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Original Article

Evaluation of Regression in Epicardial Adipose Tissue Thickness on Cardiac MRI in Post-Acute Coronary Syndrome Patients

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ABSTRACT

Objective: To evaluate changes in epicardial adipose tissue (EAT) on cardiac magnetic resonance imaging (CMRI) in patients treated for acute coronary syndrome (ACS) and to assess its association with the severity of coronary artery disease (CAD).
Study Design: Prospective longitudinal study.
Place and Duration of Study: Cardiology Department of Armed Forces Institute of Cardiology, National Institute of Heart Diseases, Rawalpindi, Pakistan; from Aug 2025 to Nov 2025.
Methods: Overall, 102 ACS patients aged 18–65 years, underwent both PCI and cardiac magnetic resonance imaging (CMR) were enrolled through consecutive sampling. Prior CABG patients, patients with LVEF <40%, MRI contraindications, eGFR <30 mL/min/1.73m², or statin intolerance were excluded from study. EAT thickness was measured on baseline and 3-month follow-up CMR scans, with regression defined as ≥10% reduction in EAT thickness. Cardiovascular outcomes, included stroke, revascularization, heart failure, and mortality, and assessed at follow-up.
Results: A total of 102 patients with a median age of 60(52.75-63.00) years were included, of whom 64(62.7%) were male. The median EAT thickness decreased from 0.48 mm (IQR: 0.42–0.58) at baseline to 0.45 mm (IQR: 0.40–0.53) at 3 months. The EAT regression was 0.03 mm (IQR: 0.05-0.02). This reduction was statistically significant (p<0.001). Which did not meet the predefined ≥10% regression criterion. The correlation between CAD severity and EAT regression was weaker but statistically significant (r=0.302, p=0.002).
Conclusion: EAT thickness showed a strong positive correlation with CAD severity but did not demonstrate significant regression over three months.

Keywords: Acute Coronary Syndrome, Cardiac Magnetic Resonance Imaging, Coronary Artery Disease, Epicardial Adipose Tissue.

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INTRODUCTION

Epicardial adipose tissue (EAT) is a metabolically active layer of visceral fat that is present between the visceral layer of the pericardium and the heart muscle (myocardium). Unlike subcutaneous fat, EAT is in direct contact with the coronary arteries and shares their microcirculation, allowing it to influence the heart through local (paracrine and vasocrine) signalling.[1] This close anatomical link gives EAT a unique role in heart health and disease, specifically in the coronary artery disease (CAD) development and progression. By releasing pro-inflammatory cytokines and adipokines, EAT can promote endothelial dysfunction, atherosclerotic plaque formation, and ultimately plaque instability.[2,3]

Increased EAT thickness and volume have been associated to poorer cardiovascular outcomes in acute coronary syndrome (ACS) patients, including recurrent ischemic events and adverse cardiac remodelling.[4] Subsequently, EAT has emerged as both a potential therapeutic target and a valuable prognostic biomarker.[4,5] Cardiac magnetic resonance imaging (CMR) gives a high-resolution, and noninvasive method for assessing EAT thickness, making it particularly beneficial for tracking longitudinal changes in fat volume.[6]

Numerous pharmacological therapies, (e.g., statins, glucagon-like peptide-1 receptor agonists (GLP-1 RAs), and sodium-glucose cotransporter-2 (SGLT2) inhibitors), have shown potential in reducing visceral and epicardial fat in both metabolic and cardiovascular disease contexts.[7,8] However, there is still limited prospective evidence on how EAT volume changes following an ACS event, especially in real-world clinical practice.

However, despite such evidence, data evaluating short-term EAT regression following ACS and its

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relationship with clinical outcomes remain scarce. Given the inflammatory and pro-atherogenic role of EAT in ACS, tracking its regression may serve as an indirect indicator of cardiovascular recovery and therapeutic response. Evaluating changes in EAT thickness after an ACS event could present a new contribution to the cardiometabolic impact of post-ACS management strategies and help refine risk stratification and secondary prevention approaches. The study aimed to evaluate changes in epicardial adipose tissue (EAT) on cardiac magnetic resonance imaging (CMRI) in patients treated for acute coronary syndrome (ACS) and to assess its association with the severity of coronary artery disease (CAD).

METHODOLOGY

This Prospective longitudinal study was conducted in the Cardiology Department of the Armed Forces Institute of Cardiology/National Institute of Heart Diseases (AFIC/NIHD), Rawalpindi, Pakistan, over a period of three months after receiving ethical approval under IERB letter number (9/2/R&D/2025/374; Dated: 24th Jul,2025). A non-probability consecutive sampling technique was used to recruit eligible participants with ACS who underwent both CMR and PCI.

The sample size was calculated based on a previously reported mean EAT thickness of 0.41±0.18 cm in post-PCI patients,[9] 95% confidence level and a 5% margin of error was maintained, which yielded 95 participants. However, for increasing the study's statistical power, a total of 102 patients were finally enrolled.

Inclusion Criteria: Patients aged between 18 and 65 years, regardless of gender, diagnosed with ACS (including STEMI, NSTEMI, or unstable angina), and who underwent baseline CMR within 15 days of the index event were included.

Exclusion Criteria: Patients with a prior history of coronary artery bypass grafting (CABG), LVEF <40%, contraindications to MRI (such as metallic implants or severe claustrophobia), estimated glomerular filtration rate (eGFR) less than 30 mL/min/1.73m², or statin intolerance and lost to follow-up were excluded.

Regression of epicardial fat was defined as a reduction of 10% or more in EAT thickness measured on CMR between baseline and three-month follow-up.[10] After obtaining written informed consent from the study participants, the baseline detailed demographic data, clinical data and laboratory investigations were recorded. EAT measurements were performed using standardised short-axis cine images, and fat boundaries were delineated through validated manual or semi-automated contouring techniques on contiguous slices.[10] All scans were performed on a 1.5-T scanner using ECG-gated, breath-hold cine bSSFP sequences with constant, vendor-optimised parameters. EAT thickness was measured on end-diastolic frames, primarily on mid-ventricular short-axis slices at the right ventricular free wall, with complementary measurements on mid-ventricular 4-chamber and long-axis views for reproducibility. On each image, EAT thickness was defined as the shortest perpendicular distance from the epicardial surface to the visceral pericardium or, when indistinct, to the outer boundary of epicardial fat.

Medications initiated during hospitalisation, such as, antiplatelets, beta-blockers. During hospitalisation, patients were monitored following PCI or medical stabilisation until discharge. Clinical stability, discharge medications, and echocardiographic parameters were observed. All patients received standardised post-discharge care in accordance with current ACS management guidelines. They were counselled regarding medication adherence, dietary and lifestyle modifications, and follow-up schedules.

At three months post-discharge, a follow-up reminder was sent by telephone, and patients returned for a follow-up evaluation. Clinical assessment and repeat CMR were performed to measure changes in EAT thickness. Medication adherence, any change in treatment regimen, and interval cardiovascular events were recorded as described above. Clinical outcomes included stroke (new focal neurological deficit lasting ≥24 hours and confirmed on CT or MRI), repeat revascularisation (any subsequent PCI or CABG for recurrent ischemia), new or worsening heart failure requiring intravenous therapy, and all-cause mortality. For heart failure events, LVEF, BNP or NT-proBNP levels, and NYHA class were noted. All data were anonymised and entered into a predesigned pro forma. Ethical standards were maintained throughout the study.

Data were analysed using Statistical Package for Social Sciences (SPSS) version-23. Data normality was assessed using the Shapiro-Wilk test, and all continuous variables (age, blood pressure, hemoglobin, HbA1C, Syntax score and EAT thickness)

were non-normally distributed, therefore, they were reported as medians and interquartile ranges. Categorical variables such as gender, comorbidities, and clinical outcomes were presented as frequencies and percentages. The Wilcoxon signed-rank test was used to compare EAT measurements at baseline and at the 3-month follow-up. A correlation between baseline EAT thickness and CAD severity was found using Spearman's correlation analysis. A *p*-value of <0.05 was considered statistically significant.

RESULTS

The study included 102 participants with a median age of 60.00(52.75–63.00) years. The majority were male 64(62.7%), while female participants were 38(37.3%). Among comorbidities, followed by diabetes mellitus, 35(34.3%). The median diastolic blood pressure was 79.00(75.00–85.00) mmHg, and the systolic blood pressure was 120.00(115.00–128.00) mmHg. The median haemoglobin level was 13.35(12.50–13.60) g/dL, HbA1c was 6.00(5.20–6.60)%, and the SYNTAX score was 22.00 (14.00–33.25). Regarding the extent of CAD, SVCAD was the most common pattern, observed in 39(38.2%) participants. This was followed by DVCAD in 35(34.3%) and TVCAD in 28(27.5%) participants. Medication adherence among participants was high, with 96(94.1%) reported good compliance, 4(3.9%) fair compliance, and only 2(2.0%) poor compliance. (Table-I)

Table-I: Baseline characteristics of study participants (n=102)

Variables	Frequency (%)
Gender	
Male	64(62.7%)
Female	38(37.3%)
Comorbids	
Diabetes Mellitus	35(34.3%)
Hypertension	23(22.5%)
Dyslipidemia	26(25.5%)
CKD	9(8.8%)
CAD	
SVCAD	39(38.2%)
DVCAD	35(34.3%)
TVCAD	28(27.5%)
Medication Compliance	
Good	96(94.1%)
Fair	4(3.9%)
Poor	2(2.0%)
	Median(IQR)
Age	60.00(52.75-63.00)
Diastolic Blood Pressure (mmHg)	79.00(75.00-85.00)
Systolic Blood Pressure (mmHg)	120.00(115.00-128.00)
Hemoglobin (g/dL)	13.35(12.50-13.60)
HbA1C (%)	6.00(5.20-6.60)
SYNTAX Score	22.00(14.00-33.25)

CAD=Coronary Artery Disease; ACS=Acute Coronary Syndrome; STEMI=ST-Elevation myocardial Infarction; NSTEMI=Non- ST-Elevation myocardial Infarction

Figure-1 the scatter plot shows a strong and statistically significant positive correlation between Coronary Artery Disease (CAD) severity and Epicardial Adipose Tissue (EAT) thickness at both baseline and at 3-month follow-up. At baseline, CAD severity showed a very strong positive correlation with EAT thickness (r = 0.806, p<0.001), which remained strong at 3 months (r = 0.770, p<0.001).

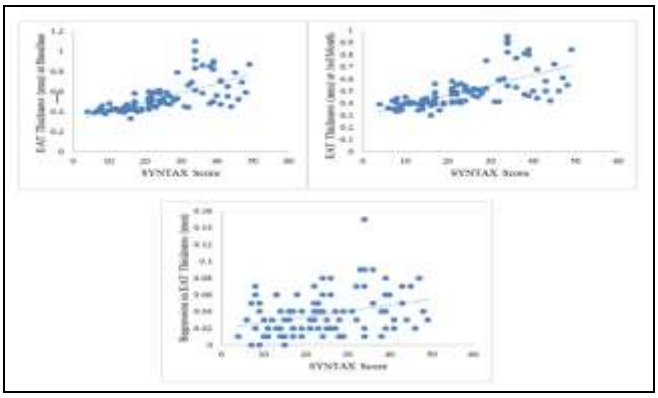

Figure-1: Correlation of CAD severity with EAT Thickness at Baseline, 3rd Month and Regression in EAT (n=102)

In contrast, the correlation between CAD severity and EAT regression was weaker but still statistically significant (r = 0.302, p=0.002), indicating that patients with more severe CAD experienced greater in EAT thickness over time.

Epicardial Adipose Tissue (EAT) thickness showed a statistically significant reduction from baseline to 3 months. The median EAT thickness decreased from 0.48 mm (IQR: 0.42–0.58) at baseline to 0.45 mm (IQR: 0.40–0.53) at 3 months. This reduction was statistically significant (p<0.001). The median EAT regression was 0.03 mm (IQR: 0.05-0.02).

However, according to the study definition, EAT regression was characterised as a ≥10% reduction in thickness. Since the observed decrease was less than 10%, no significant regression of EAT thickness was documented among our patients with ACS. This finding predicts that while there may be a minor improvement trend, short-term follow-up or standard therapeutic management alone may not be sufficient to induce meaningful regression of epicardial fat in ACS patients.

As there was no regression in EAT thickness per the defined criteria. However, at the 3-month follow-up, heart failure was the most frequent adverse event, affecting 4(3.9%) participants, followed by stroke 3(2.9%), mortality 3(2.9%), and revascularisation

1(1.0%). These outcomes, illustrated in Figure-2, indicate that major clinical events occurred in a small proportion of the cohort during follow-up.

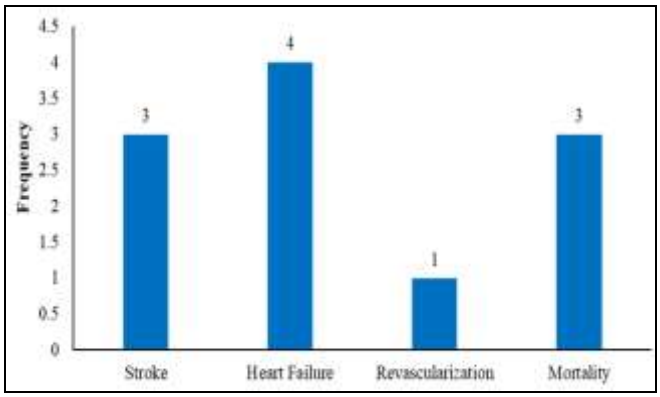

Figure-2: Clinical Outcomes of the Study Participants at 3rd Month follow-up (n=102)

Table-II: Comparison of Baseline and 3-Month Epicardial Adipose Tissue Thickness and Regression

Variable	Median(IQR)	*p*-value
EAT Thickness Beginning	0.48 (0.42-0.58)	<0.001
EAT Thickness 3rd.Month	0.45(0.40-0.53)	
EAT Regression	0.03(0.05-0.02)	-

DISCUSSION

This prospective study examined regression in EAT thickness on cardiac MRI among patients recovering from ACS, focusing on whether meaningful regression of EAT could be achieved within a 3-month follow-up period. The study found a significant increase in CAD severity, as determined by Syntax score, with increased EAT thickness. There was a decrease in EAT thickness at the third-month follow-up, but, as defined, a ≥10% decrease was considered regression, which was not achieved.

EAT, a specialised visceral fat layer enveloping the myocardium, has gained recognition as an active metabolic tissue involved in CAD through the secretion of inflammatory and atherogenic mediators that influence the coronary vessels.[11,12] Because of its intimate structural and functional proximity to the heart and coronary arteries, changes in EAT thickness are thought to mirror shifts in cardiovascular metabolic activity and disease progression.[13] Given the limited literature on EAT regression among patients with ACS, the present study provides valuable insight into an underexplored area. Till date, the research evidence from local Pakistani studies has evaluated EAT thickness or its longitudinal changes on follow-up imaging, that highlighted a significant gap in regional cardiovascular imaging data. Nonetheless, number of international studies provided findings that align with and support our study's observations. For instance, a study from China,[14] investigated EAT volume and revealed a strong positive correlation between EAT accumulation and both the presence and severity significant CAD, shadowing EAT's role as a marker of disease burden. While, our study focused on EAT thickness as a simpler, yet equally meaningful, morphometric parameter. Irrespective of methodological differences, the direction and strength of the association observed in our findings closely align with the ones reported in volumetric analyses.

EAT volume and thickness reinforce that even linear EAT measurements obtained by cardiac MRI can play role as reliable indicators of coronary atherosclerotic burden.[15] From a clinical point of view, EAT thickness measurement is less time-consuming, thus making it a more practical and reproducible approach for routine assessments. Both parameters thereby grab similar pathophysiological processes, such as, the interplay among local visceral adiposity, vascular inflammation, and plaque development.[11]

An Egyptian comparative study,[16] used 2-D echocardiography, where EAT was significantly thicker in patients with ACS than in healthy controls, with a diagnostic cut-off value of 6.6 mm. Similar to this, the Romano *et al.*,[9] found greater EAT thickness with more severe CAD. Our study findings aligned to this, showing a strong correlation between EAT and SYNTAX score ($r=0.774$, $p<0.001$), reinforcing that higher EAT reflects more advanced, complex coronary atherosclerosis.

Moreover, the Romano *et al.*,[9] found a modest decline in EAT thickness over three years, indicated its dynamic, modifiable nature. Comparatively, our study found differences during follow-up and the degree of regression highlighted variable EAT responsiveness, reflecting its potential role as an alterable cardiovascular risk marker in clinical practice for improved management. Our three-month post-ACS follow-up showed only a small decline in EAT thickness [(0.48 mm (IQR: 0.42–0.58) to 0.45 mm (IQR: 0.40–0.53)], below our ≥10% threshold for meaningful regression. This modest change suggests EAT remodels slowly, with structural alterations likely requiring longer periods of metabolic and inflammatory stabilisation beyond the early post-ACS phase.[17,18] Romano *et al.*'s long-term EAT regression

likely reflects sustained therapy and lifestyle change, whereas our short-term results indicate that EAT is relatively resistant to rapid modification despite standard post-ACS management.

Another evidence from meta-analysis,[19] reported the prognostic significance of EAT in cardiovascular disease. Increased EAT thickness and volume have been associated with a markedly higher risk of adverse cardiac events (p<0.05) including cardiac death [OR=2.53; CI95%:1.17–5.44], MI [OR=2.63; 95%CI: 1.39–4.96;)], coronary revascularization [OR=2.99; 95%CI: 1.64–5.44)], and AF [aOR=4.04; 95%CI: 3.06–5.32)]. In our study sample, only a small number of patients showed adverse events during 3-month follow-up such as; heart failure in 4(3.9%), stroke in 3(2.9%), mortality in 3(2.9%), and revascularization in 1(1.0%). Outcomes were reported only as frequencies and percentages without risk estimation due to few events. Our lower event rate, compared with the higher odds ratios seen in Chong *et al.*, likely shadows differences in study design, follow-up duration, and patients' characteristics. Thereby, the absence of early EAT regression in our patients should not be taken as a lack of therapeutic effect but rather as an indication that short-term assessments may underestimate the capacity of EAT to serve as a modifiable risk marker. Longer follow-up intervals are likely necessary to capture the full trajectory of EAT remodelling and its potential translation into improved cardiovascular outcomes. Collectively, the prognostic relevance of EAT suggests that its short-term modulation and its impact on outcome are gradual processes rather than immediate post-ACS phenomena.[20,21]

Our findings show that greater epicardial fat thickness is strongly associated with more severe coronary artery disease, supporting its value as a simple cardiac risk marker. However, the lack of meaningful EAT reduction over three months suggests increased follow-up duration to assess true regression and its potential impact on post-ACS outcomes.

LIMITATIONS OF STUDY

This study was limited due to its short follow-up period, that may be insufficient to identify remarkable regression in EAT. EAT volume assessment that could provide more comprehensive in-depth details than thickness measurements alone, was not performed due to technical constraints. The lack of a control arm also limits the study findings. Additionally, analyses of medications that may influence EAT regression—such as SGLT2 inhibitors, GLP-1 receptor agonists, and statins—were not performed, and more detailed biochemical and inflammatory marker profiling is also lacking.

CONCLUSION

Epicardial adipose tissue thickness in post-ACS showed positive and strong correlation with the CAD severity. However, it didn't demonstrate significant regression over a follow-up of 3-month. Thus, the findings showed that short-term standard medical therapy may not induce measurable changes in EAT.

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Authors Contribution:

Following authors have made substantial contributions to the manuscript as under:

SSS & MNK: Data acquisition, data analysis, drafting the manuscript, critical review, approval of the final version to be published.

WUR & AAC: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

MWG& GA: Conception, data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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