normal distribution is presented as mean ± standard deviation (SD). A chi-square test was performed to compare data. A one-way ANOVA test was applied to compare the numerical data of non-VES, moderate VES, and severe VES groups. An LSD test was used for groups that achieved homogeneity of variance in paired comparisons and Games Howell for those who did not in the post-hoc analyze. In addition, logistic regression analysis was used to determine the variables affecting the group with VES. Finally, ROC analysis was performed to determine the cut-off value resulting from logistic regression analysis. A p-value of less than .05 was regarded as significant. The interobserver relationship between the EAT and the ECG data was calculated using the analysis of Bland-Altman, and the coefficients of intra-class correlation were used to assess the intra-observer relationship [14].

Figure 1. Analysis of variables that may affect the presence of VES



Results

Demographic data and laboratory results of the Non-VES, Moderate VES and Severe VES groups are given in Table 1. Calcium concentration and lymphocyte count were higher (p=0.040 and p=0.001, respectively) in the Non-VES group. Creatinine concentration was higher in the Moderate VES group (p=0.043).

A comparison of the echocardiographic and electrocardiographic values of the Non-VES, moderate VES, and severe VES groups is presented in Table 2. EFT in the moderate and severe VES Group was greater (p<0.001) than that of the Control Group, and its p-value indicated that this difference was statistically more significant than the differences detected among the other variables (Table 2). Among the variables showing ventricular repolarization on ECG, analysis between non-VES, moderate VES, and severe VES, all parameters were similar.

In the post hoc analysis between moderate VES and severe VES, EAT was similar (p=0.552) apart from non-VES (p<0.001).

Multivariate logistic regression analysis was performed to examine the effect of variables on VES. Figure 1 shows the odd ratio values and confidence intervals of the variables included in the logistic regression analysis.

Figure 2 illustrates the ROC curves of EAT, LVMI, and QTd. The highest value of AUC in the ROC curve analysis was that of EAT (AUC= 0.735, p<0.001, 95% Cl= 0.674-0.795). EAT predicted the presence of VES at a cut-off value of 4.05 with a sensitivity of 54.5% and a specificity of 81.3%.

Discussion

VES are irregular contractions originating from depolarizations in the ventricles and conducted by pathways outside the standard conduction system. VES occurs in 90% of structural heart diseases, such as dilated cardiomyopathy and coronary artery disease [13]. In healthy individuals, VES can be detected in 1% of standard ECGs and in 40-75% of 24-hour

Table 1. Demographic and laboratory values of the Non-VES (Control), Moderate VES and Severe VES Groups

Parameters	Non-VES =I, n= 134	Moderate VES=II, n= 99	Severe VES =III, n= 33	p	P value. Paired comparison		
					I–II	I–III	II–III
Male, n%	78 (58.2)	50 (50.5)	17 (51.5)	0.473	0.243	0.487	0.920
Age, year	46.2 ± 12.1	49.2 ± 10.5	47.8 ± 11.0	0.146	0.051	0.475	0.547
Body mass index, kg/m^2	29.2 ± 3.4	29.1 ± 4.6	29.1 ± 5.1	0.987	0.983	0.999	0.998
Smoker, n%	31 (23.1)	26 (26.3)	8 (24.2)	0.860	0.583	0.893	0.817
Hypertension, n %	39 (29.1)	23 (23.2)	10 (30.3)	0.550	0.316	0.892	0.417
Diabetes Mellitus, n %	22 (16.4)	14 (14.1)	4 (12.1)	0.786	0.635	0.542	0.770
Creatinine, mg/dL	0.7 ± 0.1	0.8 ± 0.1	0.7 ± 0.1	0.043	0.040	1.000	0.287
Calcium, mg/dl	9.3 ± 0.4	9.1 ± 0.5	9.2 ± 0.3	0.040	0.014	0.855	0.145
Albumin, g/dl	4.0 ± 0.6	4.1 ± 0.5	4.1 ± 0.4	0.231	0.116	0.268	0.973
Total protein, g/dL	6.9 ± 0.5	6.8 ± 0.8	6.8 ± 0.6	0.350	0.152	0.532	0.733
Triglyceride, mg/dL	168.1 ± 75.5	170.1 ± 64.6	164 ± 60.9	0.909	0.824	0.768	0.666
HDL, mg/dL	44.3 ± 10.5	46.6 ± 11.8	43.5 ± 8.0	0.509	0.691	0.885	0.519
LDL, mg/dL	121 ± 37.1	114.5 ± 29.4	109.3 ± 28.2	0.124	0.142	0.074	0.448
Neutrophil, μL	4983.2 ±1584.5	4889.4 ± 1669.5	4677.8 ± 1544.8	0.776	0.986	0.492	0.514
Lymphocyte, μL	2480 ± 830.2	2168.3 ± 830.2	1997.8 ± 741.8	0.001	0.004	0.003	0.302
Platelet, 10^3/μL	265.5 ± 79.5	255.2 ± 79.7	240.6 ± 59	0.222	0.317	0.100	0.350
Hemoglobin, g/dL	13.3 ± 0.9	13.2 ± 2	13.6 ± 2.1	0.495	0.685	0.794	0.567

Data are mean (percentage) or mean ± SD. HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol.
EFT, epicardial fat thickness; IVST, interventricular septal thickness; LVMI, left ventricular mass index.

Table 2. Comparison between electrocardiographic and echocardiographic values of the Control and VES groups.

Parameters	Non-VES =I, n=134	Moderate VES =II, n= 99	Severe VES =III, n=33	p	P value. Paired comparison		
					I–II	I–III	II–III
EFT, mm	3.1 ± 0.9	4.1 ± 1.1	4 ± 1.4	<0.001	<0.001	<0.001	0.552
Heart rate, bpm	73.6 ± 10.3	77 ± 12.9	77.1 ± 12.9	0.083	0.084	0.318	0.997
LVEF, %	57.9 ± 3.4	57.9 ± 3.1	57.2 ± 4.2	0.511	0.962	0.275	0.277
IVST, mm	9.9 ± 1.4	10.6 ± 1.7	10 ± 1.6	0.007	0.002	0.942	0.053
PWT, mm	8.3 ± 1	8.6 ± 1.3	8.2 ± 1.4	0.273	0.276	0.948	0.478
sPAP, mm Hg	19.4 ± 2.4	19.5 ± 2.3	18.6 ± 1.6	0.120	0.794	0.060	0.057
QT, ms	354.7 ± 43.2	348.3 ± 27.1	351.5 ± 31.6	0.364	0.357	0.879	0.867
cQT, ms	391.4 ± 51	393.2 ± 44.9	395.7 ± 33.3	0.860	0.956	0.831	0.942
Tp-e, ms	66.7 ± 16.9	65.7 ± 12.2	70 ± 13.8	0.327	0.856	0.481	0.261
cTp-e, ms	73.8 ± 19.8	74.2 ± 15.4	79.3 ± 17.9	0.267	0.981	0.276	0.319
QTd, ms	47.5 ± 23.4	41.8 ± 21.8	42.1 ± 21.7	0.129	0.058	0.217	0.956
LVMI, g/m^2	71.7 ± 14	75.8 ± 15.9	75.5 ± 14.2	0.081	0.035	0.181	0.921

Data are mean ± SD. EAT, epicardial adipose tissue thickness; IVST, interventricular septal thickness;
LVMI, left ventricular mass index; LVEF, left ventricular ejection fraction; PWT, posterior wall thickness; QTd, QT dispersion;
sPAP, systolic pulmonary artery pressure. EFT, epicardial fat thickness; IVST, interventricular septal thickness; LVMI, left ventricular mass index.

Holter recordings [15]. The etiology of VES, which may cause sustained ventricular arrhythmias and result in sudden cardiac death, has not been completely elucidated. While the rarity of VES cannot be associated with longevity, the frequent occurrence of VES is considered a poor prognosis in terms of mortality [16]. Thus, it is necessary to consider all potential causes of VES, which do occur even in apparently healthy people and can be deadly.

The EAT acts as a protective layer surrounding the heart. However, excessive EAT has been associated with obesity, hypertension, diabetes mellitus, and many cardiovascular diseases [17]. In addition, it has been suggested that EAT may play a role in the etiology of arrhythmias by causing conduction delay between heart layers [18]. Studies showing its role in arrhythmogenic events are limited. The current study showed that an increase in EAT could be an independent predictor of VES. In addition, we reported that LVMI may play a role in the etiology of VES, even though they are weak risk factors.

Obesity is characterized by fat accumulation throughout the body [4]. Obesity has a significant role in the occurrence of arrhythmias [19]. Three different adipose tissues are observed in the body. These are white, beige, and brown adipose tissue. Obesity, with accompanying diseases such as HT and sleep apnea, may be a factor in developing a proarrhythmic effect [20].

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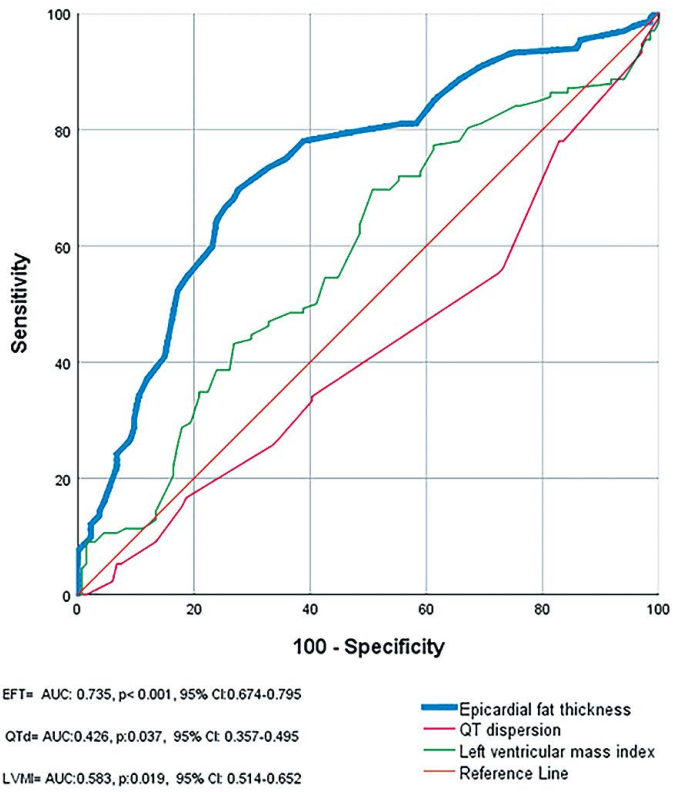
Figure 2. ROC curves of variables that predicted VES



White adipose tissue is under the skin, and brown adipose tissue is in the mediastinal and upper abdomen. Beige adipose tissue is an intermediate form created by the darkening of white adipose. EAT shows a beige character. These adipose tissues play a role in thermoregulation, but they can be held partially responsible for the pathogenesis of many diseases resulting from conditions having an excessive inflammatory effect [21]. The EAT, which can produce inflammatory cytokines such as interleukin-1β, tumor necrosis factor-α, and interleukin-6, may cause some cardiac structural changes that can lead to myocardial fibrosis [22]. The EAT does not have fascia beneath it. It interfaces directly with the myocytes. As such, it may be implicated in the etiology of VES by affecting the action potential of myocardial cells. The EAT on the outer surface of the heart has ganglia containing adrenergic and cholinergic nerve endings. These nerve endings are regulated through the sympathetic and parasympathetic system. Increased thickness of epicardial fat may result in an abundance of these nerve endings. This can cause changes in calcium uptake, which can be modified by the autonomic nervous system, affecting the action potential of the myocardium [23]. Voulgari et al. reported a link between low-grade inflammation and left ventricle arrhythmogenesis in patients with metabolic syndrome [24]. Duncan et al. stated that high tumor necrosis factor-α and interleukin-6 might increase ventricular arrhythmias in patients with sepsis [25]. In addition, EAT is often located in the interventricular and atrioventricular grooves, where it is relatively close to the conduction system [26]. It has been suggested that EAT may be part of the pathogenesis of arrhythmia by forming slow-conduction areas between the epicardium and myocardium. This has been shown to occur by infiltrating into myocardial cells under the visceral epicardium, forming a subepicardial fibro-fatty infiltration. [27].

An increase in EAT is an underlying risk factor for atrial fibrillation. The Framingham Heart study showed that a boost in EAT per cm increased the probability of developing AF by 30%. Epicardial fat volume was also associated with AF burden. Patients with persistent AF had 23% more EAT than those with paroxysmal AF [28]. In addition, EAT has been found to play a role in the frequency of recurrent AF attacks and the development of AF recurrence after cryoablation AF [29]. The mechanism responsible for this situation is that EAT participates in inflammatory processes We think the development and recurrence of VES may be due to similar mechanisms. In our study, the increase in EAT showed a positive correlation with the frequency of VES.

Several similar studies evaluated the relationship between VES and EAT. Shen et al. utilized computed tomography (CT) to analyze EAT and established a link between increased EAT volume and VES [30]. In addition, that study also showed that VES may differ between the region of origin of VES and the density of EAT in that region. Determination of EAT by CT reduces the difference between observers and may provide a more objective imaging method. However, individuals are exposed to radiation during the CT examination. Although the results of our study are similar to those of Shen et al. [5], we think EAT measurement by echocardiography, which is practical and easy to use [30]. Furthermore, echocardiographic imaging also provides information about left ventricular function and heart valves. It can help us to differentiate idiopathic VES from ischemic VES. Another study reported an association between EAT and the frequency of VES [8]. However, that study included only a small number of patients. In addition, frequent VES was defined as more than 10 beats per hour assessed by 24-hour Holter monitoring [8]. In our study, patients with ≥ 10,000 VES were grouped and evaluated separately.

Changes in QT interval, Tp-e, and QTd are considered transmural repolarization parameters, and they have been associated with cardiac diseases, including HT, left ventricular hypertrophy (LVH), coronary artery disease, and mitral valve prolapse [31]. It has been claimed that action potential differences between the cardiac layers during repolarization may increase the possibility of ventricular arrhythmia by paving the way for the ventricular re-entry mechanism [32]. Sudden cardiac death was more likely when QTd was greater than 80 ms In the same way, an increase in the Tp-e interval has been related to sudden cardiac death [33]. The ratio of Tp-e / QT was shown to be more accurate than other parameters in revealing altered transmural distribution of repolarization Tp-e /QT can

be used as a marker of ventricular arrhythmia in patients with Brugada syndrome, long QT syndrome, and cardiomyopathy [34]. In our study, a low QTd value was associated with VES. The lack of a clear cut-off value for the prediction of ventricular arrhythmia by QTd, and the conflicting results in the studies showed no complete consensus about QTd [35].

The incidence of cardiovascular diseases increases with age. The frequency of VES also correlates with age [36]. In the study conducted by Ross et al., the higher incidence of sudden cardiac death in the elderly led to the hypothesis that it may be associated with the frequency of VES [37]. However, previous studies have not fully demonstrated the relationship between increased VES and cardiac death in elderly populations. In a clinical study, patients with VES were treated with antiarrhythmic therapy, and sudden cardiac death was more frequent than in the untreated group [38]. This questioned the place of medical treatment in VES patients with a relatively low arrhythmia burden, where antiarrhythmic drugs may also play a role in forming arrhythmias [39]. In our study, age was similar between the two groups. Long-term prospective studies are needed to investigate antiarrhythmic drugs' relationship with sudden cardiac death.

Serum creatinine is used to assess the severity of both acute and chronic renal failure. Renal failure often causes an electrolyte imbalance. Generally, it is feared that hyperkalemia and hypocalcemia will occur. These electrolyte disruptions have been linked to ventricular arrhythmias and sudden cardiac death [40]. Although creatinine was within the normal range in our study, creatinine, and calcium values differed between the two groups. Altered calcium and creatinine concentrations may also cause VES in patients without renal pathology. Evaluating renal function with much more specific diagnostic methods may help to shed light on this issue.

HT is one of the most common chronic diseases. HT is a systemic condition; untreated, it can result in severe pathology by impairing vital organs. HT disrupts the heart's structure and increases collagen production, triggering fibrosis. The fibrosis formation increases the incidence of ventricular arrhythmias that the re-entry mechanism can trigger by creating a difference in the distribution of electrical conduction in the heart [41]. A study revealed that the LVH caused by HT was associated with the ventricular arrhythmia burden [42]. In endomyocardial biopsies performed in patients with LVH, subendocardial fibrosis, and increased ventricular arrhythmias were observed [43]. Some clinical studies have shown that VES frequency increases in patients with HT. A positive correlation was found between the prevalence of VES and systolic blood pressure in the ARIC study, which included more than 10,000 participants [37]. The Framingham study reported an increased incidence of sudden cardiac death from LVH compared to patients with standard cardiac structure [44]. In light of the pronounced positive correlation between LVH and HT, it is reasonable to suggest that the pathophysiological mechanisms leading to an elevated risk of sudden cardiac death may involve VES associated with hypertension. In our study, HT was identical in the non-VES and the VES groups. However, interventricular septal thickness (IVST) and LMVI were greater in the VES group. This may be because many variables other than HT affect IVST, or HT may have been diagnosed late.

Limitations

This study has some limitations. The total volume of EAT in the heart walls was not measured as this would have required radiological imaging. Second, ECG parameters could have been affected by the emotional state changes of the participants.

Conclusion

EAT is a parameter that is associated with many cardiac diseases and that can be easily evaluated using an echocardiogram during the cardiological examination. This study showed that increased EAT might be associated with VES.

No conflict of interest is reported.

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