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CMR assessment of epicardial edipose tissue in relation to myocardial inflammation and fibrosis in patients with new-onset atrial arrhythmias after STEMI

Yanfei Ren[1], Xinjia Du[1], Maochen Li[1], Lei Chen[2], Jiahua Liu[1], Yixuan Wu[1] and Yuan Lu[1*]

Abstract

Background Previous studies have shown that epicardial edipose tissue(EAT) appears to be associated with myocardial inflammation and fibrosis, but this is not clear in patients with new-onset atrial arrhythmias after STEMI. The present study focused on using CMR to assess the association of epicardial fat with myocardial inflammation and fibrosis and its predictive value in patients with new-onset atrial arrhythmias after STEMI.

Methods This was a single-centre, retrospective study. We consecutively selected patients who completed CMR during their hospitalisation for PCI after STEMI from May 2019-January 2023, and then underwent regular follow-up, grouped by the presence or absence of new atrial arrhythmias, and enrolled patients were divided into atrial arrhythmia and non-atrial arrhythmia groups.

Results In the atrial arrhythmia group, age, heart rate, Peak hs-TnT, PeakNT-proBNP, EATV, LAES, LAED, T1 native, T1*, ECV, and T2 were higher than those in the non-atrial arrhythmia group, and LVEF was lower than those in the non-atrial arrhythmia group. EATV showed a positive and significant correlation with T1native, T1*, ECV, and T2. (T1 native: $r=0.476$,$p<0.001$; ECV: $r=0.529$,$p<0.001$; T1*: $r=0.467$,$p<0.001$; T2: $r=0.538$,$p<0.001$). Multifactorial logistic regression analysis showed age, LVEF, EATV, T1*,ECV, T2 as independent risk factors for atrial arrhythmia. ($p<0.05$) ROC analysis showed that the AUC for age was 0.568; AUC for LVEF was 0.656; AUC for EATV was 0.768; AUC for ECV was 0.705; AUC for T1* was 0.612; and AUC for T2 was 0.772.

Conclusion In patients with STEMI, EAT is associated with myocardial inflammation, fibrosis. Age, LVEF, EATV, T1*,ECV, T2 are independent risk factors for new onset atrial arrhythmias and have good predictive value.

Keywords Epicardial adipose tissue, T1mapping, ECV, T2mapping, CMR, Atrial arrhythmia

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Introduction

Acute ST-segment elevation myocardial infarction (STEMI) is one of the manifestations of coronary artery disease, which has a high morbidity and mortality rate, and the current situation is grim [1]; atrial arrhythmia is a common cardiac arrhythmia, with atrial fibrillation(AF) being the more common one, which has a high incidence rate and poor prognosis, and most of the researches are also in the process of continuing to explore the problem [2]; Coronary heart disease is being reported in an increasing number of patients with atrial fibrillation, with recent studies even reporting that coronary heart disease is present in almost 70% of all patients. Endothelial dysfunction and inflammation are core pathogenesis in the context of common risk factors for atrial fibrillation and coronary heart disease [3]. in the face of the treatment of the patients with new-onset atrial arrhythmias after STEMI, we have a tremendous challenges, such as difficulties in rhythm management, risk of antithrombotic bleeding, and difficulties in the use of antiarrhythmic drugs, we should pay more attention to this population. epicardial adipose tissue (EAT) is a metabolically active substance between the myocardium and the epicardium, which can paracrine or vascularly secrete proactive and inflammatory factors to destroy the myocardium, and also promote myocardial fibrosis through direct infiltration of the myocardium, and it is now gradually being discovered that EAT has a potential role in inflammation and myocardial fibrosis [4, 5]. Cardiac magnetic resonance (CMR) has become a new tool for assessing disease and is widely used in cardiovascular disease [6], and the CMR -T1 mapping/ECV technique has diagnostic value in myocardial fibrosis by early non-invasive quantification and monitoring of focal and diffuse myocardial lesions [7]. CMR-T2mapping scholars have also made many discoveries that the pathological basis of T2 is inflammation or edema, and this technique is beginning to be used in acute ischemic myocardial injury, intramyocardial hemorrhage, myocardial infarction, and other diseases, which is helpful for patient treatment [8]; Other studies have shown that CMR is the "gold standard" for measuring epicardial fat (EAT) in atrial fibrillation, and EAT can increase atrial muscle ion flow and regulate atrial electrophysiological activities. The fat infiltration in the atrium induces the degeneration of adjacent cardiocytes, which leads to abnormal automation, interferes with atrial conduction and generates obstacles to activate wave front, which enhances the occurrence of reentry circuit. This can lead to higher atrial arrhythmias [9]. Currently EAT seems to be associated with inflammation and myocardial fibrosis, but it is not known in patients with new onset atrial arrhythmias after STEMI.This study focused on the use of CMR to assess the relationship between epicardial fat and myocardial inflammation and fibrosis and its predictive value in patients with new-onset atrial arrhythmias after STEMI.

Materials and methods

Study population

This was a single-centre, retrospective study. 550 consecutive STEMI patients were selected to complete CMR after PCI during hospitalisation from May 2019-January 2023(XYFY2023-KL435-01). Inclusion criteria: all enrolled patients underwent PCI within 12 h of the onset of the disease; STEMI patients discharged from the clinic were followed up regularly for 1, 3, 6, 9, and 12 months using Soss ambulocardiac monitoring (patients with symptoms/arriving at the follow-up date were fitted with 3–7 days of ambulocardiac monitoring for arrhythmia, and those who missed appointments or were not monitored on time were excluded ($n = 50$)). Exclusion criteria: unclear or incomplete CMR images ($n = 10$); severe valvular disease ($n = 1$), moderate to large amount of pericardial effusion, congenital heart disease, old myocardial infarction ($n = 3$); severe hepatic and renal insufficiency ($n = 1$), severe systemic inflammation, hyperthyroidism and subclinical hyperthyroidism ($n = 2$), severe anaemia; antiarrhythmic medications ($n = 4$), previous history of atrial arrhythmia ($n = 1$). Twenty-two patients were excluded from the enrolment, According to the inclusion criteria and power analysis, 528 patients were enrolled in the study, divided into the atrial arrhythmia group ($n = 88$, including 17 patients with atrial fibrillation and 71 patients with atrial tachycardia) and the non-atrial arrhythmia group ($n = 440$).(Figure.1)(Clinical trial number: not applicable.)

The study was approved by the local ethics committee, and patients waived the requirement to sign a written consent form due to the low risk of the study in accordance with relevant IRB regulatory guidelines.

General clinical information

Name, gender, BMI, age, smoking, hypertension, diabetes mellitus, use of atrial arrhythmia medication, admission systolic blood pressure, diastolic blood pressure, heart rate, troponin (Peak hs-TNT), brain natriuretic peptide precursor (PeakNT-proBNP), C-reactive protein (CRP), triglyceride (TG), total cholesterol (TC), eGFR, glycated haemoglobin (HbA1c), ejection fraction(LVEF) and other information.

CMR scanning

All patients underwent a 3.0 T magnetic resonance scanner (Ingenia 3.0 T, Philips, The Netherlands), and CMR was performed during hospitalisation (cardiac magnetic resonance imaging scans were performed at a median of 4 [IQR:3–6] days); LV long-axis and short-axis (coverage from basal to apical segments) movies were obtained

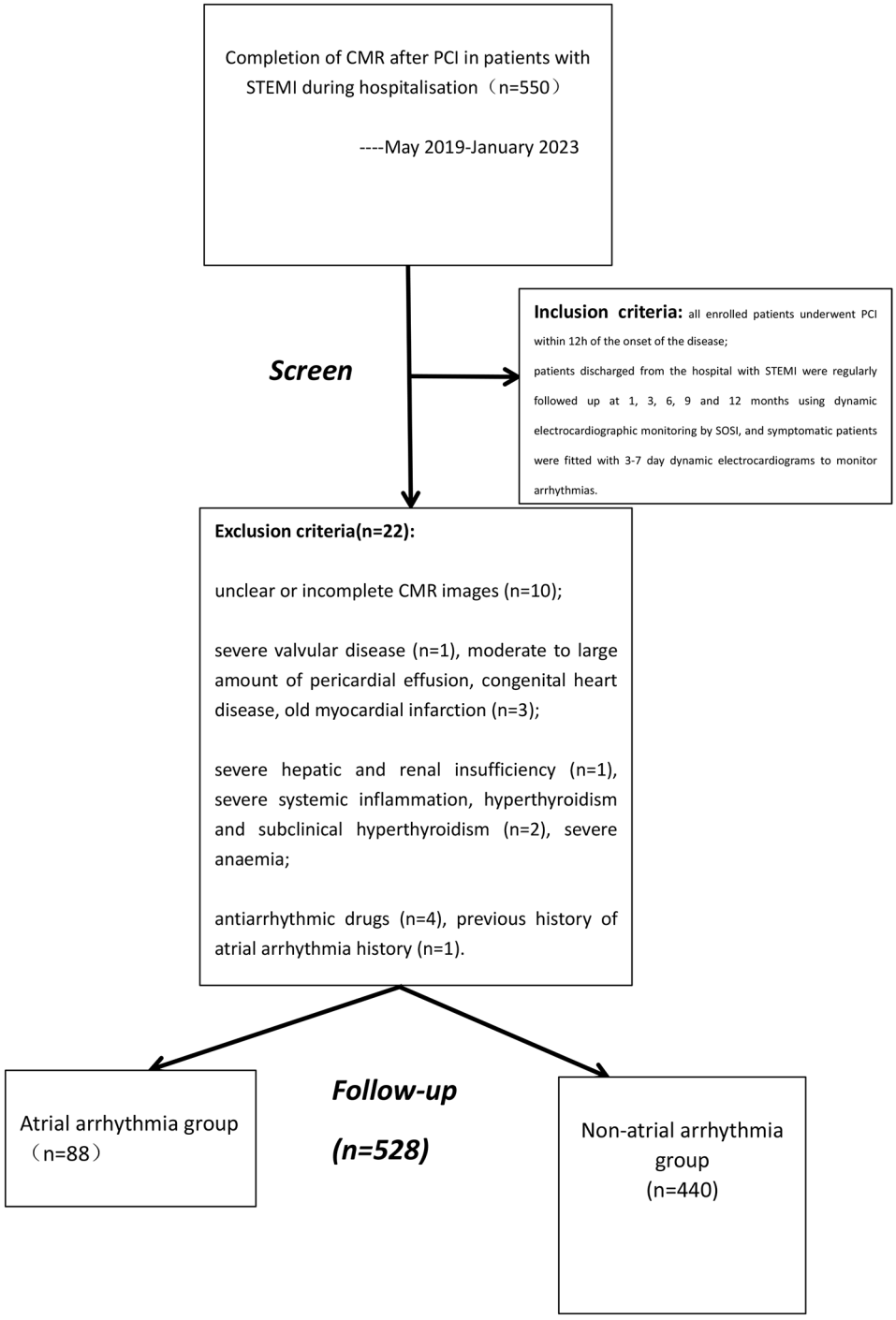

Fig. 1 Flowchart of the study

using balanced Steady State Free (b-SSFP) sequences. T1 mapping was performed in basal, middle and apical slices of the short axis of the left ventricle before and after gadolinium contrast administration using a modified Look-Locker inversion recovery (MOLLI) sequence. ECV(extracellular volume) images were automatically generated from the T1 values of the myocardium and blood pools before and after enhancement in conjunction with the input hematocrit(HCT) [10]. Single breath-hold, black blood preparations of ECG-triggered spin-echo multiple-echo sequences were obtained for T2 mapping in the whole myocardium including (basal, mid-ventricular and apical on) respectively.

$$ECV = (1 - Hct)\frac{\left(\frac{1}{T1_{myo\ post}} - \frac{1}{T1_{myo\ pre}}\right)}{\left(\frac{1}{T1_{blood\ post}} - \frac{1}{T1_{blood\ pre}}\right)}$$

MR image analysis

CMR images were analysed using Cvi42 (v5.13.5, Circle V vascular Imaging, Canada). The long axis module obtained the basic parameters of cardiac function, left atrial systolic volume (LAES) and left atrial diastolic volume (LAED); The tissue characterisation module manually outlines the myocardial and pericardial visceral layers, intermediate to obtain the overall epicardial edipose tissue volume (EATV). the T1mapping module obtained the overall myocardial initial T1 value (T1 native) and the post-contrast T1 value (T1*), which was input into the HCT ECV images were automatically obtained; T2mapping module obtained T2 values and images. The images were measured manually by two attending cardiologists with more than three years of experience in CMR imaging studies.(Fig. 2).

Statistical analyses

SPSS25.0 software was used to analyse the data collected from the patients, we carried out the K-S test for normal distribution of all the data, and the measurement data obeying the normal distribution were expressed as the mean ± standard deviation ($\bar{x} \pm s$) using the independent samples t-test; those not obeying the normal distribution were expressed as the median, quartiles, and the categorical data were tested using the X^2 test; the correlation between EAT and T1native, T1*, ECV, and T2 was analysed by the Pearson correlation analysis. Pearson's correlation was used to analyse the correlation between EAT and T1native, T1*, ECV and T2; all the indicators with meaningful differences between the two groups were included in the one-way logistic regression analysis; all the indicators with meaningful differences in the one-way logistic regression were included in the one-way logistic regression analysis; all those with meaningful differences in the one-way logistic regression were used to search for the independent risk factors of atrial arrhythmia using the stepwise multifactorial logistic regression method; and all the obtained independent risk factors were included in the ROC curve. ROC curves were used to investigate the predictive value of atrial arrhythmias. $p < 0.05$ was considered statistically significant.

Results

Comparison of general data

Atrial arrhythmia group age (61.63 ± 11.07 VS.58.79 ± 12.00) years, heart rate(81.18 ± 15.37VS.77.54 ± 12.29)Beats/min, Peakhs-TnT(2870.5(916,9519.4)VS.1566(475.25,4246.75)) ng/mL, PeakNT-proBNP(1791.85(711.43,4792.14) VS.907.85(393.73,3064.76))(pg/mL), EATV(145.66 ± 12.52VS.113.96 ± 34.40) mL, LAES (42.32 ± 20.87 VS. 35.98 ± 13.87) mL, LAED (68.29 ± 23.36 VS. 62.87 ± 19.07) mL, T1 native (1351.83 ± 81.62 VS. 1319.23 ± 64.54) ms, T1*(496.59 ± 111.07VS.421.06 ± 96.61)ms, ECV(30.88 ± 8.11VS.27.89 ± 4.92)%, T2 (88.52 ± 24.37VS.64.23 ± 23.04) ms was higher than the non-atrial arrhythmia group, and LVEF (50.57 ± 5.56VS.54.22 ± 5.31)% was lower than that of it ($p < 0.05$).(Table 1; Figure 3).

Correlation of EATV with T1native, T1*,ECV, T2

EATV was significantly positively correlated with T1native, T1*,ECV, T2. (T1 native: $r = 0.476, p < 0.001$;ECV: $r = 0.529, p < 0.001$;T1*: $r = 0.467, p < 0.001$;T2: $r = 0.538, p < 0.001$)(Table 2).

Logistic regression analysis

Indicators that differed between the two groups were used as independent variables, and atrial arrhythmia was used as the dependent variable; univariate logistic regression analysis showed that the risk factors for atrial arrhythmia were age, heart rate, Peak hs-TnT, PeakNT-proBNP, LVEF, EATV, LAES, LAED, T1 native, T1*,ECV, T2.($p < 0.05$).

Indicators with significance in one-way logistic regression analysis were used as independent variables, and atrial arrhythmia was the dependent variable; analyses using a multifactorial logistic regression stepwise method showed that age, LVEF, EATV, T1*,ECV, T2 were independent risk factors for atrial arrhythmia. ($p < 0.05$) (Table 3).

ROC curve analysis

From the above, we obtained that age, LVEF, EATV, T1*, ECV, and T2 were independent risk factors for atrial arrhythmia. ROC analysis was performed to obtain: the area under the curve (AUC) for age was 0.568 with a cut-off value of 58.5 years, the AUC for LVEF was 0.656 with a cut-off value of 50%, the AUC for EATV was 0.768 with a cut-off value of 126.87 mL, the AUC for T1* was 0.612 with a cut-off value of 507.32 ms, and the AUC for ECV was 0.705 with a cut-off value of 507.32 ms. 0.612 with a cut-off value of 30.32%,T2 had an AUC of 0.772 with a cut-off value of 75.50ms.(Fig. 4).

Discussion

To our knowledge, this is the first study to discuss the relationship between EAT and myocardial inflammation and fibrosis and its predictive value in a population with new-onset atrial arrhythmias using CMR to assess T1, T1*, ECV, and T2 laterally in a STEMI population.The main findings of this paper are: (1) EATV, LAES, LAED,

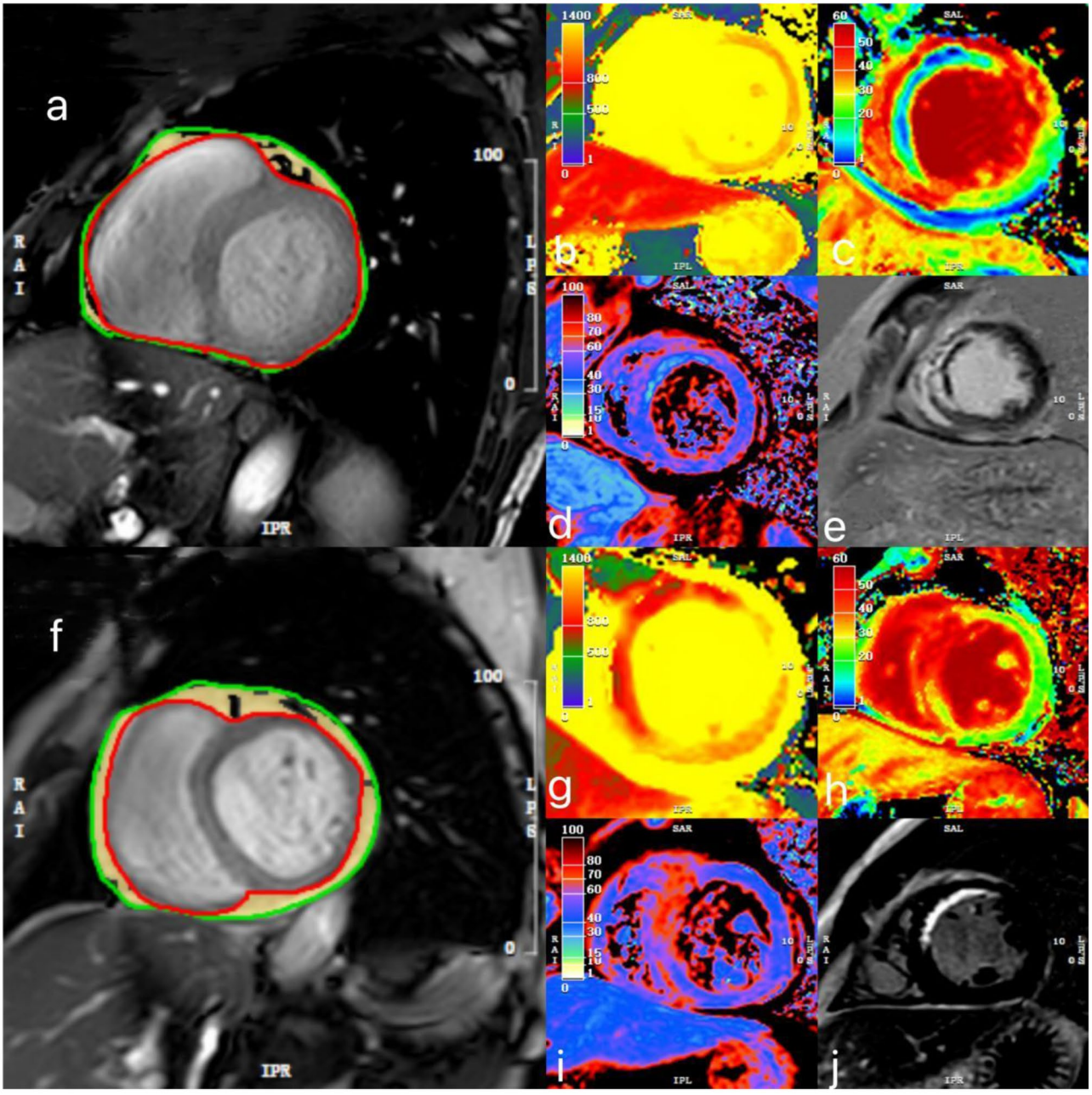

Fig. 2 CMR image analysis. (**a-e**) A patient with non-atrial arrhythmia: a-epicardial fat image (yellow part); b-T1mapping image; c-ECV image; d-T2mapping image; e-LGE image. EATV = 107 ml; T1 native = 1130ms; T1*=578ms; ECV = 31%;T2 = 56ms. (**f-j**) A patient with atrial arrhythmia: f-epicardial fat image (yellow part); g-T1mapping image; h-ECV image; i-T2mapping image; j-LGE image. EATV = 156 ml; T1 native = 1425ms; T1*=594ms; ECV = 39%;T2 = 63ms

T1 native, T1*,ECV, T2 were greater in the atrial arrhythmia group compared with the non-atrial arrhythmia group; (2) EATV had a significant positive correlation with T1 native, T1*,ECV, T2; and (3) EATV, T1*,ECV, T2 were independent risk factors and had a good predictive value for atrial arrhythmias.

EAT includes myocardial inflammation, fibrosis, oxidative stress and fatty infiltration, and the tight junctions between EAT and the myocardium allow EAT to penetrate and produce fatty infiltration of the atrial myocardium, which alters the electrophysiological properties of the atria [11]. It has been shown that peri-atrial EAT, through direct infiltration of the atrial wall and strong conduction of paracrine fibrotic signals can affect the neighbouring myocardium, ultimately leading to electrical and anatomical remodelling of the atria Stimulated AF [12].In a paper that EAT may play a role in coronavirus-related cardiac syndrome in 2019, it was described that EAT of the coronary artery and left atrium are involved in the pathogenesis of coronary artery disease

Table 1 Comparison of the two groups of general data

	Atrial-arrhythmia group($n=88$)	Non-Atrial arrhythmia group($n=440$)	X^2/t/z	*P* Value
Basic Datas				
Age (years)	61.63 ± 11.07	58.79 ± 12.00	-2.061	0.040
BMI(Kg/m^2)	24.95 ± 4.61	27.31 ± 28.06	0.787	0.526
Systolic-pressure (mmHg)	125.71 ± 23.49	128.67 ± 19.81	1.239	0.216
Diastolic-pressure (mmHg)	78.84 ± 14.64	80.40 ± 13.68	0.966	0.335
Heart-rate (Beats/min)	81.18 ± 15.37	77.54 ± 12.29	-2.442	0.016
Peak hs-TnT (ng/L)	2870.5(916,9519.4)	1566(475.25,4246.75)	-2.875	0.004
PeakNT-proBNP (pg/mL)	1791.85 (711.43,4792.14)	907.85 (393.73,3064.76)	-4.950	0.001
C-reactive protein(mg/dL)	36.63 ± 41.78	28.76 ± 41.12	-1.634	0.103
Triglyceride(mmol/L)	1.51 ± 1.22	1.72 ± 1.58	1.182	0.238
total cholesterol(mmol/L)	4.30 ± 0.96	4.45 ± 1.09	1.216	0.224
eGFR(ml/min)	100.40 ± 22.84	102.72 ± 18.56	1.028	0.304
HbA1c(%)	6.64 ± 1.46	6.57 ± 1.34	-0.388	0.698
LVEF(%)	50.57 ± 5.56	54.22 ± 5.31	5.836	0.001
Gender(male/female)	61/27	306/134	0.002	0.966
Smoking	$n=32$(36.4)	$n=216$(49.1)	4.769	0.052
Hypertension	$n=45$(51.1)	$n=166$(37.7)	5.496	0.124
Diabetes	$n=27$(30.7)	$n=88$(20.0)	4.911	0.078
Statin	$n=83$(94.3)	$n=420$(95.5)	0.034	0.647
β-blockers	$n=71$(80.7)	$n=356$(80.9)	0.002	0.961
CMR parameters				
EATV (mL)	145.66 ± 12.52	113.96 ± 34.40	-8.525	0.001
LAED (mL)	68.29 ± 23.36	62.87 ± 19.07	-2.339	0.020
LAES (mL)	42.32 ± 20.87	35.98 ± 13.87	-3.562	0.001
T1 native(ms)	1351.83 ± 81.62	1319.23 ± 64.54	-4.125	0.001
T1*(ms)	496.59 ± 111.07	421.06 ± 96.61	-6.254	0.001
ECV(%)	30.88 ± 8.11	27.89 ± 4.92	-4.592	0.001
T2(ms)	88.52 ± 24.37	64.23 ± 23.04	-8.942	0.001

troponin (Peak hs-TNT), brain natriuretic peptide precursor (PeakNT-proBNP), C-reactive protein (CRP), triglyceride (TG), total cholesterol (TC), eGFR, glycated haemoglobin (HbA1c), ejection fraction(LVEF), left atrial systolic volume (LAES) and left atrial diastolic volume (LAED), epicardial adipose tissue volume (EATV), post-contrast T1 value (T1*),extracellular volume(ECV)

and atrial fibrillation, respectively, and EAT may be a marker for future diagnosis of arrhythmia and a therapeutic target for clinical drugs [13].In our study EAT was high in the population of new-onset atrial arrhythmias and was a good predictive value as its independent risk factor, which is in line with most of the studies. Shao.et al. found that EAT secretes pro-fibrotic adipokines that promote atrial and myocardial fibrosis in a multi-parametric CMR imaging study of structural and functional changes in the myocardium of diabetic minipigs [14], and in the population of new-onset atrial arrhythmias in our study STEMI In our study of new-onset atrial arrhythmias after STEMI, we obtained a significant positive correlation between EAT and T1 native, T1*, which is consistent with their validation, and laterally inferred that epicardial adiposity may be associated with myocardial fibrosis in our study population; Some scholars chose heart failure patients to explore EAT and left atrial and left ventricular function and obtained that EAT secretion of adipokines may lead to atrial myocardial fibrosis and further atrial dysfunction [15]; in line with most of the studies, we obtained results similar to theirs, with the difference probably lying in the selection of the study population and methodology. ECV is a more precise criterion for myocardial fibrosis [16], and in our study EAT and ECV had a high correlation, and in Lin et al. high EAT and high ECV were associated with an increased risk of incident acute coronary syndromes and were significantly correlated [17], which is in line with our study, differing in that we studied a population of patients with new-onset atrial arrhythmias after STEMI. They mainly studied patients with heart failure. Interestingly in an article studying early myocardial tissue remodelling in adult obesity and its relationship with regional adipose tissue distribution and ectopic fat deposition, EAT was elevated but ECV was decreased in mild to moderately obese patients, in contrast to our study, where ECV was increased and extracellular mesenchymal dilatation was more pronounced than cardiomyocyte hypertrophy in severely obese individuals, again in agreement with us [18]. We considered the following possible reasons: (1) the study population and sample size were different; (2)

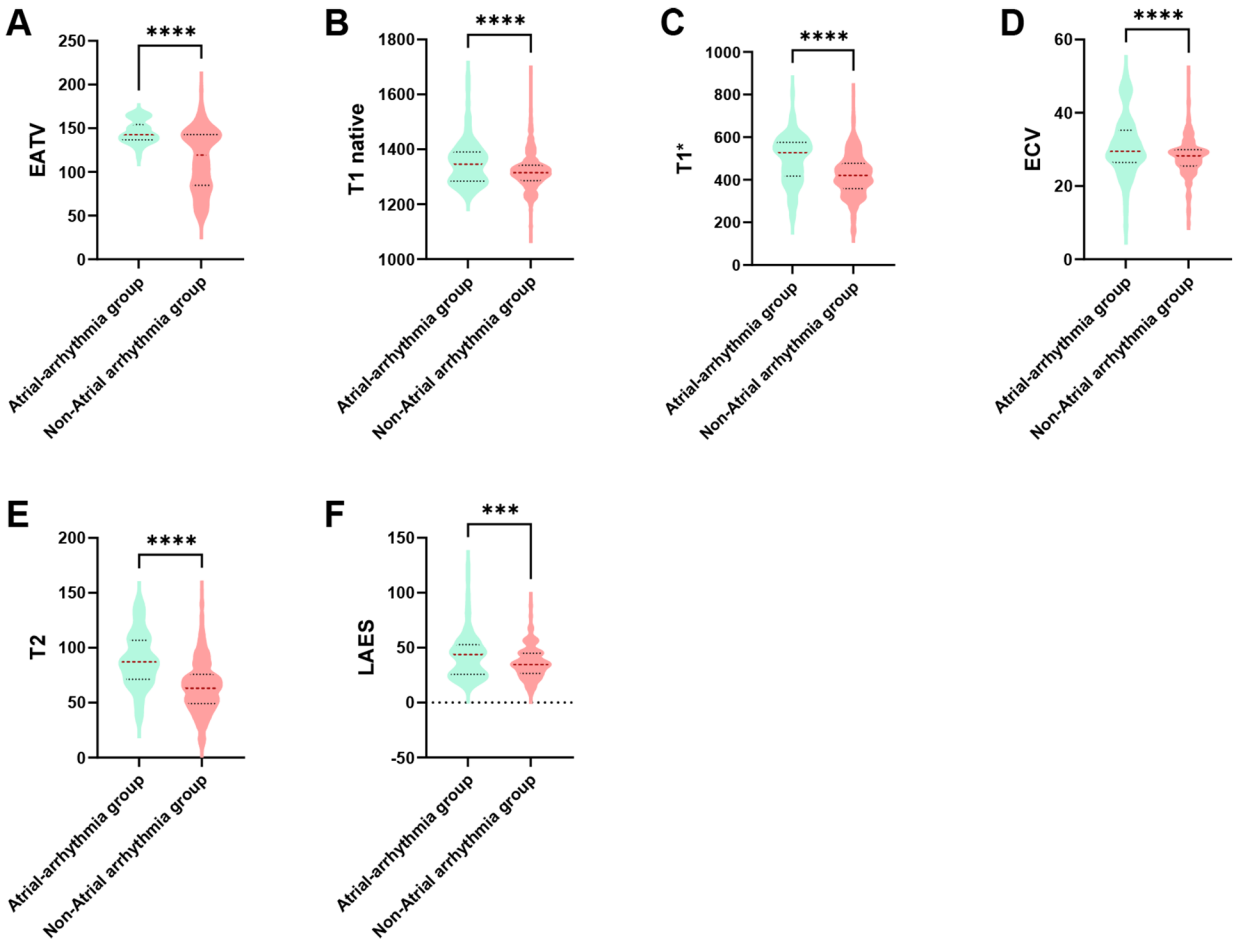

Fig. 3 Analysis of differences in CMR parameters between the two groups - violin plot

Table 2 Correlation of EATV with T1native, T1*,ECV, T2

	EATV	
Variables	***r***	***P* Value**
T1 native	0.476	0.001
ECV	0.529	0.001
T1*	0.467	0.001
T2	0.538	0.001

the study direction and methodology were different; and (3) this study was retrospective and they were prospective. It is possible that their study was longer, with early functional cardiac and later organic changes, leading to different ECV results before and after; our study had a short follow-up, with CMR performed only during hospitalisation, to obtain early myocardial changes in patients. When some scholars studied EAT in patients with cardiomyopathy, they found a significant positive correlation between EAT and T2 [19]. Now it seems that the pathological basis of T2 may be related to inflammation, and EAT is easy to break down and secrete pro-inflammatory cytokines, which is consistent with our study. Zhou, et al. found a negative correlation between T2* and EAT in dialysis patients by using T2* positioning. On the other hand, it can also be verified by us that the mechanism shows that EAT is related to myocardial inflammation and has negative micro-cardiovascular effects [20]. Mainly different from our study population, EAT secretes pro-inflammatory factors to damage blood vessels, which is a common risk factor for coronary heart disease and atrial fibrillation. The development of T2 as a new magnetic resonance module, The combination of the two may provide further targets for patients to intervene to prevent arrhythmias in the future.

T1 is used as a noninvasive surrogate for diffuse myocardial fibrosis in patients with atrial fibrillation [21].MR. et al. showed that T1 is associated with a history of atrial fibrillation [22]. Increased diffuse ventricular fibrosis leads to tachycardia episodes and shortened ventricular T1 in patients with a history of focal atrial tachycardia, comparing post-T1 (T1*) [23]. We got similar conclusions with them in the new-onset atrial arrhythmia population after STEMI: T1*, ECV is an independent risk

Table 3 Logistic regression analysis

	Univariable					Multivariable				
Variables	**B**	**SE**	**Waldx²**	***P***	**OR(95%CI)**	**B**	**SE**	**Waldx²**	***P***	**OR(95%CI)**
Age	0.021	0.010	4.186	0.041	1.022(1.001–1.043)	0.033	0.013	6.241	0.012	1.033(1.007–1.060)
Heart-rate	0.021	0.009	5.664	0.017	1.021(1.004–1.038)					
Peak hs-TnT	-1.888	0.172	120.151	0.001	0.151(0.001–0.248)					
PeakNT-proBNP	-1.773	0.145	149.887	0.001	0.172(0.001–0.386)					
LVEF	-0.132	0.025	28.979	0.001	0.876(0.835–0.919)	-0.147	0.031	22.629	0.001	0.863(0.812–0.917)
LAED	0.013	0.006	5.340	0.021	1.013(1.002–1.025)					
LAES	0.024	0.007	11.639	0.001	1.024(1.010–1.038)					
EATV	0.044	0.006	47.839	0.001	1.045(1.032–1.058)	0.037	0.008	22.394	0.001	1.038(1.022–1.054)
T1 native	0.006	0.002	15.309	0.001	1.006(1.003–1.010)					
T1*	0.007	0.001	35.539	0.001	1.008(1.005–1.010)	0.004	0.001	7.073	0.008	1.004(1.001–1.007)
ECV	0.091	0.021	19.167	0.001	1.095(1.051–1.141)	0.058	0.024	5.838	0.016	1.059(1.011–1.110)
T2	0.040	0.005	57.086	0.001	1.041(1.030–1.052)	0.030	0.006	22.321	0.001	1.030(1.018–1.043)

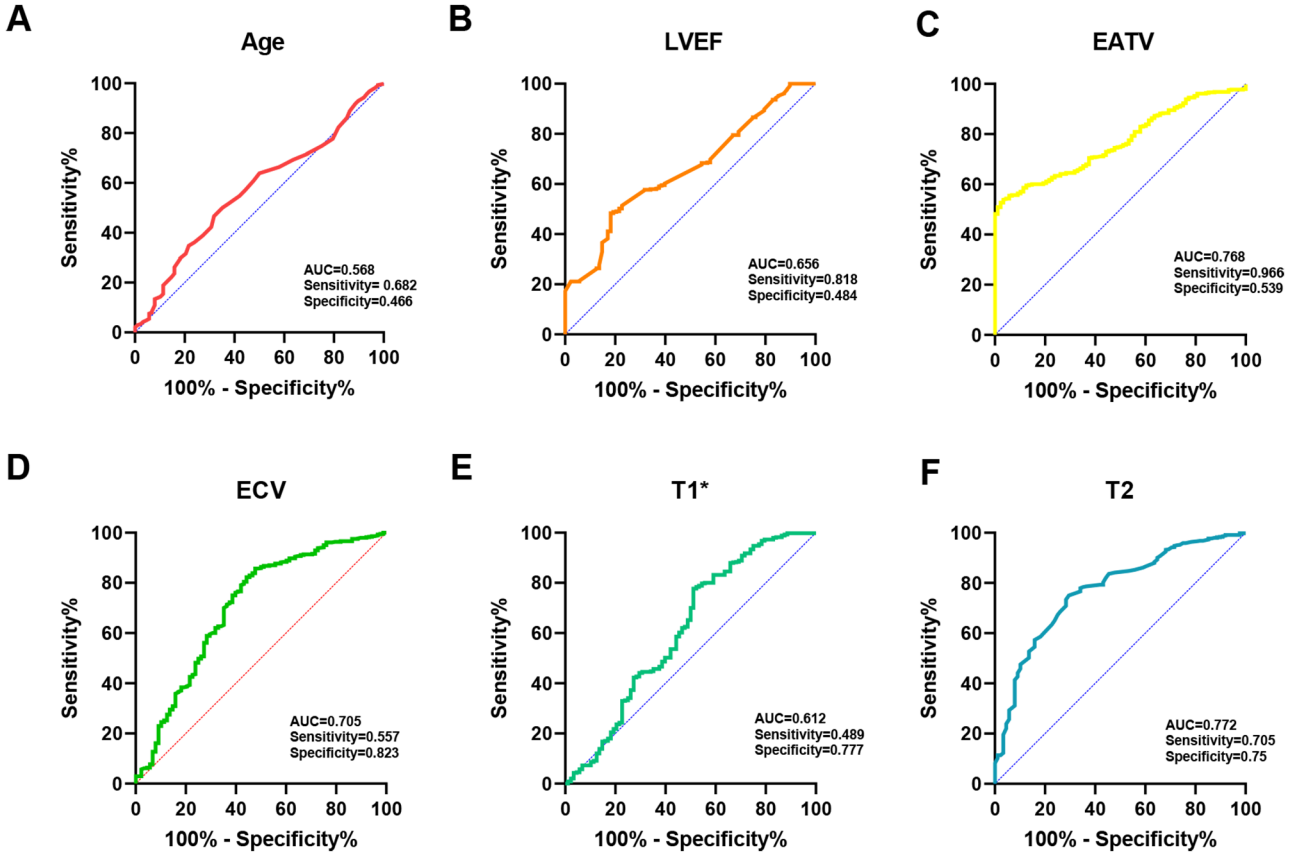

Fig. 4 ROC curve analysis

factor and has a good predictive value in the new-onset atrial arrhythmia population. Nowadays, T2 localisation is starting to be used in patients with STEMI, and T2 is an independent risk factor for STEMI with a good predictive value [24], and in our study of patients with new-onset atrial arrhythmias, the presence of T2 increased their risk, and it has been claimed that many of the underlying conditions that cause arrhythmias, including atrial fibrillation, often occur in acute myocardial ischemia - - may have an inflammatory component [25]. and we speculate that the pathological basis of T2 seems to be related to inflammation, which is why we may get that T2 is an independent risk factor with good predictive value in patients with new-onset atrial arrhythmias.

In addition to this, we obtained that age is also an independent risk factor for new onset atrial arrhythmia and has predictive value, which is in line with most of the studies [26, 27], atrial fibrillation is common in heart

failure patients with preserved left ventricular ejection fraction, which has a poor prognosis. In our study, reduction in LVEF increased the risk of new atrial arrhythmia patients after STEMI and had a good predictive value for their population, which is in line with studies [28].

This study has the following limitations: First, it is a retrospective study; The sample size is small; All patients were from a single center. Future multicenter prospective studies are needed to confirm the mechanism by which EAT affects myocardial conditions and arrhythmia development, and we should pay attention to the population with new atrial arrhythmias after STEMI. Second, there may be potential confounding factors (such as drug use or the occurrence of comorbidities) that may affect the results during the study. Although we have vigorously excluded the selection of the samples we need, the interference of potential confounding factors cannot be ruled out. Third, the arrhythmias selected in this study are not single and specific enough. Atrial arrhythmias are diverse. Due to the small sample size, during the follow-up, we found that certain single arrhythmias occurred less frequently (such as atrial fibrillation), and only a unified study on new atrial arrhythmias was done. Fourthly, because this study is retrospective, we did not evaluate the EAT of patients before their occurrence, and did not monitor whether STEMI patients had previous arrhythmias, and CMR could be obtained at multiple time points. We studied early CMR and did not perform CMR review during follow-up for some reasons, which is our defect.

Conclusion

In patients with STEMI, epicardial edipose tissue is associated with myocardial inflammation, fibrosis. Age, LVEF, EATV, T1*,ECV, T2 are independent risk factors for new onset atrial arrhythmias and have good predictive value.

Acknowledgements
Not applicable.

Author contributions
YR performed the experiments and analyzed the data. YW, JD, ML and JL were involved in data collection. YR wrote the manuscript. LC directed the entire research work and corrected the articles. All authors read and approve the manuscript and agree to be accountable for all aspects of the research in ensuring that the accuracy or integrity of any part of the work is appropriately investigated and resolved.

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Data availability
The datasets generated during and/or analyzed during the current study are available by request from the correspondence (drluyuan329@163.com).

Declarations

Ethics approval and consent to participate
The requirement for signed written consent was waived owing to no risk to the patient in accordance with the relevant IRB regulatory guidelines. This study was approved by the Ethics Committee of Xuzhou Medical University Affiliated Hospital, and complied with the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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