

Association Between Epicardial Adipose Tissue and Contrast-Induced Acute Kidney Injury in Patients with ST-Segment Elevation Myocardial Infarction

Baohe Zang, Weiwei Hu, Yali Chao, Dengji Wang

Department of Intensive Care Medicine, Affiliated Hospital of Xuzhou Medical University, Xuzhou, Jiangsu, People's Republic of China

Correspondence: Dengji Wang, Department of Intensive Care Medicine, Affiliated Hospital of Xuzhou Medical University, Xuzhou, Jiangsu, 221000, People's Republic of China, Email xyfywdj@163.com

Background: Epicardial adipose tissue (EAT) affects the kidneys by secreting various bioactive molecules. Contrast-induced acute kidney injury (CI-AKI) is a common complication after percutaneous coronary intervention (PCI) in ST-segment elevation myocardial infarction (STEMI) patients. The relationship between EAT and CI-AKI remains unclear. This study aims to explore the relationship between EAT and the incidence of CI-AKI after PCI in STEMI patients.

Methods: Patients diagnosed with STEMI were continuously included, all patients underwent PCI within 12 hours of onset. EAT volume was measured and obtained by chest CT. Logistic regression analysis was used to analyze possible risk factors for CI-AKI. Restricted cubic splines (RCS) were utilized to explore the dose-response relationship involving EAT and CI-AKI.

Results: The incidence of CI-AKI was 8.9% (57/638). Compared with the Non-CI-AKI group, the EAT volume was significantly higher ($p < 0.005$). After adjusting for confounding factors, multivariate regression analysis showed FBG, NT-proBNP, LVEF, and EAT volume were the independent predictors for CI-AKI. RCS analysis indicated a linear dose-response relationship between EAT volume and CI-AKI. The integration of EAT volume could significantly improve ability of the model for CI-AKI (NRI 0.4071, 95% CI 0.231 ~ 0.583, $p < 0.001$; IDI 0.1356, 95% CI 0.091 ~ 0.180, $p < 0.001$).

Conclusion: Higher EAT volume was an independent risk factor for CI-AKI in STEMI patients. Integration of EAT volume could significantly improve the risk model for CI-AKI.

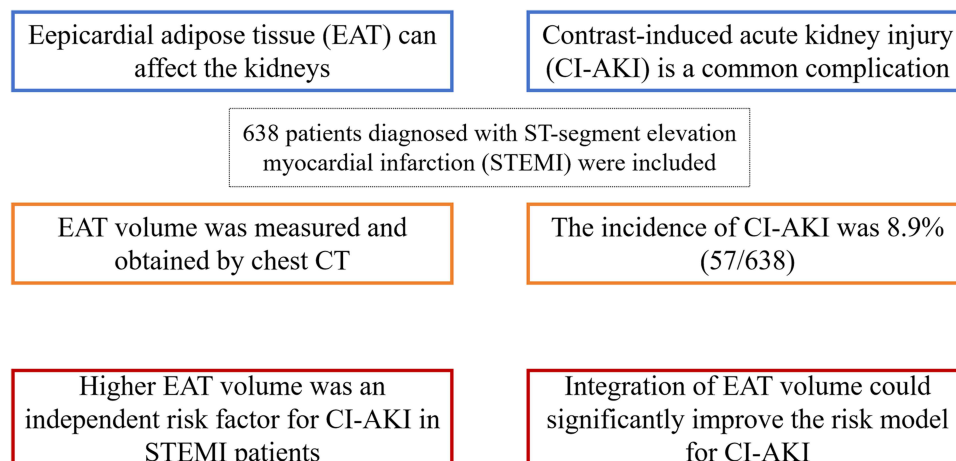
Keywords: epicardial adipose tissue, EAT, ST-segment elevation myocardial infarction, STEMI, contrast-induced acute kidney injury, CI-AKI, percutaneous coronary intervention, PCI

Introduction

Acute kidney injury (AKI) refers to the acute decline in kidney function over a short period, characterized by elevated serum creatinine levels, reduced urine output, or other signs of renal dysfunction.¹ Severe AKI may require admission to intensive care units for further medical care. For patients with ST-segment elevation myocardial infarction (STEMI), the incidence of AKI is relatively high and closely linked to patient prognosis.^{2,3} According to research, the incidence of Contrast-induced acute kidney injury (CI-AKI) in STEMI patients after percutaneous coronary intervention (PCI) ranges from 15% to 30%, which is associated with hemodynamic changes, oxidative stress, inflammatory responses, and the direct nephrotoxic effects of contrast agents.^{4,5} Currently, the main preventive treatment for CI-AKI in clinical practice is hydration.⁶ However, there may not be enough time for hydration before primary PCI in patients with STEMI. Therefore, it is important to identify more risk factors for CI-AKI in patients with STEMI.

Epicardial adipose tissue (EAT) is the adipose tissue that covers the outer surface of the heart. Recent studies have found that EAT is not merely a simple energy-storing tissue but plays an important role in inflammation, metabolism, and

Graphical Abstract



cardiovascular diseases.⁷ The relationship between EAT and cardiovascular diseases has been well studied.⁸ In patients with myocardial infarction, increased EAT volume is associated with poor prognosis.^{9,10} Furthermore, EAT secretes various bioactive molecules, such as adipokines, cytokines, and hormones, that affect not only the surrounding heart tissue but also distant organs, including the kidneys.^{11–13} However, the relationship between EAT and CI-AKI remains unclear. This study aims to explore the relationship between EAT and the incidence of CI-AKI after PCI in STEMI patients.

Materials and Methods

Study Population

This analysis reviewed patients diagnosed with STEMI¹⁴ from January 2022 to November 2024. The criteria for inclusion were: (1) completion of successful primary PCI within 12 hours following the onset of symptoms (TIMI = 3); (2) Improvement of CT examination during hospitalization; (3) possession of comprehensive clinical data. Patients were excluded if they satisfied any of the subsequent criteria: (1) poor quality of CT image; (2) undergoing dialysis prior to admission or suffering from chronic renal failure ($\text{eGFR} < 30 \text{ mL} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2}$); (3) having active inflammatory conditions (including pulmonary infections, intestinal inflammation, or autoimmune diseases); (4) a prior history of malignancy or blood disorders; (5) having been exposed to other radiographic contrast materials or nephrotoxic drugs within 48 hours prior to or 72 hours after PCI. This study complies with the Declaration of Helsinki and has been approved by the Institutional Review Board (IRB) of the Affiliated Hospital of Xuzhou Medical University. As this retrospective study posed no risk to patients, the necessity for written informed consent was waived per IRB regulations. Ultimately, 638 patients were included in the study (Figure 1).

Clinical Data Assessment

Patient records provided the clinical data, encompassing variables such as age, sex, body mass index (BMI), medical history, medications, left ventricular ejection fraction (LVEF), and PCI-related specifics. Serum creatinine (Scr) levels prior to PCI, along with Scr measurements taken 48–72 hours post-exposure to the contrast agent, were carefully noted. Contrast-induced acute kidney injury (CI-AKI) was characterized by an increase in Scr of at least 50% or a rise of 0.3 mg/dL within the 48–72 hour window after contrast administration.¹⁵ Moreover, during hospitalization, peak levels of high-sensitivity C-reactive protein (hs-CRP), high-sensitivity troponin T (hs-TnT), and N-terminal pro B-type natriuretic peptide (NT-proBNP) were documented. The medications provided to patients included aspirin, P2Y12 inhibitors, β -

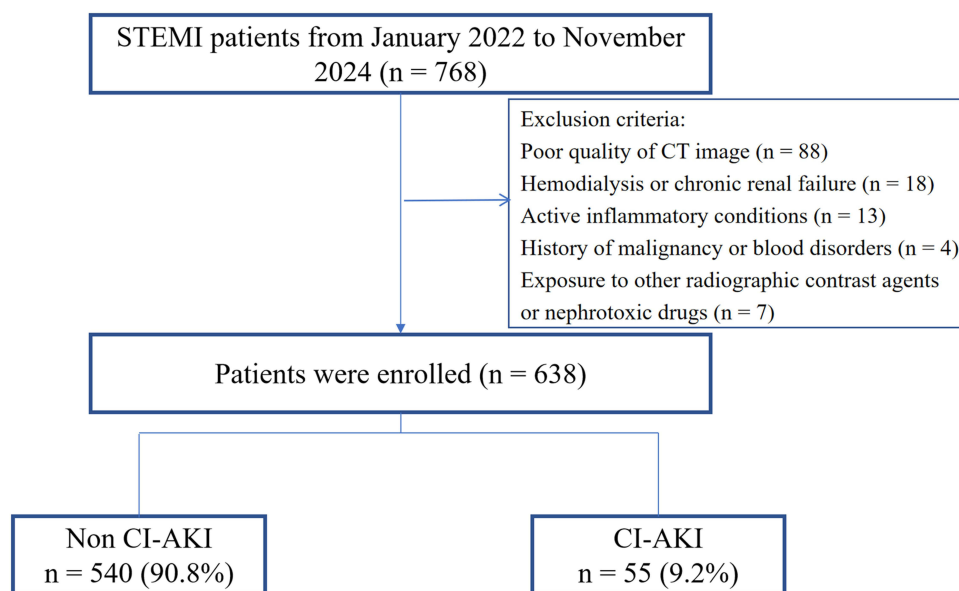


Figure 1 Study flowchart.

Abbreviations: CI-AKI, contrast-induced acute kidney injury; STEMI, ST-segment elevation myocardial infarction.

blockers, statins, nitrates, angiotensin-converting enzyme inhibitors (ACEI) or angiotensin receptor blockers (ARB), and diuretics. All PCI operations and postoperative hydration were performed according to the latest guidelines, and the infarct-related artery (IRA) was determined on the basis of the coronary angiography findings.

Measurement of Epicardial Adipose Tissue

CT imaging was performed using the spiral CT machine (SOMATOM Definition, SIEMENS, Germany). Scanning with enhancement started at the root of the ascending aorta with a threshold set between 90 and 100 hU, initiated 6 seconds post-start and continuing for a duration of 5 to 12 seconds. The range of the scan covered from 1 cm beneath the tracheal carina to 1.5 cm beneath the lower border of the heart, employing a tube current of 280 to 350 mA and a voltage of 120 kV. The total epicardial adipose tissue, positioned within the pericardial sac stretching from the bifurcation of the pulmonary artery to the diaphragm, was reconstructed from 10 mm axial slices. Enhanced images allowed for the identification of EAT using Hounsfield units in the range of -50 to -200 . The total adipose tissue was categorized as EAT, and its volume was subsequently calculated ([Figure 2](#)). The measurement of EAT was performed independently by two experienced doctors who were unaware of this study, and each reader performed repeat measurements. The inter-observer and intra-observer agreement for each EAT measurement seen in [Supplementary Table 1](#).

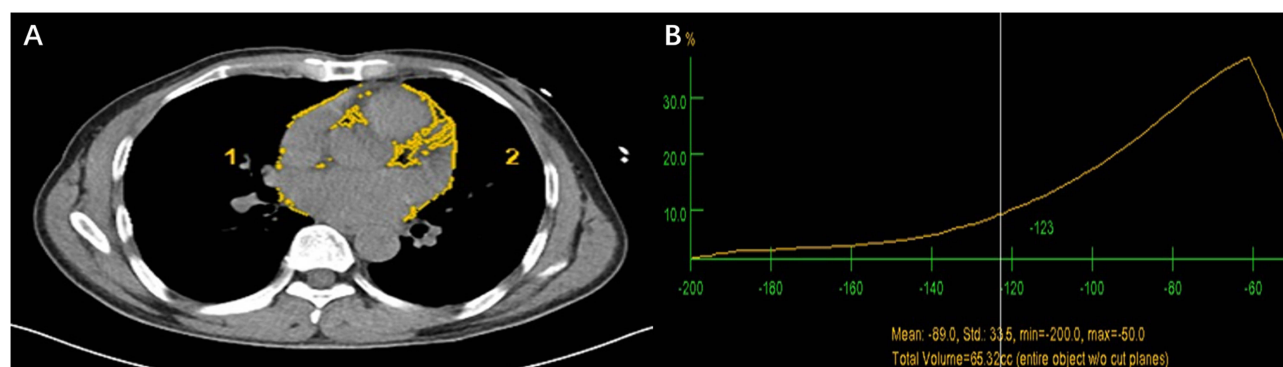


Figure 2 The measurement of epicardial adipose tissue. (A) cardiac axial map, and yellow regions represent epicardial adipose tissue; (B) At -50 and -200 hounsfield units (HU), epicardial adipose tissue volume; “1” represents the right side, and “2” represents the left side.

Statistical Analysis

The analysis of data was conducted using SPSS (version 27.0, Chicago, USA) and R (version 4.3.1). To evaluate data normality, the Kolmogorov–Smirnov test was utilized. Continuous variables conforming to a normal distribution were expressed as mean \pm standard deviation and analyzed with independent *t*-tests. In contrast, variables that did not follow a normal distribution were represented as median (Q25, Q75) and assessed using the Mann–Whitney *U*-test. Categorical variables were summarized in terms of counts and percentages, with comparisons made using the χ^2 -test. Factors deemed as potential risks, determined through univariate regression analysis, were incorporated into a multivariate regression analysis to pinpoint independent risk factors related to CI-AKI. To explore the dose-response relationship involving caIMR and CI-AKI, restricted cubic splines (RCS) were utilized. The new model's predictive accuracy and the baseline model's accuracy were evaluated through receiver operating characteristic (ROC) curves, the net reclassification index (NRI), and integrated discrimination improvement (IDI). A *p*-value less than 0.05 was regarded as statistically significant.

Results

Baseline Characteristics of the Study Population

The incidence of CI-AKI was 8.9% (57/638). Compared with the Non-CI-AKI group, the fasting blood glucose (FBG), EAT volume, hs-CRP, NT-proBNP, and the prevalence of diabetes mellitus, IRA-left anterior descending (LAD), and diuretics were significantly higher, and LVEF was significantly lower in the CI-AKI group (*p*<0.005) (Table 1).

Table 1 Comparison of Baseline Data in Patients with STEMI

	Total (n = 638)	Non-CI-AKI (n = 581)	CI-AKI (n = 57)	P
Age, years	62.92 \pm 13.41	62.77 \pm 13.63	64.44 \pm 10.98	0.288
Female, n (%)	137 (21.47)	122 (21.00)	15 (26.32)	0.351
Heart rate, bpm	79.67 \pm 14.30	79.57 \pm 14.44	80.61 \pm 12.83	0.601
SBP, mmHg	127.35 \pm 20.10	127.35 \pm 20.24	127.28 \pm 18.78	0.979
DBP, mmHg	78.97 \pm 13.97	79.00 \pm 14.07	78.63 \pm 13.03	0.850
BMI, kg/m ²	24.66 \pm 3.91	24.59 \pm 3.96	25.37 \pm 3.33	0.149
Hypertension, n (%)	287 (44.98)	257 (44.23)	30 (52.63)	0.224
Diabetes mellitus, n (%)	166 (26.02)	144 (24.78)	22 (38.60)	0.023*
CKD, n (%)	21 (3.29)	20 (3.44)	1 (1.75)	0.770
Smoking, n (%)	291 (45.61)	270 (46.47)	21 (36.84)	0.164
HGB, g/L	139.81 \pm 16.86	139.84 \pm 16.83	139.46 \pm 17.35	0.870
Plt, 10 ⁹ /L	218.16 \pm 59.40	219.02 \pm 60.28	209.40 \pm 49.16	0.244
Serum creatinine, μ mol/L	68.11 \pm 21.22	68.41 \pm 21.57	65.11 \pm 17.10	0.262
eGFR, mL/min/1.73 m ²	101.94 \pm 20.85	102.02 \pm 21.08	101.18 \pm 18.49	0.772
FBG, mmol/L	6.87 \pm 2.84	6.75 \pm 2.77	8.10 \pm 3.33	0.004 [#]
Total cholesterol, mmol/L	4.26 \pm 1.02	4.25 \pm 1.02	4.31 \pm 0.95	0.722
Triglycerides, mmol/L	1.49 \pm 1.04	1.50 \pm 1.07	1.42 \pm 0.73	0.614
HDL-C, mmol/L	0.98 \pm 0.23	0.98 \pm 0.24	1.00 \pm 0.16	0.346
LDL-C, mmol/L	2.75 \pm 0.88	2.74 \pm 0.89	2.84 \pm 0.87	0.381
EAT volume, mL	126.66 \pm 49.51	122.22 \pm 45.88	172.00 \pm 61.47	<0.001 [#]
Peak hs-CRP, mg/L	2.40 (0.60, 7.88)	2.30 (0.50, 7.50)	2.80 (1.50, 9.80)	0.028 ^{&}
Peak hs-TnT, ng/L	534.9 (101.2, 1900.3)	517.0 (86.5, 1924.0)	961.9 (158.0, 1652.0)	0.106
Peak NT-proBNP, pg/mL	1362.7 (567.4, 3105.5)	1243.0 (522.0, 2926.0)	2932.0 (1576.0, 4331.0)	<0.001 ^{&}
IABP, n (%)	18 (2.82)	15 (2.58)	3 (5.26)	0.455
LVEF, %	51.88 \pm 7.15	52.28 \pm 6.95	47.75 \pm 7.98	<0.001 [#]

(Continued)

Table 1 (Continued).

	Total (n = 638)	Non-CI-AKI (n = 581)	CI-AKI (n = 57)	P
Killip class, n (%)				0.122
I	545 (85.42)	499 (85.89)	46 (80.70)	
II	31 (4.86)	30 (5.16)	1 (1.75)	
III	2 (0.31)	2 (0.34)	0 (0.00)	
IV	60 (9.40)	50 (8.61)	10 (17.54)	
Contrast agent, mL	104.12 ± 38.69	103.75 ± 39.06	107.89 ± 34.73	0.441
IRA-LAD, n(%)	320 (50.16)	284 (48.88)	36 (63.16)	0.040*
IRA-LCX, n(%)	63 (9.87)	59 (10.15)	4 (7.02)	0.449
IRA-RCA, n(%)	253 (39.66)	236 (40.62)	17 (29.82)	0.112
IRA-LM, n(%)	2 (0.31)	2 (0.34)	0 (0.00)	1
Aspirin, n(%)	636 (99.69)	579 (99.66)	57 (100.00)	1
P2Y12, n(%)	637 (99.84)	580 (99.83)	57 (100.00)	1
Statins, n(%)	634 (99.37)	577 (99.31)	57 (100.00)	1
ACEI/ARB, n(%)	291 (45.61)	266 (45.78)	25 (43.86)	0.781
β-blockers, n(%)	558 (87.46)	510 (87.78)	48 (84.21)	0.437
Nitrates, n(%)	255 (39.97)	238 (40.96)	17 (29.82)	0.101
Heparin, n(%)	528 (82.76)	481 (82.79)	47 (82.46)	0.949
Diuretics, n(%)	311 (48.75)	274 (47.16)	37 (64.91)	0.011*

Notes: * χ^2 -test; # *t*-tests; & Mann–Whitney *U*-test.

Abbreviations: BMI, body Mass Index; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; CKD, chronic kidney disease; SBP, systolic blood pressure; DBP, diastolic blood pressure; LAD, left anterior descending; IRA, infarct related artery; LCX, left circumflex artery; RCA, right coronary artery; ACEI, angiotensin-converting-enzyme inhibitor; ARB, angiotensin II receptor blocker; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; hs-CRP, high sensitivity C-reactive protein; hs-TnT, high sensitivity troponin T; NT-proBNP, N-terminal pro-B-type natriuretic peptide; FBG, fasting blood glucose; STEMI, ST-segment elevation myocardial infarction; EAT, epicardial adipose tissue; CI-AKI, contrast-induced acute kidney injury; HGB, hemoglobin; STEMI, ST-segment elevation myocardial infarction; eGFR, estimated glomerular filtration rate.

Logistic Regression Analysis for CI-AKI

In univariate regression analysis, FBG, NT-proBNP, diabetes mellitus, IRA-LAD, and diuretics, LVEF, and EAT volume were associated with CI-AKI. In multivariate regression analysis, the potential risk variables in the univariate were included. The result showed FBG, NT-proBNP, LVEF, and EAT volume were still significantly associated with CI-AKI ([Supplementary Table 2](#), [Table 2](#)). RCS analysis indicated a linear dose-response relationship between EAT volume and CI-AKI, suggesting that higher EAT volume are associated with an increased risk of CI-AKI ([Figure 3](#)).

Diagnostic Value of EAT in CI-AKI

ROC showed that the areas under the curve (AUC) for LVEF, FBG, NT-proBNP, and EAT volume in diagnosing CI-AKI were 0.668, 0.646, 0.690, and 0.737, respectively ($P < 0.05$). The cut-off value of EAT volume was 147.36 mL, the

Table 2 Multivariate Logistic Regression Analysis on CI-AKI in Patients with STEMI

	OR (95% CI)	P
Peak NT-proBNP, pg/mL	1.453(1.115~1.895)	0.006
FBG, mmol/L	1.128(1.034~1.232)	0.007
EAT volume, mL	1.017(1.012~1.022)	<0.001
LVEF, %	0.939(0.903~0.976)	0.001

Abbreviations: FBG, fasting blood glucose; EAT, epicardial adipose tissue; NT-pro BNP, N-terminal pro-B-type natriuretic peptide; LVEF, left ventricular ejection fraction; STEMI, ST-segment elevation myocardial infarction; CI-AKI, contrast-induced acute kidney injury.

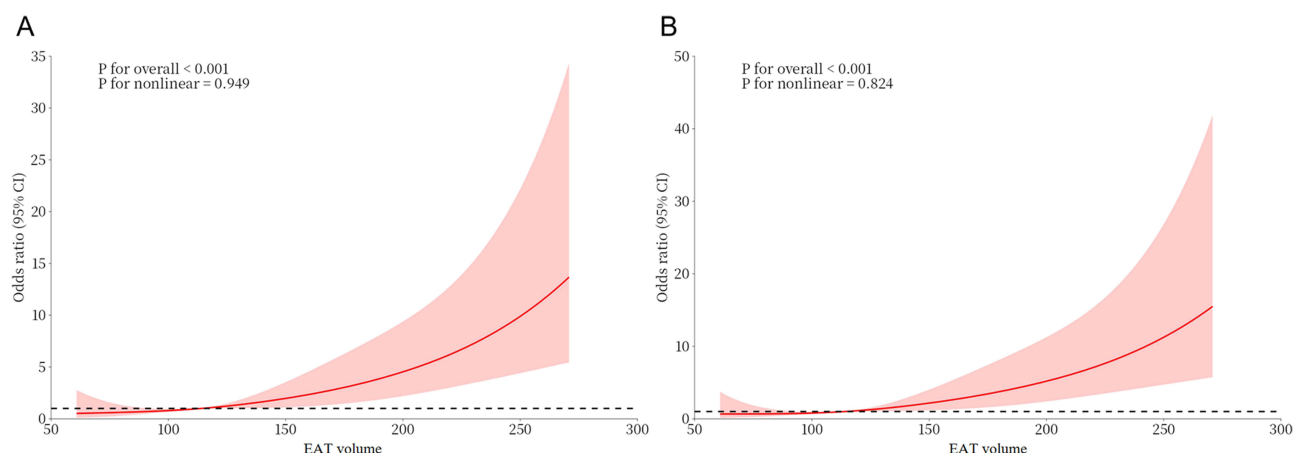


Figure 3 Dose-response relationship between EAT and CI-AKI. (A) a unadjusted dose-response relationship between EAT and CI-AKI; (B) an adjusted dose-response relationship between EAT and CI-AKI.

Abbreviations: CI-AKI, contrast-induced acute kidney injury; EAT, epicardial adipose tissue.

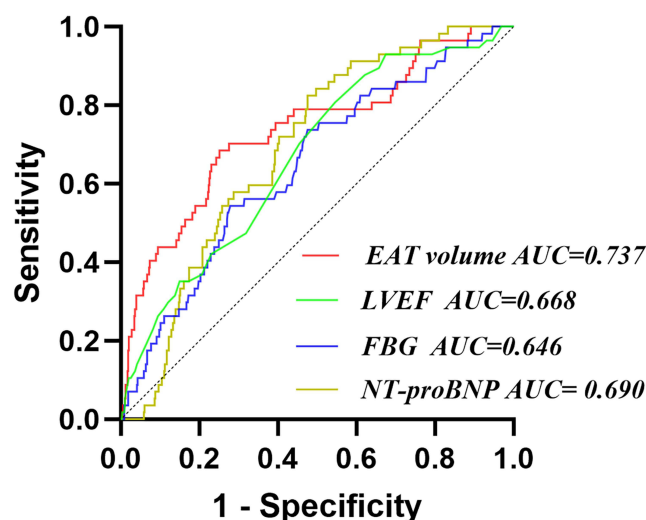


Figure 4 Receiver operating characteristic analysis (ROC) of EAT for identifying CI-AKI.

Abbreviations: CI-AKI, contrast-induced acute kidney injury; EAT, epicardial adipose tissue; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; FBG, fasting blood glucose.

sensitivity was 68.4%, and the specificity was 74.9% (95% CI: 0.663~0.811, $p < 0.001$) ([Supplementary Table 3](#), [Figure 4](#)).

Incremental Value of EAT in the Risk Model for CI-AKI

A baseline model (LVEF, FBG, and NT-proBNP) was constructed based on the results of multivariate regression analysis. ROC showed that the baseline model had an AUC of 0.745 (95% CI: 0.688~0.801), with a sensitivity of 91.2% and specificity of 52.0%. After integrating EAT volume in the baseline model, ROC showed that the AUC was 0.818 (95% CI: 0.767~0.869), with a sensitivity of 86.0% and specificity of 62.7% ([Supplementary Table 4](#), [Figure 5](#)). Next, the NRI and IDI were calculated. The results showed that when EAT volume was integrated into the baseline model, the NRI>0 (NRI 0.4071, 95% CI 0.231 ~ 0.583, $p < 0.001$) and the IDI value was improved by 13.56% (IDI 0.1356, 95% CI 0.091 ~ 0.180, $p < 0.001$), suggesting that the integration of EAT volume could significantly improve ability of the model for CI-AKI ([Table 3](#)).

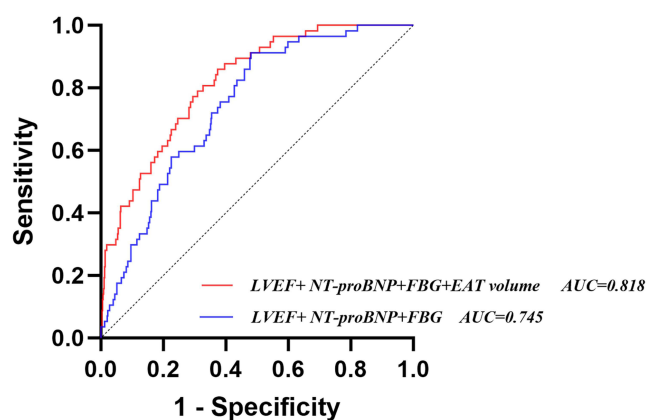


Figure 5 Receiver operating characteristic analysis (ROC) of models for identifying CI-AKI.

Abbreviations: CI-AKI, contrast-induced acute kidney injury; EAT, epicardial adipose tissue; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; FBG, fasting blood glucose.

Discussion

The main findings of this study are as follows: (1) EAT volume was independently associated with CI-AKI in STEMI patients; (2) A linear dose-response relationship exists between EAT volume and CI-AKI; (3) Integration of EAT volume could significantly improve the risk model for CI-AKI.

PCI has been proven to be the most effective method for rapidly restoring coronary blood flow in STEMI patients.¹⁶ With the widespread use of contrast agents globally, CI-AKI has become a major iatrogenic cause of kidney injury.⁵ Consistent with previous studies, our research shows that the incidence of CI-AKI in STEMI patients is 8.9%.^{17,18} In addition to a higher incidence, various data suggest that CI-AKI is associated with poor clinical outcomes.^{3–5} Therefore, early recognition of CI-AKI is crucial.

EAT is considered not only as the “energy storage tissue” of the heart but also plays a significant role in cardiovascular and systemic metabolic regulation through the secretion of various bioactive substances such as adipokines, cytokines, hormones, and metabolites.^{7–10} In STEMI patients, numerous studies have demonstrated that increased EAT volume is associated with more severe disease and poorer prognosis.^{19–21} In recent years, more studies have focused on the potential role of EAT in kidney diseases.^{11–13} Clinical research has found that EAT thickness is positively correlated with renal dysfunction, particularly in patients with chronic kidney disease, where EAT accumulation is closely related to tubulointerstitial fibrosis, changes in renal blood flow, and metabolic disturbances.^{11,22} Moreover, some cohort studies have shown that increased EAT thickness is associated with elevated urinary albumin levels, renal tubular dysfunction, and increased inflammatory markers, suggesting that EAT may influence kidney metabolism and inflammation, thereby accelerating the progression of kidney disease through its relationship with metabolic syndrome.^{23–25} Additionally, a previous study showed that patients with hypertension tend to have thicker EAT, which may be related to microvascular renal damage and tubular dysfunction.²⁶ However, the relationship between EAT and CI-AKI remains unclear. Our study found that EAT volume was independently associated with CI-AKI in STEMI patients, and there was a linear dose-response relationship between EAT volume and CI-AKI. The following mechanisms may partially explain the findings of this study. EAT secretes multiple pro-inflammatory factors, which can enter the kidneys through the

Table 3 Incremental Value of EAT Volume for CI-AKI

	NRI		IDI	
	Estimate (95% CI)	P	Estimate (95% CI)	P
FBG+LVEF+NT-proBNP	Reference	-	Reference	-
LVEF+FBG+NT-proBNP+EAT volume	0.835 (0.583 ~ 1.087)	<0.001	0.105 (0.060 ~ 0.150)	<0.001

Abbreviations: FBG, fasting blood glucose; EAT, epicardial adipose tissue; NT-pro BNP, N-terminal pro-B-type natriuretic peptide; LVEF, left ventricular ejection fraction; CI-AKI, contrast-induced acute kidney injury.

bloodstream, activate local immune responses, and lead to tubular cell damage and acute renal failure.^{24,25} Dysregulated fatty acid metabolism in EAT may result in the accumulation of free fatty acids, which can promote oxidative stress, increase reactive oxygen species production, and thereby cause kidney damage.^{11,27} Furthermore, increased EAT may be associated with chronic diseases such as obesity, diabetes, and hypertension, all of which are independent risk factors for CI-AKI. STEMI patients often have these chronic conditions, and the increase in EAT may exacerbate the renal damage caused by these underlying diseases, making the use of contrast agents even more dangerous.^{22,28,29} Finally, the effect of EAT on the kidneys is not limited to direct metabolic effects, it may also indirectly worsen acute kidney injury through its impact on the cardiovascular system. Previous studies have shown that increased EAT is associated with larger infarct size and lower LVEF, which may lead to hypoperfusion and ischemic AKI.^{30–33} Moreover, our study also found that EAT significantly improved the risk model for CI-AKI. Therefore, our study provides additional information for risk stratification in STEMI patients. Clinically, patients with larger EAT volumes may face a higher risk of CI-AKI. Future research should further explore the specific mechanisms of EAT in STEMI and CI-AKI and evaluate its feasibility as a clinical intervention target to improve the prognosis of STEMI patients.

Limitations

First, this is a retrospective study, which has some unavoidable limitations, and our research cannot establish a definitive causal relationship between EAT and CI-AKI. Second, more basic research is needed to clarify how EAT affects the occurrence of CI-AKI in STEMI patients through mechanisms such as metabolism, inflammation, and cardiac remodeling. Third, our study included only patients diagnosed with STEMI, some findings may not be applicable to other diseases and need to be validated through replication studies. Fourth, the measurement of EAT involves various methods. Cardiac magnetic resonance (CMR) is currently considered the best imaging tool for the evaluation of EAT. Therefore, in future studies, relevant evidence from CMR may be worth anticipating.

Conclusion

Higher EAT volume was an independent risk factor for CI-AKI in STEMI patients. Integration of EAT volume could significantly improve the risk model for CI-AKI.

Data Sharing Statement

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

This study complies with the Declaration of Helsinki and has been approved by the Institutional Review Board (IRB) of the Affiliated Hospital of Xuzhou Medical University. The requirement for signed written consent was waived owing to no risk to the patient in accordance with the relevant IRB regulatory guidelines.

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Disclosure

The authors declare that they have no competing interests in this work.

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