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ORIGINAL ARTICLE

Epicardial Adipose Tissue Thickness as a Predictor for New-Onset Atrial Fibrillation in Acute Coronary Syndrome Patients

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ABSTRACT

Background: The most common cardiac arrhythmia, atrial fibrillation (AF), has been associated in prior research with a worse prognosis, both short- and long-term. Although the thickness of the epicardial fat tissue has been associated with the development of AF in earlier clinical trials, little is known about the association between the development of AF and the thickness of the epicardial adipose tissue (EAFT) in the setting of acute coronary syndrome. So we aimed to investigate the relationship between epicardial fat tissue thickness and new-onset atrial fibrillation (AF) development in patients with acute coronary syndrome during in-hospital follow-up.

Methods: This cross-sectional study was carried out in the cardiology department at Zagazig University Hospitals on 100 patients with acute coronary syndrome who were divided into two groups: group (A) patients who didn't develop new-onset AF and group (B) those who developed new-onset AF. EAFT was measured in all patients.

Results: Epicardial adipose fat tissue thickness was statistically significantly higher in ACS patients who developed new-onset AF compared to those who didn't develop AF. Multivariate logistic regression analysis proved that EFT and troponin peak levels were the independent predictors of new-onset AF.

Conclusions: Increased epicardial adipose tissue (EAT) thickness is associated with a higher risk of developing new-onset atrial fibrillation in patients with acute coronary syndrome.

Keywords: Epicardial adipose tissue thickness; New-onset atrial fibrillation; Acute coronary syndrome.

INTRODUCTION

Unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction are among the many cardiovascular conditions that are included in the broad category of acute coronary syndrome (ACS). Acute coronary syndrome is responsible for around one-third of all deaths in the over-35 years age group globally. The mortality rate from coronary artery disease (CAD) has been decreasing in developed countries during the past ten years, but it is still rising in developing countries [1].

Atrial fibrillation (AF) is a well-known complication of acute coronary syndrome, with an incidence that varies from 9.4% to 37% depending on the study group, diagnostic technique, and treatment option chosen [2].

According to earlier research, the onset of AF in NSTEMI patients was linked to worse short- and long-term prognosis [3]. Several clinical factors, such as hypertension (HT), diabetes mellitus (DM), advanced age, female gender, and a lower left ventricular fraction, have been linked to new-onset AF development [2].

The fat tissue that lies between the heart and the visceral pericardium is referred to as epicardial fat tissue (EFT). EFT makes up 20% of the heart's total weight and covers 80% of its surface. EFT is metabolically active, as opposed to subcutaneous fat tissue, and it secrets different hormones, cytokines, and other vasoactive agents that affect atrial fibrillation and ventricular myocardium. Because of its proinflammatory characteristics and its proximity to the heart, the EFT has also been proposed to have a major function in arrhythmia promotion [4].

EFT secretes an adipokine called adiponectin, which possesses anti-atherogenic and anti-inflammatory properties [5]. There is evidence linking the onset of atrial fibrillation (AF) to Adiponectin levels. and have been found to be lower in several of pathologic situations, including ischemic heart disease[2].

According to a recent study, For NSTEMI patients, EFT thickness may be able to predict when AF will start. It was found that EFT was an easy-to-use, affordable, non-invasive method that might be useful for forecasting cardiac arrhythmias [2].

In earlier research, EFT was traditionally calculated using CT or CMR. Recently, echocardiography has been introduced as a reliable method to evaluate and measure EFT which reflects visceral adiposity and cardiometabolic risk. Comparing echocardiography to CT and CMR, it is less expensive and simpler to do. Echocardiography shields patients from radiation exposure and avoids the negative effects of utilizing contrast materials, in contrast to CT [4].

METHODS

This cross-sectional study, which was carried in the cardiology department at Zagazig University Hospitals, involved 100 patients who had acute coronary syndrome. The Institutional Review Board of Zagazig University approved the ethical committee (IRB 10947). There were two sets of patients: group (A) included seventy acute coronary syndrome patients without newly developed atrial fibrillation, and group (B) had thirty acute coronary syndrome patients with newly developed atrial fibrillation (Any new AF during hospitalization).

The current investigation comprised patients with unstable angina, non-ST elevation myocardial infarction (NSTEMI), or ST-elevation myocardial infarction (STEMI) who had acute coronary syndrome and were between the ages of 18 and 70.

We excluded patients less than 18 or more than 70 years, chronic kidney disease, history of chronic coronary syndrome, people with neoplastic, inflammatory, hepatic, or renal dysfunction, patients who had cardiogenic shock and received inotropic agents that potentially cause atrial fibrillation (AF), severe valvular heart diseases, people with a history of atrial fibrillation, stroke history, and chronic heart failure.

Every patient had a full history taking, clinical assessment, and laboratory testing, such as complete blood counts, liver and kidney functions, fasting blood glucose, hemoglobin A1C, cardiac enzymes especially troponin, creatine kinase-myocardial band (CK-MB), fasting lipid, C reactive protein (CRP), lactate dehydrogenase (LDH) and electrocardiography (ECG).

Global Registry of Acute Coronary Events (GRACE) Score assessment:

The risk stratification model known as the "Global Registry of Acute Coronary Events" (GRACE) Score was computed to determine the in-hospital mortality risk for each patient with ACS. It is dependent upon the subsequent factors: elevated levels of cardiac biomarkers, age, killip class, in addition to creatinine level, other parameters include heart rate, systolic blood pressure, admission cardiac arrest, and ST segment deviation. Patients were divided into three groups based on their GRACE risk score: low risk (less than 109 points), intermediate risk (between 109 and 140 points), and high risk (more than 140 points) [6].

Echocardiography

Transthoracic echocardiography (TTE) was carried out at the time of admission in accordance with the European Association of Cardiovascular Imaging and the American Society of Echocardiography. Examinations using tissue, continuous wave, and color Doppler technology were carried out [7].

The following parameters were measured: left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter

(LVESD), interventricular septum thickness, ejection fraction (EF) by M-mode and Simpson method and E/e- Ratio using tissue Doppler. Regional wall motion abnormalities, degree of mitral regurgitation, pulmonary artery pressure, and exclusion of mechanical complications were also be assessed.

Epicardial fat thickness (EFT) measurement: The procedure for measuring EFT thickness by echocardiography was performed according to the guidelines provided by Iacobellis et al. [8]. TTE was used to quantify the free wall of the right ventricle at end-diastole while seeing it from a parasternal long-axis view. The greatest perpendicular distance to the aortic annulus was measured and averaged across three cardiac cycles. EFT stands for the right ventricular free wall hypoechoic region in the parasternal long-axis window. The patient was omitted if the two investigators' measurements differed by more than 5% for any one of the factors. Measurements were averaged if the difference was less than 5% [8].

Coronary angiography and Percutaneous Coronary Intervention

Percutaneous coronary intervention (PCI) is used when necessary and coronary angiography were carried out through either the femoral or radial approach, based on the decision of the operator [9]. Judkins technique was used to perform Conventional left and right coronary artery projections were used to record coronary angiography and traditional coronary angiograms. Two interventional cardiologists then evaluated the angiograms for coronary lesions and possible revascularization.

STATISTICAL ANALYSIS

SPSS 26.0 for Windows was used to tabulate and statistically analyze all of the data (SPSS Inc., Chicago, IL, USA). The tests used were the chi-square (X2) test, the independent T-test, the univariate and multivariate logistic regression analysis, the ROC curve analysis, and the correlation test.

RESULTS

Regarding baseline demographic data, diabetes, smoking and family history of premature CAD were statistically significantly higher in patients with new-onset atrial fibrillation than those without it. Other baseline data, however, did not reveal any noteworthy variations (Table 1).

In laboratory assessment, there was no significant difference concerning complete blood counts, liver and kidney function, LDH, lipid profile and CK-MB, CRP, fasting blood glucose, and HbA1C between the two groups. On the other hand, serum troponin at admission and peak reading were significantly higher in those with newly developed atrial fibrillation than in those without such conditions (Table 1).

Regarding echocardiographic measurements, compared to people without atrial fibrillation, those with new-onset atrial fibrillation had much higher LVESD, E/e', and thicker epicardial adipose tissue. Among those with newly developed atrial fibrillation, the GRACE score was considerably higher compared to those without it (Table 2).

The thickness of the adipose tissue in the epicardium showed a strong positive correlation with HR. LVESD, E/e', LDH, and GRACE scores while there was a significant negative correlation with HDL (Table 3).

Univariate logistic regression analysis proved that a history of DM, smoking history, family history of premature CAD, troponin peak level, and EFT thickness were the independent indicators of AF with a new onset. Multivariate logistic regression analysis, however, demonstrated that EFT and troponin peak levels were independent predictors of new-onset AF (Table 4).

1. At a cut-off value of 10.5 mm, epicardial adipose tissue thickness showed a sensitivity of 81.3% and specificity to predict new-onset AF in patients with acute coronary syndrome (P value 0.004) (Figure 1).

Table 1: Baseline demographic and laboratory data among the studied groups.

		Without new-onset atrial fibrillation (n=70)	New onset atrial fibrillation (n=30)	P value
Age (years)	**Mean ± SD**	55.18±8.38	56.03±9.38	0.072
	Range	40-70	40-69	
BMI	**Mean ± SD**	27.61±1.17	27.51±1.2	0.69
	Range	25.6-29.6	25.5-29.4	
Sex	**Male**	48 (68.6%)	23 (76.7%)	0.47
	Female	22 (31.4%)	7 (23.3%)	
History of DM	**Yes**	17 (24.3%)	28 (93.3%)	**0.001**
	No	53 (75.7%)	2 (6.7%)	
History OF HTN	**Yes**	24 (34.3%)	15 (50%)	0.18
	No	46 (65.7%)	15 (50%)	
History of PAD	**Yes**	19 (27.1%)	26 (86.7%)	0.001
	No	51 (72.9%)	4 (13.3%)	
Smoking history	**Yes**	6 (8.6%)	28 (93.3%)	**0.001**
	No	64 (91.4%)	2 (6.7%)	
Family history of premature CAD	**Yes**	20 (28.6%)	12 (40%)	**0.004**
	No	50 (71.4%)	18 (60%)	
Hb	**Mean ± SD**	**12.31±2.15**	**11.6±1.95**	**0.12**
Wbcs	**Mean ± SD**	**8.3±1.61**	**7.71±1.42**	0.08
Platelets	**Mean ± SD**	**253.2±40.29**	**235.7±42.14**	0.05
Urea	**Mean ± SD**	**39.3±7.83**	**42.38±6.95**	0.06
Creatinine	**Mean ± SD**	**0.78±0.18**	**0.85±0.15**	0.064
AST(GPT)	**Mean ± SD**	**29.1±5.00**	**31.2±4.7**	0.05
ALT(GOT)	**Mean ± SD**	**28.44±5.76**	**30.83**±8.21	0.09
ALP	**Mean ± SD**	**57.02±16.84**	**54.36**±22.51	0.51
LDH	**Mean ± SD**	**166.84±14.51**	**169±14.38**	0.49
CRP	**Mean ± SD**	**1.54±0.2**	**1.66±0.23**	0.025
Total cholesterol	**Mean ± SD**	176.12±11.86	179.86±11.04	0.144
Triglycerides	**Mean ± SD**	229.54±39.18	211.46±50.0	0.05
HDL	**Mean ± SD**	37.9±4.21	38.73±3.52	0.35
LDL	**Mean ± SD**	110.185±18.22	112.26±16.47	0.59
FBS (mg/dL)	**Mean ± SD**	**81±8.96**	**106.83±9.67**	0.001
HbA1C	**Mean ± SD**	**5.01±0.41**	**6.09±0.45**	0.001
S. Troponin at admission	**Mean ± SD**	**4.78±2.47**	**6.21±2.76**	**0.013**
S. Troponin (Peak reading)	**Mean ± SD**	**7.2±2.1**	**9.6±2.9**	**0.001**
CK-MB	**Mean ± SD**	**139.38±48.06**	**129.6±59.97**	0.38

BMI: Body Mass Index, DM: Diabetes mellitus, HTN: hypertension, PAD: Peripheral Arterial Disease, CAD: Coronary artery disease, Hb: Hemoglobin, WBCs: white blood cells, AST: aspartate aminotransferase, ALT: alanine transaminase, ALP: Alkaline phosphatase, LDH: lactate dehydrogenase, FBS: Fasting blood sugar, HGA1C: hemoglobin A1C, CK-MB: creatine kinase-myocardial band, HDL: high-density lipoprotein, LDL: low-density lipoprotein, CRP: C-reactive protein.

Table 2: Echocardiography measurements and clinical examination results among the studied groups.

		Without new-onset atrial fibrillation (n=70)	New onset atrial fibrillation (n=30)	P value
LAD (mm)	**Mean ± SD**	**38.44±2.7**	**37.5**±2.1	0.09
	Range	**33-44**	**34-43**	
LVESD (cm)	**Mean ± SD**	**29.9±3.09**	**33.1±3.8**	**0.001**
	Range	**25-36**	**28-37**	
LVEDD (cm)	**Mean ± SD**	**4.5±1.1**	**5.0±1.3**	0.05
	Range	**3.2-5.9**	**3.5-6.5**	
Thickness of septum	**Mean ± SD**	**0.8±0.1**	**0.84**±0.12	0.08
	Range	**0.7-0.98**	**0.7-0.99**	
Thickness of posterior wall	**Mean ± SD**	**0.82±0.05**	**0.84±0.04**	0.05
	Range	**0.74-0.91**	**0.75-0.91**	
EF by SIMPSON	**Mean ± SD**	**58.32±4.85**	**56.93±4.74**	0.18
	Range	**48-67**	**49-66**	
E/e'	**Mean ± SD**	**5.40±0.46**	**9.02±0.32**	**0.001**
	Range	**4.2-6.3**	**8.2-9.5**	
Epicardial adipose tissue thickness (mm)	**Mean ± SD**	7.6±1.73	9.15±1.92	**0.001**
	Range	5-11	6-11	
SBP	**Mean ± SD**	**132.86±14.09**	**128±15.17**	**0.12**
	Range	110-160	110-160	
DBP	**Mean ± SD**	84.57±14.46	79.66±13.45	0.11
	Range	70-90	70-80	
HR	**Mean ± SD**	78.65±5.62	144.53±10.3	**0.001**
	Range	70-90	131-163	
Site of MI	**Inferolateral**	9 (12.9%)	3 (10%)	0.9
	Anterolateral	9 (12.9%)	4 (13.3%)	
	Lateral	3 (4.3%)	1 (3.3%)	
	Anterior	33 (47.1%)	15 (50%)	
	Inferior	16 (22.9%)	7 (23.4%)	
Culprit vessel	**LCX**	15 (21.4%)	6 (20%)	0.77
	LAD	45 (64.3%)	18 (60%)	
	RCA	10 (14.3%)	6 (20%)	
GRACE SCORE	Mean ± SD	**126.55±19.0**	**162.8±17.48**	0.001

LAD; left atrium diameter, LVESD: left ventricular end-systolic diameter, LVEDD: Left ventricular end-diastolic diameter, EF: Ejection fraction, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate.
MI: Myocardial infarction, LCX: left circumflex artery, LAD: left anterior descending artery, RCA: right coronary artery.
GRACE SCORE; Global Registry of Acute Coronary Events Score

Table 3: Correlation between epicardial adipose tissue thickness and different parameters among the studied Groups.

Epicardial adipose tissue thickness(mm)		
	r	P value
Age	-0.083	0.412
BMI	0.15	0.137
HR	0.376**	**0.001**
SBP	0.055	0.589
DBP	-0.199	0.051
LAD (mm)	0.185	0.274
LVESD (cm)	0.250*	**0.012**
LVEDD (cm)	0.07	0.488
Thickness of septum	-0.023	0.07
Thickness of posterior wall	-0.047	0.643
EF by M-mode	-0.109	0.281
EF by SIMPSON	-0.099	0.325
E/e'	0.463*	**0.015**
Hb	0.126	0.213
WBCs	0.196	0.05
Platelets	0.004	0.97
Urea	-0.109-	0.281
Creatinine	0.063	0.531
AST(GPT)	0.17	0.091
ALT(GOT)	-0.190-	0.058
ALP	-0.188-	0.061
LDH	0.214*	**0.033**
FBS mg/dL	0.133	0.186
HGA1C	-0.108-	0.284
S.Troponin at admission	-0.086-	0.395
S. Troponin (Peak reading)	-0.109	0.279
CK-MB	0.148	0.072
Total cholesterol	-0.059	0.563
HDL	-0.380**	**0.001**
LDL	-0.087-	0.389
Triglycerides	0.092	0.361
CRP	-0.072-	0.48
GRACE score	0.452**	**0.001**

BMI: Body Mass Index, DM: Diabetes mellitus, HT: hypertension, PAD: Peripheral Arterial Disease, CAD: Coronary artery disease, HR: heart rate, BP: blood pressure, MI: Myocardial infarction, LAD: left anterior descending artery, LVESD: left ventricular end-systolic diameter, LVEDD: Left ventricular end-diastolic diameter, EF: Ejection fraction, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: Hemoglobin, Wbcs: white blood cells, AST: aspartate aminotransferase, ALT: alanine transaminase, ALP: Alkaline phosphatase, LDH: lactate dehydrogenase, FBS: Fasting blood sugar, HGA1C: hemoglobin A1C, CK-MB: creatine kinase-myocardial band, HDL: high-density lipoprotein, LDL: low-density lipoprotein, CRP: C-reactive protein, AF: atrial fibrillation.

Table 4: Univariate and multivariate logistic regression analysis for prediction of new-onset AF.

	Univariate				Multivariate			
	Exp(B)	95% C.I		P value	Exp(B)	95% C.I		P value
		Lower	upper			Lower	upper	
Sex	1.50	0.562	4.033	0.415				
History of DM	22.4	4.22	135.3	**0.04**				
history OF HT	1.92	0.80	4.57	0.14				
History of PAD	0.413	0.127	1.34	0.141				
Smoking history	46.191	5.492	388.48	**0.001**				
Family history of premature CAD	1.572	0.275	8.976	**0.01**				
culprit Vessel	0.734	0.385	1.398	0.347				
Site of MI	0.599	0.397	0.901	0.342				
Troponin peak level	1.3	1.2	4.5	**0.03**	2.2	1.7	5.9	**0.001**
EAT Thickness	3.6	1.22	7.65	**0.003**	6.21	1.95	11.87	**0.001**

DM: Diabetes mellitus, HT: hypertension, PAD: Peripheral Arterial Disease, CAD: Coronary artery disease, MI: Myocardial infarction.

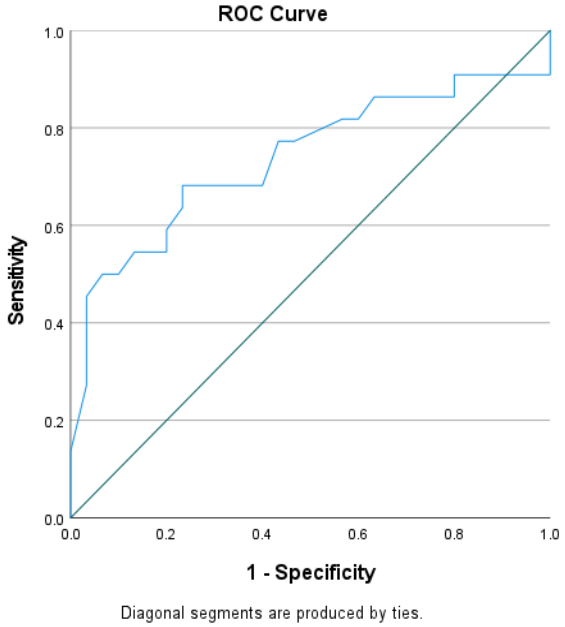

Figure 1: Receiver observing characteristic analysis of epicardial adipose tissue thickness for prediction of new-onset AF

DISCUSSION

Atrial Fibrillation (AF) is one of the well-known complications for patients who have acute coronary syndrome (ACS), which can occur in 9.4% to 37% of cases, depending on the research group, diagnostic technique, and course of treatment. According to earlier research, the onset of AF in ACS patients was

linked to a worsening prognosis over the short- and long-term [3].

Several clinical variables, such as hypertension, diabetes mellitus (DM), advanced age, the quantity of damaged coronary arteries, and a decreased left ventricular systolic performance, have been connected to atrial fibrillation (AF) development. The fat tissue situated between the heart and the visceral pericardium is known as epicardial fat tissue (EFT). Prior research in the clinical sector has shown a substantial correlation between the beginning of AF and EFT thickness. [10].

This study aimed to assess the association between the "Global Registry of Acute Coronary Events" (GRACE) and the thickness of the epicardial fat tissue and to assess the predictive significance of epicardial fat tissue thickness for the onset of atrial fibrillation in hospitalized acute coronary syndrome patients.

In the present study, there was significant difference between the two groups with respect to smoking history, family history of premature CAD, and diabetes, all of which were associated with a higher risk of new-onset atrial fibrillation in ACS patients.

There was no considerable difference in the current study between both groups regarding to site of MI and culprit vessel. This was in line with the findings of Nakanishi et al. [11] who discovered that there was no discernible variation between individuals with acute coronary syndrome with new onset AF and those without regarding to affected culprit vessel.

The two groups in the current study did not significantly differ in terms of CK-MP; however, the individuals with new onset atrial fibrillation had significantly higher serum troponin levels at admission and peak reading than the non-affiliated group. This aligns with Eren et al. [2], who found that elevated troponin levels seemed to be linked to a higher risk of arrhythmia development in the AF group.

GRACE score in our study has been demonstrated to be higher in ACS patients who experienced new-onset atrial fibrillation than those who did not.

Given that the majority of the GRACE risk score's characteristics have been determined to be the main risk factors for AF, this connection seems reasonable. According to Global Utilization of Streptokinase and TPA (alteplase) for Occluded Coronary Arteries (GUSTO-I) trial, age, higher Killip class, greater heart rate, and lower SBP were identified as risk factors and independent predictors for new onset atrial fibrillation in ACS setting [12].

Regarding echocardiography, our study showed that E/e’ was much greater in cases of atrial fibrillation with a recent onset in contrast to people who do not have it. Consistent with our finding, Takagi et al. [13] showed that higher E/e′ is linked to development of new onset AF. In addition, Darweesh et al. [14] who found that atrial fibrillation patients had significantly higher E/e′ but they found a lower mitral E deceleration time, also corroborated our findings.

Our study's primary discovery was the correlation between the thickness of the epicardial tissue and new-onset AF in ACS patients. When comparing individuals with new onset atrial fibrillation to those without, EAT was considerably higher in the former group. Furthermore, our study declared by multivariate logistic regression that the independent predictors of new onset AF were EFT and troponin peak level.

Numerous investigations have demonstrated a similar association link between occurrence and severity of AF and the higher EFT thickness [15, 16]. This was in line with the findings of Eren et al. [2], who observed that the AF group's EFT was thicker. Furthermore, in individuals with paroxysmal AF, Duman et al. [17] discovered a strong correlation between the onset of AF and EFT thickness. Nakanishi et al. [11] presented in accordance with previous investigations that peri-atrial EAT volume is a predictor of new-onset AF.

In addition, in Framingham Heart study, for each 1 (SD) increase in pericardial fat thickness, AF risk was found to be 30% higher. Furthermore, there was strong association between pericardial fat tissue thickness and AF burden. Compared to

paroxysmal AF, pericardial fat tissue was higher by 23% in persistent AF. A large meta-analysis of sixty-three observational studies conducted on 352,275 patients also reported that for each 1 (SD) increased EAT volume, there was a 2.2-fold higher chance of chronic AF than paroxysmal AF [18, 19].

It has been proposed that the EAT's proinflammatory characteristics and close anatomical proximity to the heart contribute significantly to arrhythmogenesis. EAT experiences a shift in the substrate's phenotype from protective to inflammatory under pathological circumstances. For instance, EAT has a more aggressive proinflammatory behavior in CAD patients compared to normal persons. After adjusting Proinflammatory cytokines including Concomitant hypertension, diabetes, dyslipidemia, and body mass index have been demonstrated to be associated with elevated levels of tumour necrosis factors-α (TNF-α), interleukin (IL)-6, and monocyte chemoattractant protein (MCP-1) (BMI), while anti-inflammatory adiponectin has been downregulated. Additionally, EAT plays a part in the regulation of other triggers, such as metabolic and physiological triggers, which result in atrial fibrillation. [20-23].

The current investigation found a strong positive association between the GRACE score and the thickness of the epicardial adipose tissue. Several investigations have shown the relationship between EFT and significant adverse cardiovascular outcomes [24].

CONCLUSION

Our study suggests that patients with acute coronary syndrome may be more likely to develop new-onset atrial fibrillation if their epicardial adipose tissue (EAT) thickness is greater Moreover, there was a strong positive correlation observed between the GRACE risk score and EAT thickness, indicating a possible association between EAT and a worse prognosis in individuals with acute coronary syndrome.

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