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Impact of Epicardial Adipose Tissue on Heart Failure with Reduced Ejection Fraction: A Coronary computed tomography Angiography-Based Imaging Analysis

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Impact of Epicardial Adipose Tissue on Heart Failure with Reduced Ejection Fraction: A Coronary computed tomography Angiography-Based Imaging Analysis

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ABSTRACT:

Objective: This study aimed to explore the association between epicardial adipose tissue (EAT) volume quantified by coronary computed tomography angiography (CCTA) and prognosis in patients with heart failure with reduced ejection fraction (HFrEF).

Patients and methods: In this retrospective study, 119 HFrEF patients undergoing CCTA between January 2022 and December 2023 were enrolled. EAT volume were semi-automatically quantified using CCTA sequences. The primary endpoint was the composite outcome of all-cause mortality and heart failure rehospitalization during follow-up. Cox regression and Kaplan–Meier analyses assessed the association between EAT volume and events. Conventional and time-dependent receiver operating characteristic (ROC) analyses were used to evaluate predictive performance, and the corresponding areas under the curve (AUC) were calculated.

Results: Over a median 649-day follow-up, 50 patients (42.0%) experienced endpoint events. Compared with patients without endpoint events, those with endpoints had lower EAT volume (56.55 ± 21.33 cm^3 vs. 74.64 ± 25.19 cm^3, $P < 0.001$) and higher EAT density (-72.94 ± 6.80 Hounsfield Units vs. -76.61 ± 5.07 Hounsfield Units, $P = 0.002$). Multivariate regression analysis showed that EAT volume was an independent prognostic predictor in HFrEF patients; lower EAT volume was associated with poorer prognosis, with a determined cut-off value of 65.53 cm^3. Subgroup analysis revealed that patients with lower EAT volume combined with lower body mass index had the poorest prognosis compared with other subgroups.

Conclusion: In patients with HFrEF, EAT volume is an independent prognostic predictor with moderate predictive value, and its cut-off value may provide additional information for clinical management.

Keywords: epicardial adipose tissue, heart failure with reduced ejection fraction, coronary computed tomography angiography

INTRODUCTION

Heart failure (HF) is a global health challenge. With population ageing and only modest improvement in survival after diagnosis with contemporary therapy, the number of people living with HF is projected to increase by 46.0% by 2030. [1] This increase is expected across all age groups. [2] Despite advances in therapeutic intervention, 5-year survival after HF diagnosis remains approximately 50.0%, comparable to that of many cancers. [3,4] This poor outlook is consistent with the progressive course of HF, in which periods of relative stability alternate with recurrent decompensation despite ongoing treatment. [5] On the basis of left ventricular ejection fraction (LVEF), HF is classified as heart failure with reduced ejection fraction (HFrEF) or heart failure with preserved ejection fraction (HFpEF). [6] Of them, HFrEF comprises nearly 50% of the HF population. [7] Within this population, patients hospitalized for acute decompensation have a substantially increased risk of short- and long-term death and rehospitalization and represent a critical point in clinical management. In these patients, prompt initiation and standardized implementation of HF therapy during the index admission are essential to improve outcomes; risk stratification at admission is also required to identify high-risk individuals for intensified monitoring and individualized care. However, methods currently available to predict prognosis in patients with HFrEF remain insufficient for routine clinical use.

Epicardial adipose tissue (EAT) is located between the myocardium and the visceral pericardium and is distributed mainly in the atrioventricular and interventricular grooves. [8,9] Unlike other visceral adipose depots, EAT is not separated from the myocardium by a fascia and shares a common microcirculation. [10] Under physiological conditions, EAT takes up and metabolizes free fatty acids, serving as a local myocardial energy source and buffering excess fatty acids; it also exhibits brown-fat–like thermogenic properties and secretes adipokines that regulate cardiac function via paracrine signalling. [11,12] Under pathological conditions, the same depot may shift toward a proinflammatory and profibrotic phenotype and impair autonomic function, thereby compromising systolic and diastolic performance and accelerating HF progression. [11,13] EAT can be assessed by echocardiography, computed tomography (CT), or cardiac magnetic resonance (CMR). Echocardiography is widely available but is largely limited to two-dimensional thickness measurement and is highly operator-dependent. CMR allows volumetric quantification without ionizing radiation but is time-consuming and costly. [14] CT provides high spatial resolution and clear delineation of the pericardial contour, enabling three-dimensional measurement of

epicardial adipose tissue volume (EATv) using attenuation thresholds and facilitating semi-automated post-processing. In the present study, EATv was quantified on coronary computed tomography angiography (CCTA) using a semi-automated method. Although EAT is associated with prognosis in HF, evidence on volumetric thresholds and on the prognostic value of EATv in HFrEF remains limited, particularly when assessment is performed during hospitalization.

Thus, this study aimed to investigate the association between CCTA-quantified EATv and the composite of heart failure rehospitalization and all-cause mortality in hospitalized patients with HFrEF, to determine whether EATv provides incremental prognostic information beyond conventional clinical factors, and to explore a volume threshold for risk stratification.

METHODS

Study population

Patients aged≥18 years with HFrEF who underwent CCTA between January 2022 and December 2023 were screened, totaling 225 patients (**Figure 1**). In accordance with the 2026 ESC Guidelines, HFrEF was defined as LVEF <50% and symptoms and/or signs of HF. Patients in New York Heart Association functional (NYHA) class II–IV were eligible. [6] Of these, 95 patients were excluded due to: (1) presence of malignant tumors; (2) end-stage renal failure with estimated glomerular filtration rate (eGFR) < 30 ml/min/1.73m^2 or receiving hemodialysis; (3) acute coronary syndrome or undergone coronary artery bypass grafting or percutaneous coronary intervention within 3 months; (4) severe valvular heart disease requiring surgical intervention; (5) missing coronary CT images. Additionally, 11 patients were excluded due to loss to follow-up. Thus, a total of 119 patients were enrolled in the study.

Data collection

Data on patient demographics and vital variables were retrieved from the electronic medical record system. The collected variables included age, sex, medical history, clinical diagnoses, vital signs during hospitalization, laboratory results, and echocardiographic findings obtained during hospitalization.

Body mass index (BMI) was calculated as weight divided by height squared (kilograms per square meter, kg/m^2). Body surface area (BSA) was determined using Stevenson's formula: 0.0061 × height (cm) + 0.0128 × weight (kg) − 0.1529. The eGFR was computed using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. [15]

CCTA protocol

CCTA was performed using a Siemens third-generation dual-source CT scanner (SOMATOM definition force; Siemens AG, Munich, Germany). Patients rested for at least 15 minutes before scanning, and no medications, such as β-blockers, were used to modify heart rate. The scan covered the region from the superior sternal notch to the diaphragmatic surface of the heart. A prospective electrocardiogram-gated sequence was employed with the following settings: tube voltage, 100 kV; tube current, 228-300 mA; CT rotation time, 0.25 s; layer thickness, 0.75 mm; reconstruction interval, 0.50 mm; and display matrix, 512 × 512.

Quantification of CCTA-derived EATv

All CCTA images were analyzed on a Syngo Via workstation (VB40, Siemens Healthineers, Germany) by two experienced cardiac CT specialists who were blinded to the patients' clinical information. EATv was measured on short-axis views with a 0.75-mm slice thickness using semi-automated software (Cardiac Risk Assessment Prototype, Syngo. via Frontier, Siemens Healthineers). [16] The software automatically quantified EATv by calculating the volume of tissue with CT attenuation values between −190 and −30 Hounsfield units (HU). [15,17,18] EAT was defined as the adipose tissue between the parietal and visceral layers of the pericardium. The software automatically delineates the EAT region from the pulmonary trunk to the cardiac apex, designating it as the region of interest (ROI). [18] If the automatic delineation is inaccurate, the ROI is manually adjusted. Subsequently, the software reconstructs EAT as a three-dimensional volume and automatically calculates the EAT volume and EAT density by applying the predefined adipose tissue threshold. An intra-observer consistency test was carried out on the results measured by the same doctor, and an inter-observer consistency test was carried out on the results measured by 2 doctors. Features with ICC > 0.9 were considered to have good agreement and were retained for subsequent analysis (**Supplementary Table 1**).

Follow-up and endpoints

Electronic medical records were reviewed to assess outcomes and obtain medical history data. For cases with insufficient inpatient records, patients or their first-degree relatives were directly contacted via telephone. Each patient was followed up for at least 1 year after discharge, continuing until the end of February 2025, when the dataset was locked. All endpoint events occurring during the follow-up period were recorded. The primary endpoint was a composite of heart failure rehospitalization and all-

cause mortality. The secondary endpoint was heart failure rehospitalization, defined as unplanned hospitalization due to heart failure progression or directly related to heart failure.[19]

Statistical analysis

Statistical analyses were conducted using IBM SPSS® Statistics, version 29. Data were collected until February 2025. All endpoint events, including all-cause mortality and rehospitalization for HF, were recorded. Normally distributed continuous variables are reported as mean ± standard deviation, while skewed variables are presented as median with interquartile range (25th to 75th percentiles). Categorical variables are expressed as percentages. The Shapiro-Wilk test was used to evaluate variable normality. Continuous variables were compared using the t-test, and categorical variables were analyzed with the chi-square (χ^2) test.

Univariate Cox proportional hazards regression was performed, and variables with a *P*-value < 0.05 were included in the multivariable regression analysis. Pearson's correlation coefficient was used to assess correlations between EATv and normally distributed variables, while Spearman's rank correlation was applied for skewed variables. A Cox regression model was constructed to evaluate the risk of endpoints across EATv quartiles. Hazard ratios (HRs) and 95% confidence intervals (CIs) for normally distributed variables are reported per standard deviation (SD) increase or decrease.

To evaluate the discriminative performance of EATv, conventional receiver operating characteristic (ROC) curves were constructed using composite-endpoint status during follow-up as a binary outcome, and areas under the curve (AUC) were calculated. The analyses included a head-to-head comparison of EATv with B-type natriuretic peptide (BNP), and a combined score was derived from a logistic model including both markers. Time-dependent ROC analysis was further used to assess the temporal change in discriminative performance during follow-up and the incremental value of adding EATv to a multivariable clinical Cox model. The relationship between EATv and endpoint risk was examined on a continuous scale using a restricted cubic spline (RCS) curve. Kaplan-Meier survival curves were plotted, and group differences were evaluated using the log-rank test.

RESULTS

A total of 119 patients with HFrEF were enrolled in this cohort study. The median age of the cohort was 68 years (interquartile range, 58-75 years), with 36 patients (30.3%) being female. The mean BMI of the cohort was 24.69 kg/m^2. Following a median follow-up period of 649 days (interquartile range,

407-795) days, 50 patients (42.0%) experienced the primary endpoint, including all-cause mortality and rehospitalization for HF, while 69 (58.0%) did not. Baseline characteristics of the study population are presented in **Table 1** and **Supplement Table 2**. Significant differences were observed between the two groups (those who experienced the primary endpoint and those who did not) in terms of sex, atrial fibrillation, calcium channel blocker (CCB), statins, total protein, albumin, serum cholinesterase, triglyceride-Glucose Index (TyG), chylomicrons (CM), eGFR, BNP, platelet hematocrit (PCT) and antithrombin III activity (AT-III) (all $P < 0.05$). No significant differences were found in BMI, BSA, history of hypertension, diabetes mellitus, hyperlipidemia, number of smokers, number of alcohol drinkers, total cholesterol, troponin I and other echocardiographic parameters between the two groups between the groups ($P > 0.05$).

Then, we used CCTA sequences to semi-automatically quantify EATv and EATd. **Figure 2A-D** illustrated representative images used to quantify EATv, with slices taken every 30 mm from the coronal, sagittal, and axial planes. Notably, EATv differed significantly between the two groups ([74.64 ± 25.19] cm^3 vs. [56.55 ± 21.33] cm^3, $P < 0.001$) (**Figure 2E**), as did EATd ([-76.61 ± 5.07] HU vs. [-72.94 ± 6.80] HU, $P = 0.002$) (**Figure 2F**).

Subsequently, we sought to investigate the association between EATv and prognosis in patients with HFrEF. We stratified the patients into quartiles based on EATv and performed comparative analyses. Significant intergroup differences were observed in body weight, BMI, BSA, furosemide use, CCB use, EATv, EATd, composite endpoint, and heart failure rehospitalization (all $P < 0.05$) (**Supplement Table 3**). Additionally, left atrial diameter, albumin, serum cholinesterase, low-density lipoprotein, BNP, RBC and RDW-SD also showed significant differences among the groups (**Supplement Table 4**).

Based on these findings, we performed cox regression analysis on the data stratified by EATv levels, using the highest EATv group (Q4) as the reference. We constructed three adjusted regression models: Model 1 adjusted for age, sex and BMI; Model 2 included all variables from Model 1 plus comorbidities including hypertension and diabetes; Model 3 incorporated all variables from Model 2, LVEF and BNP. As shown in **Table 2**, lower EATv was associated with poorer prognosis ($HR_{unadjusted} = 3.587$, 95% CI: 1.422-9.049 for Q1 vs. Q4, $P_{trend} = 0.004$; $HR_{model3} = 3.558$, 95% CI: 1.174-10.781 for Q1 vs. Q4, $P_{trend} = 0.018$). After multivariate regression adjustment, each standard deviation increasing in EATv reduced the likelihood of endpoint events by 42.5%. The probability of endpoint events was 63.3% in Q1 and 20.7% in Q4, with significant differences across the four groups ($P < 0.001$), indicating that smaller

EATv may suggest a worse prognosis (**Figure 3A**).

We further evaluated the prognostic value of EATv using ROC analysis. In single-indicator analyses, the discriminative performance of EATv for the composite endpoint was comparable to that of the established cardiovascular biomarker BNP, with a numerically superior trend in AUC (0.700 vs. 0.662). The combination of EATv and BNP yielded further improved discriminative performance ($AUC_{combined}$ = 0.737). Over the follow-up period, the time-dependent AUC of EATv remained stable in the range of 0.6 - 0.7, indicating stable discriminatory performance for long-term prognosis. After incorporation of EATv into a multivariable Cox model adjusted for clinical covariates, the time-dependent AUC of the model demonstrated an overall increasing trend. These findings suggest that EATv may serve as a complementary imaging marker for prognostic assessment in patients with HFrEF. These results are shown in **Figure 3B-E and Supplement Table 5-6**.

The RCS results showed a non-linear relationship between EATv and the prognosis of HFrEF patients, as well as for EATd. When EATv was increasingly less than the cut-off value of 65.53 cm^3, the prognosis worsened (**Figure 4**). Furthermore, EATv demonstrated a linear positive correlation with body weight, BMI, BSA and LVEF, and a linear negative correlation with BNP and EATd. Specifically, there was a significant but weak association between BMI and EATv (Spearman's r = 0.380, $P < 0.001$), and a moderate correlation between EATv and EATd (Spearman's r = -0.601, $P < 0.001$) (**Figure 5A-F**). However, the consistency between EATv and BMI in predicting the prognosis of patients with HFrEF remains unclear.

Then, patients were divided into four groups based on the range of BMI and EATv above or below 65.53 cm^3 (**Figure 5G**) [20]. Specifically, 34 (28.6%) patients were classified into the low EATv and low BMI group, and 25 (21.0%) patients into the low EATv but high BMI group. The low EATv and low BMI group had a significantly higher event rate compared to the other groups ($P = 0.013$) (**Supplement Table 7**). Patients with low BMI were older than high ones. As shown in **Figure 5H**, the trend of EATv changes is consistent with that of BMI, with patients having lower BMI exhibiting smaller EATv.

Using the cut-off value of 65.53 cm^3, the cohort was divided into high and low EATv groups. Kaplan-Meier survival analysis revealed significant differences between the two groups, both for the primary composite endpoint (Log Rank $\chi^2 = 9.083$, $P = 0.003$) and for the secondary endpoint of heart failure rehospitalization (Log Rank $\chi^2 = 6.132$, $P = 0.013$). The low EATv group had a significantly higher event rate than the high EATv group (**Figure 6A, 6B**). When performing survival curve analysis

combining EATv and BMI, we found that the low EATv and low BMI group had the highest endpoint event rate than the other groups (composite endpoint, Log Rank $\chi^2 = 7.303$, $P = 0.007$; heart failure rehospitalization, Log Rank $\chi^2 = 6.311$, $P = 0.012$) (**Figure 6C, 6D**). The low EATv and low BMI group had the highest composite endpoint event rate, with significant differences between groups ($P < 0.05$), indicating that this group may be associated with a poorer prognosis (**Figure 6E**).

DISCUSSION

In summary, our findings are as follows: (1) EATv is an independent risk factor for the composite endpoint of all-cause mortality and heart failure rehospitalization in patients with HFrEF; (2) The group with EATv < 65.53 cm^3 is associated with worse prognosis; (3) The subgroup with EATv < 65.53 cm^3 and BMI < 24 kg/m^2 exhibits the poorest prognosis compared to other groups.

Previous studies have demonstrated that EAT density is highly sensitive to contrast enhancement, whereas EATv shows excellent consistency between contrast-enhanced and non-enhanced CT acquisitions. [21-23] Therefore, EATv was selected as the primary quantitative parameter in the present study. We investigated the relationship between EATv and prognosis in HFrEF patients, stratifying them based on the occurrence of endpoint events. Minimal baseline differences between groups indicate strong comparability. Significant differences were observed in EATv and EATd between groups ($P < 0.001$). We also found that EATv was negatively correlated with brain natriuretic peptide (BNP), consistent with Xu' s study, [24] and positively correlated with BMI, BSA, and body weight, consistent with Rabkin' s study. [25] Some studies have reported a negative correlation between EATv and eGFR, [26] however, no such difference was observed in our study, possibly due to our exclusion of patients with severe renal insufficiency. After stratifying patients into quartiles based on EATv, we found that the low-EATv group was older, with lower body weight, BMI, serum albumin, and higher BNP levels. Previous studies have demonstrated that both EAT thickness and volume are associated with the prognosis of patients with HF; however, these associations exhibit opposing prognostic relationships across different HF subtypes. Accumulation of EAT is linked to adverse outcomes in patients with HFpEF, [27,28] whereas in patients with HFrEF, the converse is true, with smaller EATv correlating with poorer prognosis. [24,29,30]

One potential explanation may account for the association between low EATv and poorer prognosis observed in this study. Sacks et al. investigated EAT collected during open-heart surgery and found that uncoupling protein 1 (UCP-1) expression in epicardial fat was fivefold higher than in substernal fat, [31]

suggesting that EAT can function similarly to brown adipose tissue (BAT) in thermogenesis. Furthermore, brown adipose tissue-like EAT actively participates in maintaining energy homeostasis by supplying energy substrates such as free fatty acids, with previous studies indicating that free fatty acid oxidation accounts for 70% of cardiac energy production. [32] With aging, the brown fat characteristics of EAT decline under physiological conditions. [11] Sacks et al. also observed that UCP-1 expression in EAT decreases with increasing age. [31] Our cohort primarily comprised elderly patients, particularly those experiencing endpoint events (66 vs. 71.5 years, $P = 0.018$). Under pathological conditions, especially in advanced disease or among the elderly, the thermogenic function of EAT further declines. [11] Additionally, Peterson et al. examined the genetic profiles and signaling pathways of EAT from patients with advanced heart failure and obese mice, revealing that upregulation of BAT and mitochondrial signaling genes was associated with reduced inflammatory adipokines in epicardial fat and improved left ventricular function in obese mice. [33] Brunetta et al. induced browning of adipose tissue in obese mice using β3-adrenergic agonists, which upregulated genes associated with BAT formation and demonstrated that this process alleviates local inflammation and ameliorates left ventricular hypertrophy. [34] These findings collectively suggest that the expression of thermogenic genes in EAT serves as a protective factor in HFrEF, whereas downregulation of thermogenic gene mRNA expression in HFrEF patients indicates that reduced functional EAT is detrimental to the heart. [35] In conjunction with our conclusions, we speculate that the abnormal reduction in EAT volume among patients with HFrEF may indicate impaired metabolic and paracrine functions of functional EAT. Jin et al. reported significantly reduced expression of brown adipose tissue-related genes in EAT from HFrEF patients, supporting this hypothesis.[36]

Moreover, ROC analyses showed that the discriminative performance of EATv for the composite endpoint in patients with HFrEF was comparable to that of BNP. Combined assessment of both markers was associated with a trend toward improved discrimination, and the prognostic performance of EATv remained stable across the follow-up period. After incorporation of EATv into a multivariable clinical model, the time-dependent AUC showed an overall upward shift, but this increment did not reach consistent statistical significance, likely because the baseline model already included several established strong prognostic factors. Overall, EATv may serve as a complementary imaging marker to refine risk stratification, and these findings still require validation in larger cohorts. Using RCS analysis, we determined an EATv cut-off value of 65.53 cm^3. To our knowledge, few studies have reported EATv cut-

off values in HFrEF patients. One study identified an optimal cut-off of 22 g/m² for predicting cardiac death. [37] Our study provides additional data on EATv cut-off values to predict the risk of cardiac death and heart failure hospitalization in HFrEF patients, and this conclusion will be further validated in a larger cohort.

Then, we conducted subgroup analyses based on BMI. Although BMI is widely recognized as an imperfect indicator of health risks and mortality, because it does not account for body composition, nutritional status, cardiorespiratory fitness, or fat distribution, particularly visceral fat, [38] it remains the most readily available screening measure at the population level. Many obesity guidelines continue to recommend BMI as a practical tool for predicting obesity-related disease outcomes. [39,40] Previous studies have shown that BMI effectively predicts myocardial infarction risk 20 years later. [25] Both BMI and EAT are associated with impaired cardiac structure and function, as well as increased myocardial fibrosis. [38] Autopsy studies have reported a significant but weak correlation between BMI and EAT, [25] consistent with our findings, likely because BMI reflects only height and weight and does not accurately represent total or visceral fat distribution. Some studies have reported a U-shaped relationship between BMI and mortality; however, we did not observe this, possibly due to our smaller cohort size and limited inclusion of extremely obese patients. Huang et al. reported that lower BMI and reduced EAT were associated with higher cardiovascular event rates. [38] Our study also observed this finding. This may relate with cardiac cachexia. Cardiac cachexia lacks a definitive consensus, but some studies define it as unintentional weight loss of ≥ 7.5% over the past 6 months. [41] EAT size correlates with BMI, and studies have shown that cachectic individuals have less EAT on average than healthy controls. [42] Cachexia is often accompanied by preferential fat tissue loss and reduced adipocyte volume. [43] HF is a chronic wasting disease, with more pronounced wasting symptoms in advanced stages, potentially leading to generalized fat loss. Fat tissue depletion in cachexia is primarily driven by net loss of stored triglycerides, affecting fatty acid metabolism and cardiac energy supply. Disruption of cardiac metabolic homeostasis plays a critical role in cardiovascular disease progression. Imbalance between catabolism and anabolism can lead to a shift toward cachexia. [37] Cachexia is characterized by increased lipolysis and a shift to catabolic metabolism. [24] Jin et al. found that thicker EAT was associated with better LVEF. [36] Similarly, Nicola et al.'s subgroup analysis in HFrEF patients showed more severe left ventricular systolic dysfunction and remodeling in the low-EAT group compared to the high-EAT group, potentially indicating cardiac cachexia. [24] We hypothesize that reduced EAT in HFrEF patients, whether in thickness or volume, may

be a marker of pathological catabolic states and a signal of cardiac cachexia.

To our knowledge, this is the first study in China to investigate the association between EATv quantified by CCTA and prognosis in patients with HFrEF. We are the first to report that, in a Chinese population, an EATv < 65.53 cm^3 is an independent risk factor for the composite endpoint of all-cause mortality and heart failure rehospitalization. Our study not only establishes the association between EATv and adverse HF outcomes but also examines its association with commonly used clinical indicators and risk factors. Furthermore, as an imaging-derived marker, EATv may provide incremental prognostic information that is complementary to established clinical and biochemical risk factors. The combination of low EATv and low BMI identifies an extremely high-risk subgroup with the poorest prognosis, which may indicate the presence of cardiac cachexia.

There are several limitations in the current study. First, this study is a single-center, retrospective cross-sectional investigation, which precludes the determination of a causal relationship between EATv and prognosis of patients with HFrEF. The causal relationship between reduced EAT and left ventricular dysfunction in HF remains unclear. Future studies are needed to validate in larger, more diverse heart failure cohorts. Second, there is currently no standardized method for quantifying EATv, and discrepancies may exist between quantification software and actual measurements. However, we minimized errors by having two experienced radiologists manually adjust the region of interest. Finally, the EATv cut-off value may vary across different heart failure populations, countries, or ethnic groups, necessitating external validation in other cohorts.

CONCLUSION

In patients with HFrEF, EAT volume is an independent prognostic predictor with moderate predictive value, and its cut-off value may provide additional information for clinical management.

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DECLARATIONS

Author contributions W.J.Y. designed the study methodology, curated the data, and drafted the original manuscript. D.L. performed formal statistical analysis and validated the research results. H.J.Y. collected clinical data and conducted subject investigation. D.Y. carried out data analysis and created the figure visualizations. C.Q.W. participated in data curation and subject investigation. G.Q.Q. validated the findings and revised the manuscript content. P.L. developed the study methodology and conducted formal statistical analysis. C.L. and W.Z. oversaw study implementation and took charge of overall project administration. S.L. secured funding support for the research. W.Q.J. critically reviewed the manuscript and provided funding resources. W.J. conceived the study, provided overall supervision, and acquired funding. All authors reviewed and approved the final manuscript.

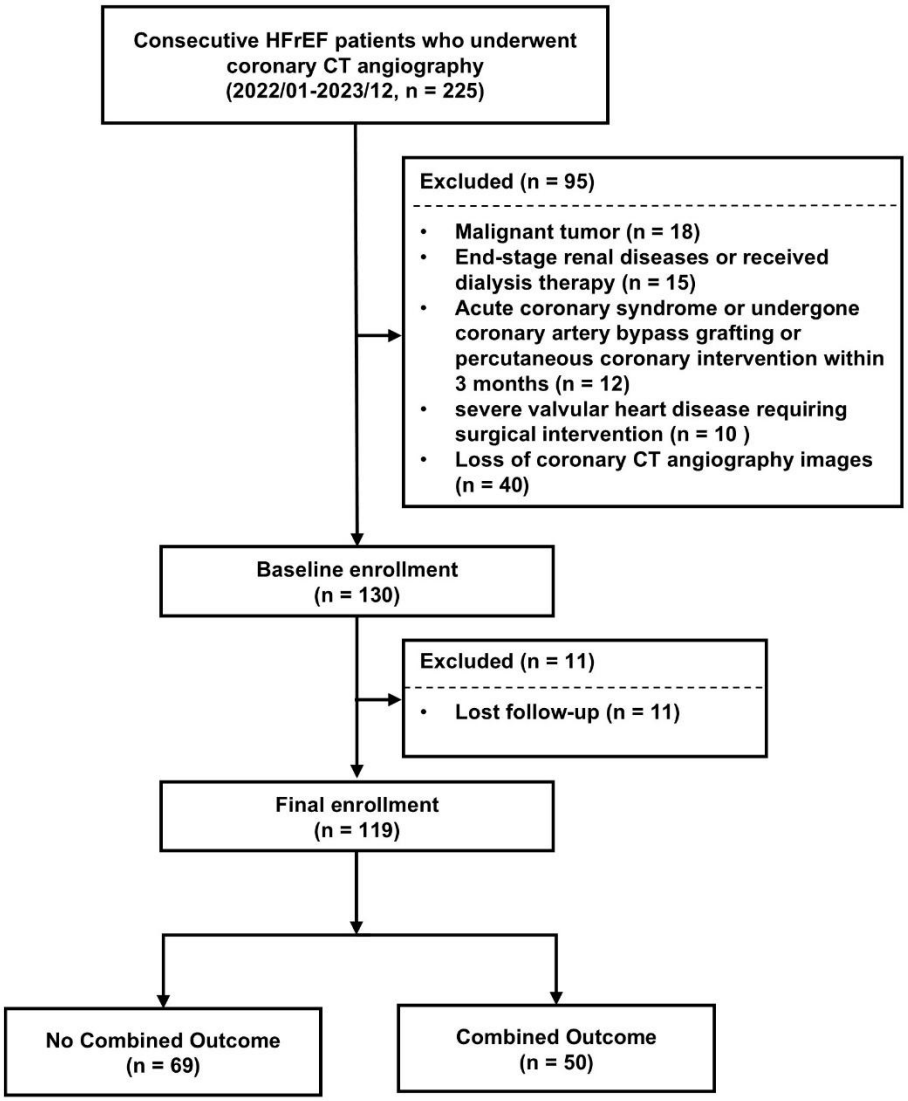
Ethics approval and consent to participate All individuals enrolled in the study provided written informed consent prior to participation. The research adhered to the ethical principles of the Declaration of Helsinki and was approved by the Ethics Committee of the Third Affiliated Hospital of Nanjing Medical University (No.2024-KY313-01).

Clinical trial number Not applicable.

Consent for publication Not applicable.

Conflicts of Interest The authors declare no competing interests.

Figure



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373 **Figure 1. Study flow chart.** HFrEF, heart failure with reduced ejection fraction; CT, computed

374 tomography; eGFR, estimated glomerular filtration rate.

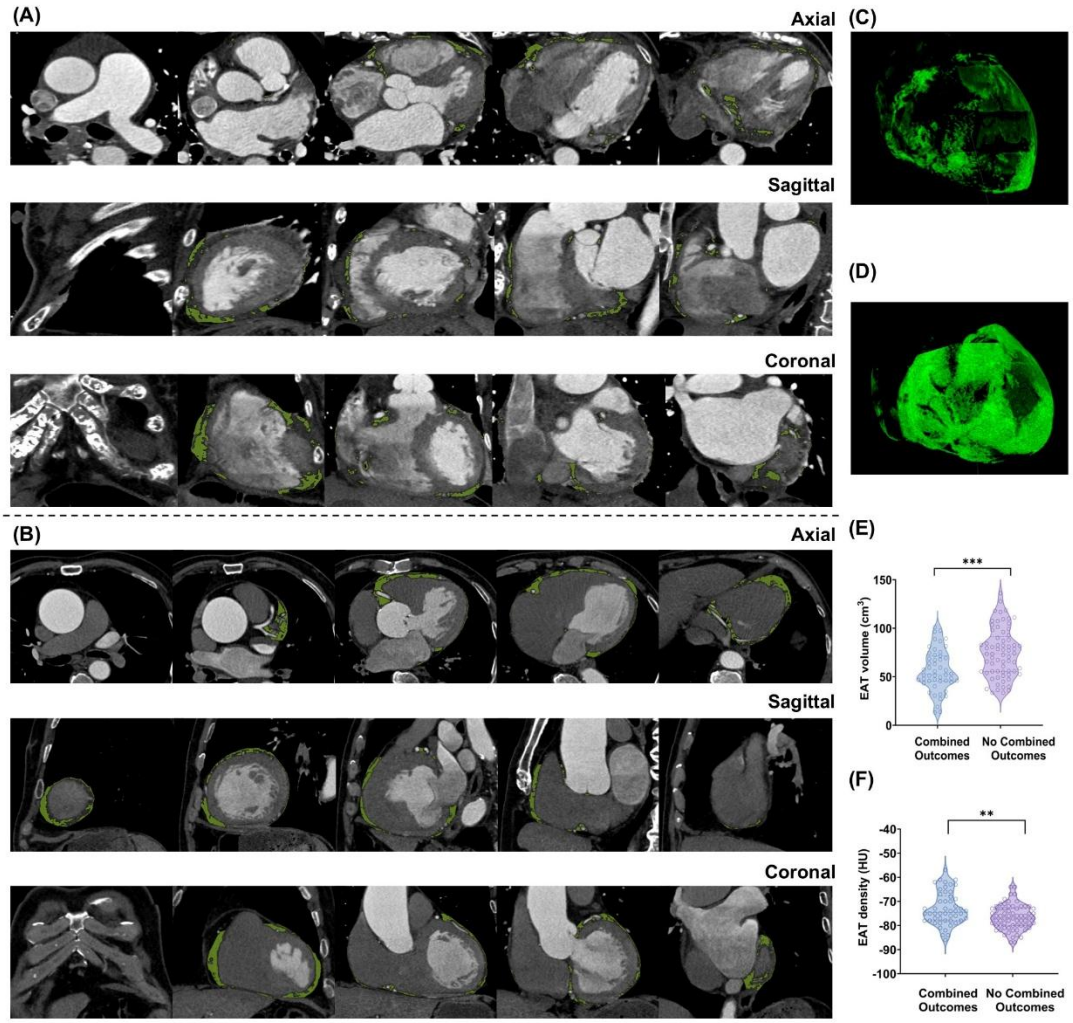
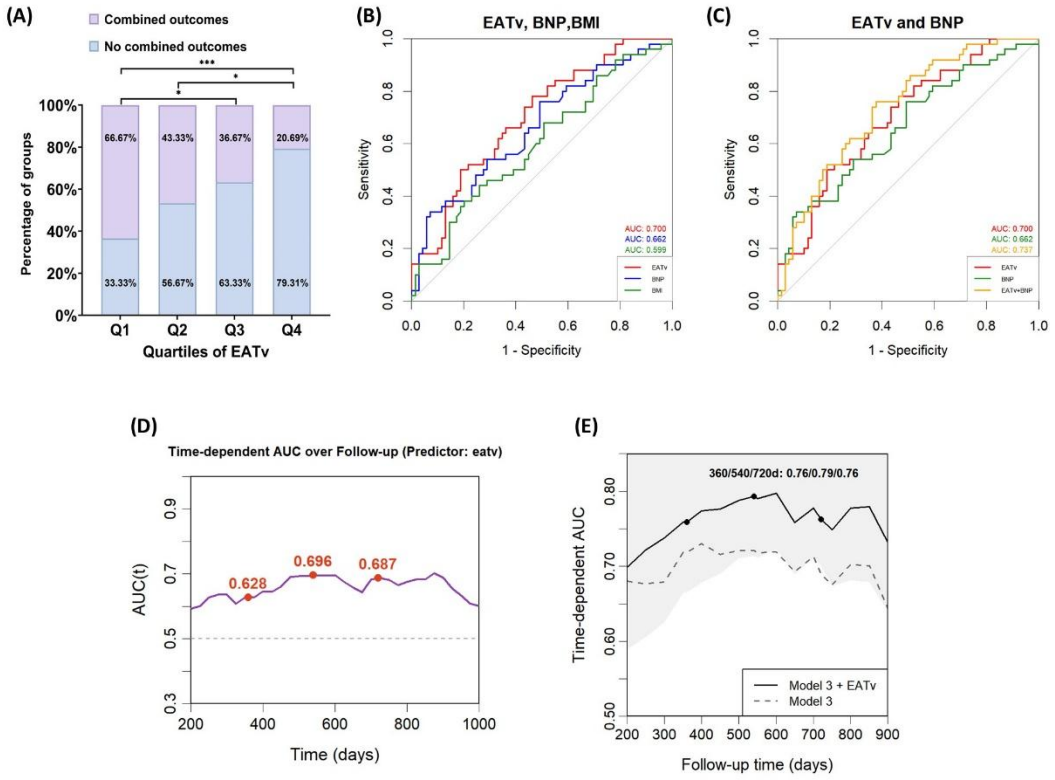


Figure 2. EAT on the series of CCTA in HFrEF patients. Shown are representative axial, sagittal and coronal images of EAT in patients with endpoints and without endpoints. 3D visualization of EAT was performed by reconstruction. (A) EAT in the patient with combined outcomes; (B) EAT in the patient without combined outcomes. (C) 3D reconstruction of EAT in the patient with combined outcomes. (D) 3D reconstruction of EAT in the patient with no combined outcomes. (E-F) Patients with combined outcomes had lower EATv but higher EATd compared with patients without combined outcomes. $**P<0.01$; $***P<0.001$. CCTA, coronary CT angiography; EAT, epicardial adipose tissue; HFrEF, heart failure with reduced ejection fraction; EATv, epicardial adipose tissue volume; EATd, epicardial adipose tissue density; HU, Hounsfield units.



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388 **Figure 3. Distribution of patients in different EATv groups according to quartiles and receiver**
389 **operating characteristic curve showing the identification of patients at increased risk of all-cause**
390 **mortality by EAT volume.** The event rate is shown numerically. ∗∗∗P<0.001. ∗P<0.05. AUC indicates
391 area under the curve. (A) Patients' distribution in different groups according to EATv quartiles. (B)
392 Conventional ROC curves for individual variables. (C) Conventional ROC curves for the model
393 constructed using a combination of both variables. (D) Time-dependent AUC of EATv during follow-up.
394 (E) Time-dependent AUC of Cox regression model 3 and model 3 plus EATv. Dots indicate prespecified
395 time points (360, 540 and 720 days). EATv, epicardial adipose tissue volume; BNP, B-type natriuretic
396 peptide; BMI, body mass index; AUC, Area under the curve.

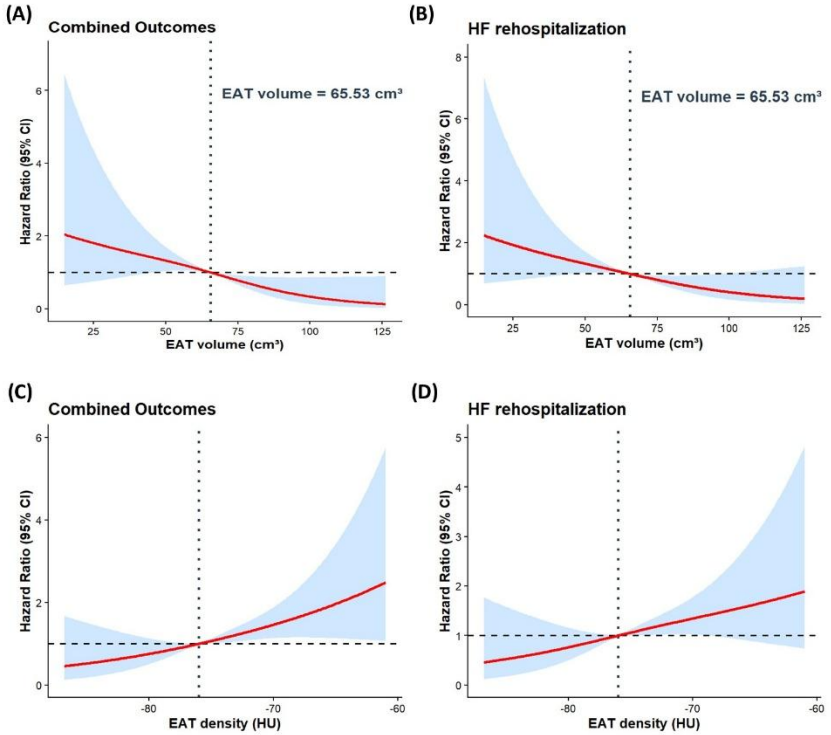
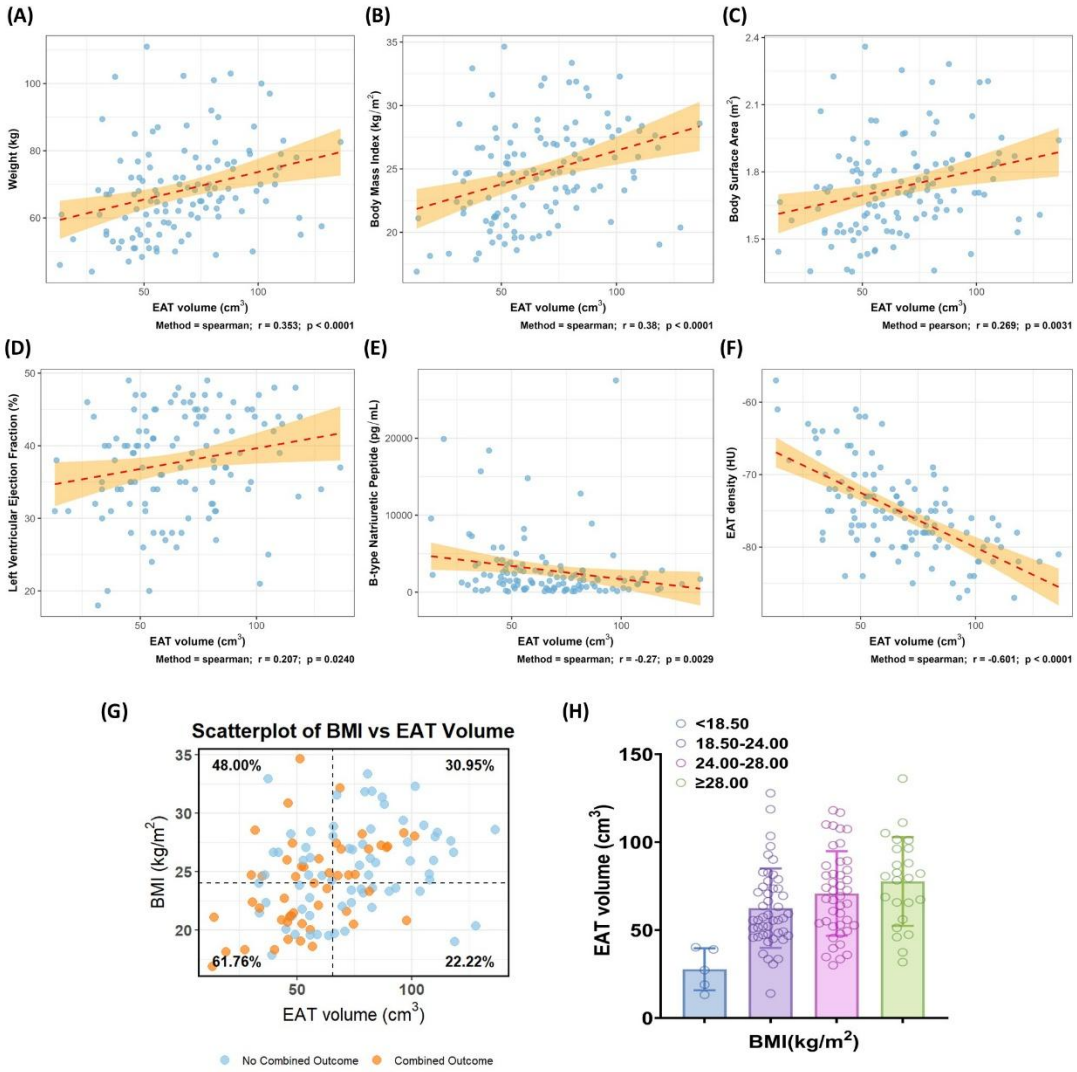


Figure 4. The relation of EAT volume and EAT density with the risk of endpoints in a restricted cubic splines model. The relation of EAT volume and EAT density with the risk of combined outcomes (A,C) and just HF rehospitalization (B,D) on a continuous scale. The hazard ratio is indicated by the solid red line, and the 95% CI is represented by the shaded regions derived from the threenode restricted cubic spline regression. The horizontal reference line represented by a dashed line indicates a odds ratio of 1.0, and the vertical reference line is the corresponding EAT volume at this time. EAT, epicardial adipose tissue; CI, confidence intervals; HF, heart failure.



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408 **Figure 5. Correlation between EAT volume and other biomarkers and distribution and endpoint**
409 **event rates by BMI and EAT volume categories.** The dashed line indicates the predictive EAT volume
410 by weight (A) or body mass index (B) or body surface area (C) or left ventricular ejection fraction (D)
411 or B-type natriuretic peptide (E) or EAT density (F) using linear regression. 95% CI is represented by the
412 shaded regions derived from linear regression. (G) Scatterplot between BMI and EAT volume, rendering
413 4 groups based on BMI = 24kg/m^2 and EAT volume cut-off value. Percentages are relative event rates
414 for the composite of all-cause mortality and heart failure rehospitalizations per group. (H) The
415 distribution of EAT volume in the entire cohort classified by BMI levels. EAT, epicardial adipose tissue;
416 CI, confidence intervals; BMI, body mass index.

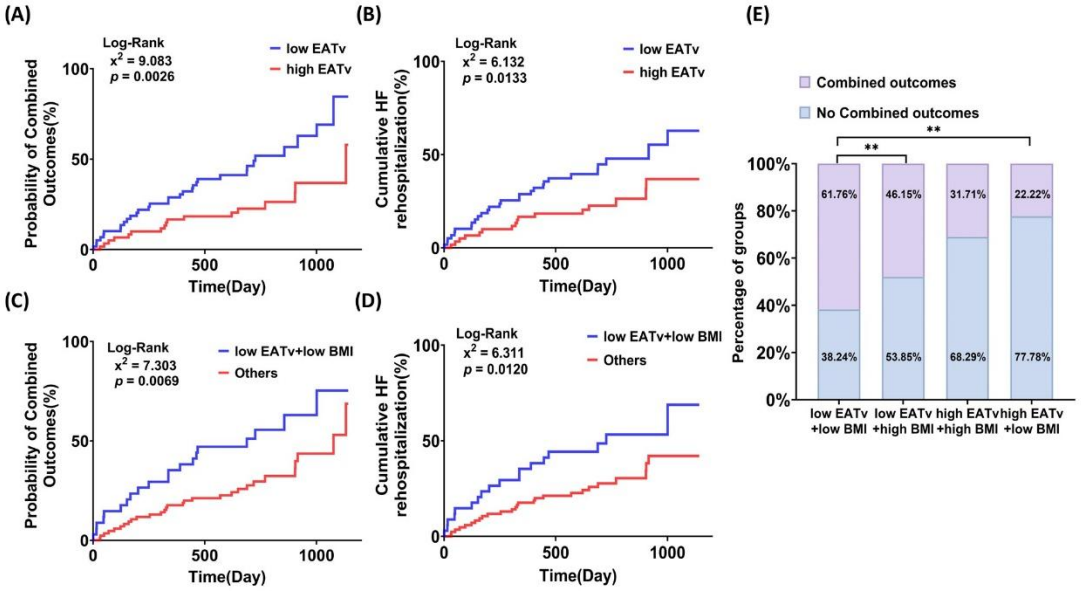


Figure 6. Kaplan-Meier curves and endpoint event rates based on EATv cut-off value and BMI levels. (A) Kaplan-Meier curves for the combined outcome stratified for EATv cut-off value. (B) Kaplan-Meier curves for heart failure hospitalization stratified for EATv cut-off value. (C) Kaplan-Meier curves for the combined outcome stratified by EATv cut-off value and BMI. (D) Kaplan-Meier curves for heart failure hospitalization stratified by EATv cut-off value and BMI. (E) Stacked bar graphs showing the risk of combined outcomes by EATv cut-off value and BMI. $*P<0.05$. EATv, epicardial adipose tissue volume; BMI, body mass index.

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Table 1 Baseline characteristics in the entire cohort

Variables	All (N=119)	No Combined Group (N=69)	Combined Group (N=50)	*P*-value
Demographic characteristics				
Age, year	68.00 [58.00, 75.00]	66.00 [54.00, 74.00]	71.50 [60.75, 77.25]	0.018
Female, n (%)	36 (30.3%)	20 (29.0%)	16 (32.0%)	0.724

Weight, kg	67.00 [58.50, 76.00]	68.00 [60.20, 77.55]	65.81 ± 13.12	0.157
BMI, kg/m^2	24.69 ± 3.87	25.26 ± 3.79	23.91 ± 3.86	0.060
BSA, m^2	1.71 [1.59, 1.84]	1.72 [1.60, 1.87]	1.70 ± 0.20	0.258
Comorbidities				
Hypertension, n (%)	88 (74.0%)	52 (75.4%)	36 (72.0%)	0.680
Diabetes mellitus, n (%)	32 (26.9%)	17 (24.6%)	15 (30.0%)	0.515
Hyperlipidemia, n (%)	64 (53.8%)	39 (56.5%)	25 (50.0%)	0.481
Smoking, n (%)	38 (31.9%)	24 (34.8%)	14 (28.0%)	0.433
Drinking, n (%)	25 (21.0%)	16 (23.2%)	9 (18.0%)	0.493
Atrial Fibrillation, n (%)	40 (33.6%)	18 (26.1%)	22 (44.0%)	0.041
NYHA, n (%)				0.255
Class II	67 (56.3%)	43 (62.3%)	24(48.0%)	
Class III	26 (21.8%)	14 (20.2%)	12(24.0%)	
Class IV	26 (21.8%)	12 (17.3%)	14(28.0%)	
Biochemical examinations				
TC, mmol/L	3.99 ± 1.02	4.1 ± 1.12	3.85 ± 0.85	0.168
CM, mmol/L	1.18 [0.85, 1.61]	1.36 [0.90, 1.83]	1.03 [0.81, 1.43]	0.024
HDL, mmol/L	1.04 [0.89, 1.24]	1.05 [0.89, 1.22]	1.09 ± 0.28	0.904
LDL, mmol/L	2.36 ± 0.87	2.43 ± 0.96	2.27 ± 0.71	0.288
Lp(a)	0.18 [0.11, 0.34]	0.20 [0.11, 0.34]	0.17 [0.11, 0.35]	0.682
eGFR, ml/min*1.73m^2	71.28 [57.15, 95.74]	77.50 [60.14, 98.00]	64.11 [52.35, 90.71]	0.038
TnI, ng/ml	0.02 [0.01, 0.04]	0.01 [0.01, 0.03]	0.02 [0.01, 0.05]	0.183
BNP, pg/ml	1540.00 [538.00, 2826.41]	1140.00 [419.50, 2385.00]	2215.00 [1140.00, 4625.00]	0.003
Echocardiographic parameters				
IVST, mm	10.00 [9.00, 11.00]	10.00 [9.00, 10.01]	10.00 [9.00, 11.00]	0.524
LVEDD, mm	60.00 [56.00, 65.00]	60.00 [56.50, 66.00]	59.50 [52.00, 64.25]	0.153
LVPWT, mm	9.00 [9.00, 10.00]	9.00 [9.00, 10.00]	9.00 [9.00, 10.25]	0.929
AO, mm	31.00 [29.00, 35.00]	31.52 ± 3.32	32.26 ± 4.58	0.333
LA，mm	46.49 ± 7.47	46.22 ± 6.88	46.00 [43.00, 51.25]	0.718
LVEF, %	39.00 [33.00, 44.00]	39.00 [32.50, 44.00]	38.50 [33.50, 44.00]	0.769

Abbreviations: BMI, Body mass index; BSA, Body surface area; NYHA, New York Heart Association functional; HF, heart failure; TC, Total Cholesterol; CM, Chylomicrons; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; Lp(a), Lipoprotein(a); eGFR, Estimated Glomerular Filtration Rate; Tnl, Troponin I; BNP, B-type Natriuretic Peptide; IVST, Interventricular Septal Thickness; LVEDD, Left Ventricular End-Diastolic Dimension; LVPWT, Left Ventricular Posterior Wall Thickness; AO, Aortic Root Diameter; LA, Left Atrial Diameter; LVEF, Left Ventricular Ejection Fraction.

Table 2 EAT volume level and risk of endpoints in entire cohort

EAT volume	Range	Hazard Ratio (95% CI) and *P* value			
		Unadjusted	Model 1	Model 2	Model 3
Q4	82.53-136.10	Reference			

Q3	65.60-82.52	1.758 (0.648, 4.769)	1.962 (0.696, 5.525)	1.700 (0.585, 4.946)	2.090 (0.699, 6.248)
		0.268	0.202	0.330	0.187
Q2	47.97-65.59	2.856 (1.094, 7.453)	3.601 (1.298, 9.988)	3.612 (1.307, 9.981)	4.137 (1.404, 12.188)
		0.032	0.014	0.013	0.010
Q1	13.21-47.96	3.587 (1.422, 9.049)	4.443 (1.593, 12.396)	4.575 (1.642, 12.751)	3.558 (1.174, 10.781)
		0.007	0.004	0.004	0.025
P for trend		0.004	0.003	0.002	0.018
EAT volume		0.575 (0.420, 0.788)	0.514 (0.352, 0.752)	0.489 (0.328, 0.729)	0.575 (0.374, 0.885)
(per 1 S.D.)					
		<0.001	<0.001	<0.001	0.012

Model 1: Adjusted for age, sex, BMI

Model 2: Model 1 plus Hypertension, Diabetes

Model 3: Model 2 plus BNP, LVEF

Abbreviations: EAT, Epicardial Adipose Tissue; BMI, Body Mass Index; LVEF, Left Ventricular Ejection Fraction.

Supplementary Files

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- Supplementarytable17.docx