

Epicardial Adipose Tissue Thickness and Coronary Artery Disease: Role of Cardiac Magnetic Resonance Imaging

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ABSTRACT

Objective: To determine mean epicardial adipose tissue (EAT) thickness using cardiac MRI in individuals with coronary artery disease (CAD) compared to controls.

Study Design: Comparative cross-sectional study.

Place and Duration of Study: Armed Forces Institute of Cardiology/National Institute of Heart Diseases (AFIC/NIHD), Rawalpindi, Pakistan; from July to September 2025.

Methodology: Eighty-four adults were recruited through consecutive sampling and allocated equally to CAD-Group (n = 42) and non-CAD-Group (n = 42). Greater than 50% luminal stenosis was used to identify CAD on CT coronary angiography (CTCA). Inclusion criteria included adults aged 25–55 years with either CTCA-confirmed CAD or normal coronary anatomy. Exclusion criteria comprised MRI contraindications (such as pacemakers, cochlear implants, or severe claustrophobia), nondiagnostic CMR images, prior PCI or CABG, previous valve surgery, known cardiomyopathies, eGFR <30 mL/min/1.73m², and active malignancy or recent chemotherapy or radiotherapy. All participants underwent non-contrast 3.0T cardiac MRI, and EAT thickness was measured at the left atrioventricular groove during end-diastole.

Results: Mean EAT thickness was significantly higher in CAD participants (13.24±3.32 mm) than in controls (11.49±2.15 mm) (*p*-value=0.005). Logistic regression demonstrated that each 1 mm increase in EAT thickness was independently associated with CAD (adjusted OR 1.256; 95% CI 1.061–1.487; *p*-value = 0.008).

Conclusion: EAT thickness assessed on non-contrast CMR was significantly elevated in CAD patients and independently associated with disease presence, supporting its potential as a noninvasive imaging biomarker for CAD risk assessment.

Keywords: Adipose Tissue; Coronary Artery Disease; Magnetic Resonance Imaging; Pericardium.

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INTRODUCTION

Coronary artery disease (CAD) continues to be one of the foremost contributors to morbidity and mortality worldwide, with a particularly high burden in South Asia, including Pakistan.¹ A substantial number of individuals develop CAD regardless of traditional risk factors, like hypertension, diabetes mellitus, dyslipidemia, and cigarette smoking. This highlights the need for novel biomarkers and imaging predictors that can upgrade risk assessment and early diagnosis.²

Epicardial adipose tissue (EAT), a metabolically dynamic fat deposit surrounding the myocardium beneath the visceral pericardium, has appeared as a potential surrogate marker for cardiovascular risk. EAT has numerous exocrine and endocrine effects.³ In normal physiological states, EAT supports the heart through biochemical, structural, and heat-regulating

effects.⁴ In contrast, pathologically, it can compromise coronary circulation through the release of proinflammatory cytokines, promotion of local inflammation, endothelial dysfunction, oxidative stress, and acceleration of atherosclerosis — especially in adjacent coronary vessels.⁵

There have been few local studies that have examined the association between EAT thickness in CAD patients versus healthy adults. For example, mean EAT thickness was 16.77±9.80 mm vs 14.13±4.52 mm (*p*=0.02) in the CAD group and the healthy adult group, respectively.⁶ But the method used was echocardiography. Although it is a convenient, highly reproducible, and cost-effective method, it is not accurate. Moreover, echocardiography imaging is subject to operator expertise and can be impeded in measurement in obese subjects.⁷

Cardiac MRI is considered a reference modality and provides excellent image resolution for assessing EAT thickness. Across international studies, CMR studies have shown that patients with CAD or

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atherosclerosis consistently demonstrate elevated EAT thickness or volume compared with healthy subjects. Additionally, emerging data show that higher EAT volume is associated with the severity and complexity of CAD, especially among high-risk subgroups like diabetic patients.⁸

Despite rising international attention, CMR-based research investigating EAT thickness in patients with CAD remains largely unexplored in Pakistan. The current study was designed to investigate CMR-based thickness in CAD patients versus healthy controls within a local cohort, aiming to clarify its role as a potential imaging biomarker for CAD risk.

METHODOLOGY

This study, designed as a comparative cross-sectional study, was performed at the Armed Forces Institute of Cardiology / National Institute of Heart Diseases (AFIC/NIHD), Rawalpindi, Pakistan, from July to September 2025. Ethical approval was granted by the Institutional Ethical Review Board (IERB No. 9/2/R&D/2025/369, 26 June 2025). A non-probability consecutive sampling technique was used to recruit eligible participants who visited the AFIC outpatient department and had CT angiography reports. The subjects were selected based on either normal coronary anatomy or those who have CAD characterized by >50% luminal stenosis on CT coronary angiography. CMR (non-contrast) was performed on all selected subjects. After being made aware of the goals, methods, and potential dangers of the study, each participant gave written informed consent.

Based on prior studies showing a mean EAT thickness of 13.0 ± 2.6 mm in CAD and 11.5 ± 2.1 mm in the control group, a sample size of 84 participants, split equally between groups, was determined to ensure 90% statistical power and $\alpha = 0.05$ to detect a significant difference.⁹ Sample size calculation was determined with the WHO calculator. Participants were assigned to 2 separate groups. CAD group (n=42): Subjects with diagnosed CAD, characterized by greater than 50% luminal stenosis, as determined by CT coronary angiography (CTCA), in one or more main epicardial coronary arteries. Non-CAD group (n=42): Healthy participants without both clinical and CT coronary angiography (CTCA) proven CAD.

Inclusion Criteria: Participants aged 25–55 years who underwent CT coronary angiography and were found to have either coronary artery disease (CAD) or normal coronary anatomy.

Exclusion Criteria: Contraindications to MRI (e.g., pacemakers, cochlear implants, severe claustrophobia), cardiac MRI showing artifacts, history of percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), or valvular replacement, known cardiomyopathies (e.g., hypertrophic, dilated, or restrictive cardiomyopathy), significant renal dysfunction characterized by $\text{eGFR} < 30 \text{ mL/min/1.73 m}^2$, active malignancy or recent chemotherapy/radiation (<6 months).

Participants' baseline demographic details (age, gender), anthropometric measurements (BMI), and established traditional cardiovascular risk factors (hypertension, dyslipidemia, and smoking) were recorded systematically. Laboratory investigations comprised fasting blood glucose, LDL cholesterol, high-sensitivity CRP (hs-CRP), high-sensitivity troponin I (hs-Troponin I), NT-proBNP, and creatinine to determine their metabolic and inflammatory profiles.

Based on a non-contrast cardiac MRI performed at 3.0T, this measurement was acquired using SSFP Cine sequences, such as Dixon or T1-weighted GRE. The imaging was conducted in the horizontal long-axis orientation, with the slice positioned at a basal to mid-ventricular level, approximately at the left main coronary artery or mitral valve plane.

The epicardial adipose tissue (EAT) quantification was performed at the left AV groove in the end-diastolic phase. The caliper was oriented perpendicular to the myocardial wall, extending from the epicardial surface to the pericardium. To ensure accuracy and reproducibility, it was recommended to calculate an average value from three consecutive slices. The results were reported in millimeters (mm).

Measurements were completed by two independent cardiologists, both blinded to the participants' CAD status. In cases where there was a discrepancy between observers, a third senior cardiologist provided the final judgement to ensure reliability. Additional cardiac measurements like left ventricular ejection fraction and segmental wall motion defects were also documented.

Following cardiac MRI, the epicardial fat thickness was measured in the CAD group (over 50% luminal narrowing in any major coronary artery) and the non-CAD group (normal CT angiography). CAD-RADS scoring was performed and noted to provide a descriptive characterization of coronary artery disease

(CAD). All findings were entered into a pre-designed database by trained research personnel.

All data were evaluated using IBM Statistical Package for Social Sciences version 23. Normality of records was checked by Shapiro-Wilk; normally distributed continuous variables (age, LVEF, BP) were reported as Mean $\pm$ SD whereas non-normally distributed variables (BMI) were reported as Median (IQR), with mean differences compared via independent t-test for normally distributed data and Mann-Whitney U test for non-normally distributed variables. For qualitative data, variables were expressed as frequencies and percentages; comparisons were done using the chi-square test. To identify whether EAT thickness acts as an independent risk factor for CAD, binary logistic regression was utilized, adjusting for potential confounders (age, gender, BMI, hypertension, diabetes mellitus, and dyslipidemia). Statistical significance was defined as a *p*-value less than and equal to 0.05.

RESULTS

Eighty-four participants were included, with 42(50.0%) in the CAD group and 42(50.0%) in the non-CAD group; 52(61.9%) were male, and 32(38.1%) were female. The mean age was 45.04 $\pm$ 6.31 years in the CAD group and 44.73 $\pm$ 6.86 years in the non-CAD group (*p*=0.82).

Hypertension was more frequent in participants with CAD, 29(69.0%), than in those without CAD, 8(19.0%); *p*<0.001, and diabetes mellitus was also more common in the CAD group [17 (40.5%) vs. 9 (21.4%); *p*=0.05]. Median BMI was higher in the CAD group [28.1 (24.6–29.6) vs. 26.4(23.3–27.9) kg/m²; *p*=0.01], and left ventricular ejection fraction was lower (57.07 $\pm$ 5.58 vs. 59.87 $\pm$ 4.67; *p*=0.01). Other clinical, hemodynamic, and laboratory parameters showed no statistically significant differences between the groups. The mean Epicardial Adipose Tissue (EAT) thickness in patients with Coronary Artery Disease (CAD) than in those without CAD. Patients with CAD demonstrated a higher mean EAT thickness (13.24 $\pm$ 3.32) compared to individuals without CAD (11.49 $\pm$ 2.15), indicating an increased accumulation of epicardial fat in the presence of coronary artery disease. This finding supports the potential role of EAT thickness as a marker associated with CAD. (*p*=0.05) (Table-I).

Higher Body Mass Index (BMI) in univariate logistic regression analysis was significantly linked with increased odds of coronary artery disease (OR 1.165, 95% CI: 1.012–1.341; *p*=0.034). Increased

Epicardial Adipose Tissue (EAT) thickness was also significantly associated with CAD (OR 1.256, 95% CI: 1.061–1.487; *p*=0.008). Hypertension showed a significant association with CAD (OR 0.105, 95% CI: 0.038–0.290; *p*<0.001). Additionally, Left Ventricular Ejection Fraction (LVEF) demonstrated a significant inverse association with CAD (OR 0.898, 95% CI: 0.821–0.982; *p*=0.018). Other variables, including age, diabetes, dyslipidemia, smoking status, systolic and diastolic blood pressure, fasting glucose, LDL cholesterol, creatinine, inflammatory markers (hsCRP), cardiac biomarkers (NT-proBNP and hs-troponin I), and gender, were not significantly associated with CAD in univariate analysis (all *p*>0.05).

Table-I: Comparison of Demographic and Clinical Characteristics Between CAD and without CAD Groups (n = 84)

Variable	With CAD-Group (n = 42)	Without CAD-Group (n = 42)	<i>p</i> -value
Frequency (%)			
Gender	Male	27(64.3)	0.65
	Female	15(35.7)	
Hypertension	29(69.0)	8(19.0)	<0.001
Diabetes mellitus	17(40.5)	9(21.4)	0.05
Dyslipidemia	21(50.0)	19(45.2)	0.66
Smoking	15(35.7)	9(21.4)	0.14
Wall motion abnormality	30(71.4)	42(100)	<0.001
Mean$\pm$SD			
Age (years)	45.04 $\pm$ 6.31	44.73 $\pm$ 6.86	0.82
Systolic BP (mmHg)	129.19 $\pm$ 15.56	127.07 $\pm$ 14.04	0.51
Diastolic BP (mmHg)	80.17 $\pm$ 9.84	80.43 $\pm$ 11.00	0.90
Fasting glucose (mg/dL)	112.82 $\pm$ 33.87	112.89 $\pm$ 27.10	0.99
LDL-C (mg/dL)	126.97 $\pm$ 38.51	126.67 $\pm$ 26.73	0.96
Creatinine (mg/dL)	0.93 $\pm$ 0.20	0.98 $\pm$ 0.19	0.25
hs-CRP (mg/L)	3.61 $\pm$ 1.14	3.52 $\pm$ 1.22	0.71
NT-proBNP (pg/mL)	195.25 $\pm$ 65.32	183.48 $\pm$ 73.60	0.44
hs-Troponin I (ng/L)	12.41 $\pm$ 6.05	13.83 $\pm$ 5.45	0.26
LVEF (%)	57.07 $\pm$ 5.58	59.87 $\pm$ 4.67	0.01
Mean Epicardial Adipose Tissue (EAT)	13.24 $\pm$ 3.32	11.49 $\pm$ 2.15	0.05
Median (IQR)			
BMI (kg/m ²)	28.1 (24.629.6)	26.4 (23.327.9)	0.01

CAD= Coronary Artery Disease; BP=Blood Pressure; LDL-C=Low-Density Lipoprotein Cholesterol; Hs-CRP= High-Sensitivity C-Reactive Protein; LVEF= Left Ventricular Ejection Fraction; BMI= Body Mass Index; EAT= Epicardial Adipose Tissue; NT-ProBNP=N-Terminal Pro-B-Type Natriuretic Peptide

In multivariable logistic regression analysis, BMI (adjusted OR 1.231, 95% CI: 1.038–1.459; *p*=0.017) and EAT thickness (adjusted OR 1.344, 95% CI: 1.065–1.698; *p*=0.013) remained independently associated with higher odds of CAD. Hypertension also remained a significant independent predictor (adjusted OR 0.077, 95% CI: 0.023–0.262; *p*<0.001).

Other variables, including age, metabolic parameters, inflammatory and cardiac biomarkers, LVEF, and gender, did not retain statistical significance after adjustment, as mentioned in Table-II.

dominant in the CAD group, they did not maintain independent significance in multivariate analysis.⁵ This finding emphasizes the limitations of global cardiovascular risk assessment tools and highlights

Table-II: Predictors and Associated Factors of Coronary Artery Disease (n= 84)

Variables	Study Parameter		Univariate logistic regression		Multivariate logistic regression	
	Mean±SD	p-value	Unadjusted OR (95% CI)	p-Value	Adjusted OR (95% CI)	p-value
Age (years)	45.04 ± 6.31	0.82	1.007 (0.943–1.076)	0.826	-	-
Systolic BP (mmHg)	129.19 ± 15.56	0.51	1.010 (0.981–1.040)	0.509	-	-
Fasting Glucose (mg/dL)	112.82 ± 33.87	0.99	1.000 (0.986–1.014)	0.991	-	-
Diastolic BP (mmHg)	80.17 ± 9.84	0.90	0.998 (0.957–1.040)	0.907	-	-
Creatinine (mg/dL)	0.93 ± 0.20	0.25	0.266 (0.028–2.568)	0.252	-	-
LDL (mg/dL)	126.97 ± 38.51	0.96	1.000 (0.987–1.013)	0.966	-	-
hsCRP (mg/L)	3.61 ± 1.14	0.71	1.071 (0.742–1.547)	0.714	-	-
NTproBNP (pg/mL)	195.25 ± 65.32	0.44	1.002 (0.996–1.009)	0.436	-	-
hsTroponin I (ng/L)	12.41 ± 6.05	0.26	0.957 (0.887–1.033)	0.262	-	-
EAT Thickness (mm)	13.24±3.32	0.05	1.256 (1.061–1.487)	0.008*	1.344 (1.065–1.698)	0.013*
Median(IQR)						
BMI (kg/m ²)	28.1 (24.6–29.6)	0.01	1.165 (1.012–1.341)	0.034*	1.231 (1.038–1.459)	0.017*
Frequency (%)						
Hypertension	29 (69.0)	<0.001	0.105 (0.038–0.290)	0.000*	0.077 (0.023–0.262)	<0.001*
Diabetes	17 (40.5)	0.05	0.401 (0.153–1.048)	0.062	-	-
Dyslipidemia	21 (50.0)	0.66	0.826 (0.350–1.947)	0.662	-	-
Smoking	15 (35.7)	0.14	0.491 (0.186–1.295)	0.151	-	-
LVEF (%)	57.07 ± 5.58	0.01	0.898 (0.821–0.982)	0.018*	0.897(0.798–1.008)	0.068
Gender (male)	27 (64.3)	0.65	0.817 (0.338–1.974)	0.653	-	-

BMI = Body Mass Index; BP = Blood Pressure; LDL = Low-Density Lipoprotein; hsCRP = High-Sensitivity C-Reactive Protein; NT-proBNP = N-Terminal pro-B-Type Natriuretic Peptide; hs-Troponin I = High-Sensitivity Troponin I; EAT = Epicardial Adipose Tissue; LVEF = Left Ventricular Ejection Fraction

DISCUSSION

This study demonstrates a significantly increased epicardial adipose tissue (EAT) thickness among patients with Coronary Artery Disease (CAD) related to those without CAD in a Pakistani cohort. Our findings showed a mean EAT thickness of 13.24 ± 3.32 mm in CAD patients versus 11.49±2.15 mm in non-CAD individuals ($p=0.05$), reinforcing EAT thickness as a strong, independent predictor of CAD even after adjustment for traditional risk factors. This supports the potential utility of EAT measurement by cardiac magnetic resonance imaging (CMR) as a non-invasive biomarker for CAD risk stratification in the local population.^{10,11}

These results align with international data, including the meta-analysis by Wang *et al.*, which reported significantly higher EAT volume and thickness in CAD patients versus controls across diverse populations ($p<0.001$). Their subgroup analysis confirmed a graded association between EAT burden and CAD severity, consistent with our observation of increased EAT thickness correlating with CAD presence.¹² This suggests a pathophysiological continuum where EAT contributes to the progression of atherosclerosis and coronary artery stenosis.⁷

Although conventional risk factors such as diabetes mellitus and dyslipidemia were more

the added value of regional visceral adiposity assessment. Our data suggest that EAT thickness may reflect localized metabolic and inflammatory processes that are not captured by systemic risk factors or traditional anthropometric measures such as BMI.²

Mechanistically, our findings resonate with the evidence from Guglielmo *et al.*, (2021), who described EAT as a metabolically active tissue producing pro-inflammatory cytokines with paracrine effects on coronary arteries. This pericoronary adipose tissue (PCAT) modulates endothelial function, plaque formation, and local inflammation, particularly in metabolic dysregulation. These insights provide biological plausibility for our independent association between increased EAT thickness and CAD, implicating EAT not only as a biomarker but also as a potential mediator of coronary atherosclerosis.¹³

Earlier work by Iacobellis *et al.*, (2019) also supports this concept, demonstrating correlations between epicardial fat, coronary plaque burden, and metabolic syndrome components.¹⁴ Further, recent reviews underscore the role of dysfunctional EAT in promoting atherosclerosis via secretion of inflammatory adipokines such as IL-6 and TNF- α , advancing myocardial remodeling and endothelial injury. These pathophysiological links validate the

clinical relevance of EAT quantification for early CAD detection before symptomatic disease manifests.¹⁵

Findings from acute chest pain cohorts, including the EPIC-ACS study (2023), reinforce the predictive power of EAT thickness, showing a nearly twofold increase in odds of obstructive CAD per millimeter increase in EAT (OR 1.87, $p < 0.0001$). Importantly, the addition of EAT thickness to established risk models significantly improved their diagnostic accuracy. Our study's parallel results support incorporating EAT thickness measurement into standard risk assessment protocols to enhance CAD prediction beyond traditional factors.

Functional cardiac parameters further differentiated CAD patients, with lower left ventricular ejection fraction (LVEF) observed in the CAD group ($57.07 \pm 5.58\%$ vs. $59.87 \pm 4.67\%$; $p = 0.01$), suggesting subclinical myocardial impairment linked to epicardial fat accumulation. This finding underscores the relationship between EAT and adverse myocardial remodeling, potentially mediated by inflammatory and metabolic stress.

EAT, anatomically situated between the myocardium and visceral pericardium, serves physiological functions including myocardial protection, thermoregulation, and energy provision.¹⁶ However, under pathological states such as obesity, diabetes, or metabolic syndrome, EAT becomes dysfunctional, secreting pro-inflammatory adipokines that facilitate local inflammation, endothelial dysfunction, and atherogenesis as reported by Sun *et al.*¹⁷ Our findings of increased EAT thickness in CAD patients are consistent with this model, suggesting that EAT contributes both as a marker and as a mechanistic factor in coronary artery disease development.

Yang *et al.*, describes that by incorporating EAT thickness measurement into routine CMR cardiac imaging, especially in high-risk populations, could improve early identification of individuals at elevated risk for CAD before clinical symptoms arise. CMR offers a safe, reproducible alternative to CT-based fat quantification, particularly valuable for patients with renal impairment or requiring serial monitoring.¹⁸

EAT's metabolic activity and interventions aimed at reducing EAT—through lifestyle modification or pharmacotherapy—may confer cardiovascular benefits. Prospective longitudinal studies are needed to evaluate if EAT reduction translates into decreased CAD incidence or slowed progression. With rising

CAD prevalence in Pakistan, routine assessment of EAT may enhance risk prediction and inform tailored prevention strategies suited to local population dynamics.

LIMITATIONS OF STUDY

The single-center, cross-sectional design limits generalizability and precludes causal inference. The sample size was sufficient for primary analysis but relatively modest, potentially limiting analytical depth. Measurement was restricted to the left atrioventricular groove, potentially underrepresenting total epicardial fat volume or regional heterogeneity, such as perivascular fat.

CONCLUSION

Epicardial adipose tissue thickness, assessed using non-contrast 3.0T CMR, is significantly greater in individuals with CAD than in healthy controls, and this parameter independently serves as a predictor for CAD. These findings indicate that EAT thickness could serve as a convenient, non-invasive imaging marker for stratifying CAD risk. EAT quantification as part of routine cardiac imaging could improve early detection, risk prediction, and targeted prevention, particularly in Pakistani populations with elevated CAD risk.

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Authors' Contribution

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

AA & AA: Data acquisition, data analysis, critical review, approval of the final version to be published.

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

MA: 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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