Original Article

Relation of Epicardial Fat Thickness by Multi-Detector Computed Tomography Scan and Coronary Artery Disease

Den Prasad Acharya[1], Arun Maskey[2], Sanjay Shrestha[3*], Kunjang Sherpa[3], Kijan Maharjan[3], Sudip Lamsal[3], Nirmal Ghimire[4], Binod Dangol[3], Abhishek Tiwari[3], Anupam Bista[5]

[1]*Department of Internal Medicine , Koshi Hospital, Biratnagar 56613, Nepal*
[2]*Department of Cardiology, Shahid Gangalal National Heart Center, Kathmandu 44600, Nepal*
[3]*Department of Cardiology, National Academy of Medical Sciences, Bir Hospital, Kathmandu 44600, Nepal*
[4]*Department of Internal Medicine, Nepal Police Hospital, Maharajgunj, Kathmandu 44600, Nepal*
[5]*Department of Cardiology, Bharatpur Hospital, Bharatpur, Chitwan 44200, Nepal*

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Abstract

Background: Coronary artery disease is one of the major causes of morbidity and mortality in the world as well as in Nepal and are associated with several risk factors and predictors. Epicardial fat thickness (EFT) is also a predictor of obstructive coronary artery disease based on various studies done elsewhere. Epicardial fat, the adipose tissue located between the visceral pericardium and myocardium of the heart serves as both pro-inflammatory and anti-inflammatory roles in various physiological and pathological states. Measurement of EFT and its analysis may help to predict the likelihood of obstructive coronary artery disease and help to use preventive measures to prevent or delay the occurrence of obstructive coronary artery disease.

Aims: This study was done to assess relation of epicardial fat thickness by multi-detector computed tomography scan with coronary artery disease and its severity.

Methods: This was a hospital based observational study conducted in Shahid Gangalal National Heart Centre and total 524 patients undergoing MDCT coronary angiography were included from Jun 21, 2024 to Jun 20, 2025 (12 months). 320 slice MD CT coronary angiography was done using standard protocol. Reconstruction was done and epicardial fat thickness was measured at basal short axis view in RV at 25%,50% and 75% level at RV free wall at 75% of RR interval. Mean of three measurement was taken as mean epicardial fat thickness. Patients were grouped into 4 groups based on the luminal stenosis: no CAD, Mild , Moderate and Severe stenosis, with no stenosis, luminal stenosis < 50%, 50-70% >= 70% stenosis in any or all 3 major coronary arteries respectively. Luminal stenosis of 50% or greater taken as obstructive CAD.

Results: Among 524 patients undergoing CT coronary angiography, 81 (15.5%) had obstructive coronary artery disease (CAD), while 443 (84.5%) had no obstructive disease. The mean epicardial fat thickness (EFT) was 2.75 ± 0.72 mm. Patients with obstructive CAD had higher mean EFT compared to those without disease (3.9 ± 0.6 vs. 2.5 ± 0.5 mm, $p < 0.001$). EFT progressively increased with disease severity: 2.33 mm in normal coronaries, 3.17 mm in mild CAD, 3.80 mm in moderate CAD, and 4.06 mm in severe CAD ($p < 0.001$). Similarly, EFT rose with vessel involvement, from 3.7 mm in single-vessel disease to 4.4 mm in triple-vessel disease ($p < 0.001$).ROC curve analysis showed EFT of 3.15 mm as optimal cut-off for predicting obstructive CAD, with an AUC of 0.969, sensitivity 96.3%, and specificity 86.9%. On multivariate regression, EFT ≥3.15 mm was the strongest independent predictor of CAD (AOR 91.3; 95% CI 23.3–357.0, $p < 0.001$), followed by higher coronary artery calcium score (CACS ≥400: AOR 7.08, $p = 0.012$). EFT demonstrated a moderate positive correlation with CACS ($r = 0.566$, $p < 0.001$).

Conclusion: Higher epicardial fat thickness measured by multidetector CT in patients undergoing CT coronary angiography was strongly associated with obstructive coronary artery disease.

1. Introduction

Cardiovascular disease (CVD) is the commonest cause of mortality and morbidity worldwide. Among CVDs, Coronary artery disease (CAD) is the most common cause of mortality and loss of disability adjusted life years globally accounting for 9.44 million deaths in years 2021 with 185 million losses of DALYs annually.[1] In Southeast Asia, CVD is the leading cause of deaths which accounts for the 35% of the total deaths of which 47% is attributable to the coronary artery disease.[2] Nepal is in the first or early stage of CVD epidemic with prevalence of coronary artery disease ranging from 2.9 % to 5.7% in various studies. Coronary artery disease is the most common CVDs in Nepal and it is the leading cause of CVD deaths.[2,3]

There are various predictors of coronary artery disease. Traditional risk factors includes age, sex, family history, smoking, dyslipidemia, obesity, hypertension, diabetes mellitus, physical activity, diet and stress etc.[4] Other predictor include Inflammatory biomarkers like high sensitive C-reactive protein (hs-CRP), Lipoprotein(a), Matrix Metalloproteinases-3 and 9 (MMP-3 & MMP-9), Tissue inhibitors of matrix metalloproteinase-1 and 2 (TIMP-1 and TIMP-2), Apo-CII and Apo-CIII etc.[4,5] Similarly, coronary calcium score and epicardial fat pad thickness and volume measured by Multi-detector computed tomography (MDCT) are other noninvasive markers predictive of coronary artery disease and its severity.[4,6,7]

Corresponding Author:

Dr. Sanjay Shrestha

Department of Cardiology, National Academy of Medical Sciences, Kathmandu 44600, Nepal,
Email: shrestha834@gmail.com
Phone: +977-9851214949

Epicardial fat is visceral adipose tissue located between visceral pericardium and myocardium[7]. Epicardial fat has evolved from brown adipose tissue.[8] It has variable distribution pattern being most prominent in lateral wall of right ventricle, atrioventricular groove and interventricular groove.[6] Epicardial fat is anatomically and functionally contiguous with myocardium and coronary arteries and has unique and complex physiological functions.[9] The epicardial fat similar to the brown adipose tissue helps in thermogenesis to provide heat to the myocardium during cold or ischemia and also acts mechanically to protect coronary arteries against torsion induced by arterial pulse wave and also permits expansion and positive remodeling of the coronary arteries.[9,10]

The epicardial fat is cardioprotective in normal physiological condition with secretion of anti-inflammatory cytokines and non-atherogenic vasoactive substances such as adiponectin and adrenomedullin which act to protect coronaries from atherogenesis.[11,12] In pathological states, the excess epicardial fat secretes pro-inflammatory cytokines such as IL-1β, IL-6, tumor necrosis factors into the adjacent myocardium and coronary artery bloodstream.[13] Whether increase in cytokines in disease states like CAD or heart failure is cause or effect is controversial.

Various studies of epicardial fat thickness measured by 2 D echocardiography or MDCT has demonstrated association with coronary artery disease severity.[14-16] MDCT is non invasive and is more accurate in quantifying epicardial fat because of its high spatial resolution.[17] There is no study of epicardial fat thickness measured by MDCT and its relation with coronary artery disease in Nepal . Therefore, measurement of epicardial fat thickness while doing CT coronary angiography will be helpful for the evaluation of relation of epicardial fat thickness with coronary artery disease and its severity without added cost to the participants.

2. Materials and Methods

2.1 Study Design and Participants

This was a Hospital based, observational study done at Shahid Gangalal National Heart Centre, Kathmandu between Jun 21, 2024 to Jun 20, 2025 (12 months). Total of 524 patients were recruited fulfilling the inclusion and exclusion criteria.

Inclusion Criteria

1) Age >= 18 years
2) All patients suspected of coronary artery disease (Chest pain or shortness of breath on exertion) undergoing computed tomography coronary angiography (CTCAG)

Exclusion Criteria

1) Patients with previous history of allergy to contrast material
2) Patient with renal impairment with eGFR < 30ml/min/m2
3) Patient with previous history of Coronary Artery Bypass Grafts (CABG) surgery or angioplasty
4) Previous history of coronary artery disease
5) Patient in Atrial fibrillation (AF)
6) Patient with pulmonary edema
7) Pregnancy

2.2. Sample size Calculation

The Sample size was calculated by the formula,

$n = z^2 \text{ Sen X } (1- \text{Sen}) / d^2 \text{ X P}$ and

$n = z^2 \text{ Spe X } (1-\text{Spe}) / d^2 \text{ X P}$

Where,

n =required sample size

P =Prevalence of coronary artery disease

d =10 % of maximum tolerable error

Sen= Sensitivity

Spe = Specificity

z=1.28 at 90 % confidence interval

In a study by Maskey et.al, the prevalence of coronary artery disease in Nepal is 5%.[2] In a study done by Bacher et al[14] sensitivity and specificity of Epicardial fat thickness > 2.4 mm by MDCT in predicting obstructive coronary artery disease are 80 % and 73% respectively. So, the sample size for this study at 5% prevalence of coronary artery disease with 10 % maximum tolerable error and 90% confidence interval was 524.

2.3. Sampling technique

Subjects admitted and attended outpatient department in Shahid Gangalal National Heart Centre were included in the study after obtaining informed consent.

2.4. Data Collection

Data was collected using a structured proforma covering the relevant details. Patients fulfilling the inclusion criteria were explained about the nature of the study and informed written consent was obtained from those willing to get enrolled.

2.5. Procedural Details

Patients admitted in medical ward or visiting Out Patient Department (OPD) of Shahid Gangalal National Heart Centre for the evaluation of suspected coronary artery disease and were planned for CT coronary angiography were evaluated.

All patients underwent detailed medical history, clinical examination and relevant investigations. Review of previous investigations and treatment including ECG, Echocardiography, TMT, previous cardiac surgery was done. Necessary laboratory investigations including serum creatinine, hemoglobin, lipid profile, ECG and Echocardiography were sent and reports were collected. Radiological examination was done using a 320-slice CT scanner (Aquilion one system, Toshiba Medical Systems, Tokyo, Japan). Multislice CT coronary angiography was done for all the patients using standard protocol for image acquisition and reconstruction, and image analysis.Image analysis:

Coronary calcium is defined as an area of at least three "face-connected" voxels in the axial plane in the course of a coronary artery, with an attenuation threshold value of 130 HU or greater. Non-contrast CT scan of the heart was done with configuration of 3mm slice for 20 contiguous slices. Each image was triggered at 80% of RR interval so that slices were obtained at same point during diastole during a single breath hold. To calculate Coronary calcium score, each of 20 slices were evaluated sequentially. An area of > $1mm^2$ with an attenuation threshold of 130 HU or greater was taken as calcium containing lesion. Automated measurement of lesion area and maximal computed tomographic number of each region of interest was recoded. A lesion score calculated based on CT numbers as follows; 1:130-199,2:200-299,3:300-399,4: ≥400). Score at each area of interest was calculated by multiplying density score and the area. Total coronary calcium score was determined by adding the scores at each of 20 slices. Coronary calcium score was calculated and expressed in Agatston's score.[35]

Epicardial fat was determined by CT attenuation threshold of -190 to -30 HU unit[36]. Epicardial fat thickness was measured on the right

ventricular anterior free wall. Measurements were done at the base of the ventricles (basal level) on short axis view. Three measurements of epicardial fat thickness: inferior, central and superior, chosen at 25%, 50% and 75% level of the RV wall, respectively, from the visceral epicardium to the outside of the myocardium and perpendicular to the surface of the heart were taken. The mean of the three measurements was used for analyses.

Coronary arteries were evaluated for presence of CAD. Coronary artery stenosis was graded as:

None (No CAD): 0% luminal stenosis

Mild CAD: 1-49 % stenosis

Moderate CAD: 50-69% stenosis

Severe CAD: ≥ 70% luminal stenosis[37]

Vessel score was calculated. Vessel score denotes the number of vessels with significant stenosis of 50% or greater reduction in luminal diameter. The score ranges from 0 to 3, depending on the number of vessels involved. LMCA is scored as double vessel disease[38].

Vessel score:

0 = no vessel involvement

1 = single vessel involvement (SVD)

2 = double vessel involvement (DVD)

3 = triple vessel involvement (TVD)

Obstructive coronary artery disease was defined by luminal diameter reduction of 50 % or more.

2.6. Statistical Analysis

All data were entered into an electronic spreadsheet (Microsoft Excel, Redmond) and the statistical analysis were done using the SPSS version 23 software. Appropriate statistical tests were carried out to analyze the data. All Categorical variables were expressed in frequency and percentage. All numerical data were presented in mean ± SD, if data were found to be normal otherwise median (Q1, Q3) were assessed. Student t test was applied to determine mean difference between EFT in patients with normal coronaries with obstructive CAD. ANOVA was used to compare mean epicardial thickness among various severity of coronary artery disease and also for various severity of coronary artery calcium scores. ROC was drawn to determine optimal EFT for predicting obstructive CAD and Sensitivity and specificity of epicardial fat pad thickness was determined. For the purpose of this study a 95% confidence interval was accepted. Multivariate regression analysis was done to evaluate the role of epicardial fat thickness in obstructive coronary artery disease.

2.7. Ethics

Ethical approval was obtained from Institutional Review Board at National Academy of Medical Sciences and Shahid Gangalal National Heart Centre before the start of data collection.

3. Results

3.1. Baseline Characteristics

In this study, out of 524 patients, most patients (84.5%, n = 443) had no obstructive coronary artery disease (CAD) on CT coronary angiography, while 15.5% (n = 81) had obstructive CAD. Patients in the CAD group were significantly older (61 ± 10 years) than those without CAD (53 ± 12 years) ($p < 0.001$). Men were more common in the CAD group (59%) compared to the no-CAD group (46%), a difference that was statistically significant ($p = 0.03$). Baseline characteristics in patients with or without obstructive coronary artery disease on CT coronary artery angiography is shown in Table 1.

Table 1: Baseline characteristics in patients with or without obstructive coronary artery disease on CT coronary artery angiography

Variables	No obstructive CAD (n=443, 84.5%)	Obstructive CAD (n=81, 15.5%)	p value
Age, years, mean ± SD	53±12	61±10	<0.001
Female, n (%) Male, n (%)	242 (54%) 201(46%)	33 (41%) 48 (59%)	0.03
Body mass index, kg/m^2, mean± SD	25.1±3.17	25±3.17	0.88
Dyslipidemia	45 (10%)	20 (24%)	<0.001
Hypertension	145 (33%)	40 (49%)	0.01
Diabetes Mellitus	58 (13%)	25 (31%)	<0.001
Family h/o CAD	5 (1%)	1 (1.2%)	0.85
Smoking	51 (12%)	13(16%)	0.36

3.2. Epicardial fat thickness (EFT) Distribution

The mean epicardial fat thickness (EFT) was 2.75 ± 0.715 mm. The distribution is slightly positively skewed, with the majority of measurements clustering between 2.0 mm and 3.0 mm. The highest frequency was observed just above 2.0 mm, while fewer observations extended toward the higher end of the range (up to approximately 6 mm) shown in figure 1.

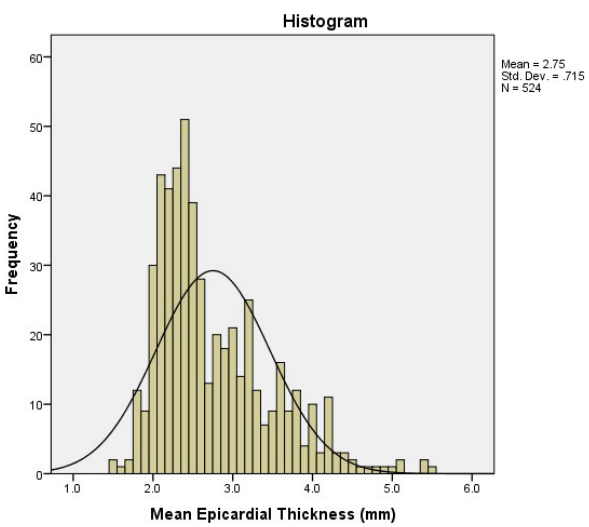

Figure 1: Distribution of EFT among patients undergoing CTCAG

Patients with obstructive CAD had significantly higher mean EFT (3.9 ± 0.6 mm) compared to those without obstructive CAD (2.5 ± 0.5 mm, $p < 0.001$) .

3.3. Distribution of Epicardial Fat thickness with CAD severity

Mean epicardial fat thickness were 2.33±0.3 mm, 3.17±0.4 mm, 3.80±0.4 mm and 4.06±0.5 mm respectively among normal coronaries, mild CAD, moderate CAD and severe CAD with intergroup P value <0.001 .(Table 2)

Table 2. Distribution of Epicardial Fat thickness with CAD severity

CAD Severity	N (%)	Mean Epicardial fat thickness (Mean ±SD)	P value
No CAD	337(65)	2.33±0.3 mm	<0.001
Mild CAD	106(20)	3.17±0.4 mm	
Moderate CAD	39(7)	3.80±0.4 mm	
Severe CAD	42(8)	4.06±0.5 mm	
Total	524 (100%)	2.75 ± 0.715 mm	

3.4. Distribution of epicardial fat thickness with Vessel involvement

Among 81 patients who had obstructive CAD, 46 patients (57%) had single vessel disease (SVD), 22 patients (27%) had double vessel disease (DVD) and 13 patients (16%) had triple vessel disease (TVD). Mean epicardial fat thickness showed graded increments as the number of vessels involved increased. The mean EFT among SVD, DVD and TVD were 3.7±0.4 mm, 4.0±0.5 mm and 4.4±0.6 mm respectively with p value <0.001.

3.5. Receiver Operating Curve

The ROC curve analysis revealed that a mean epicardial fat thickness threshold of 3.15 mm optimally predicted the presence of obstructive coronary artery disease with sensitivity of 96.3% and specificity of 86.9%, corresponding to the highest Youden's Index value of 0.832. The model showed excellent discriminative ability, with an area under the curve of 0.969 (95% CI: 0.95–0.98, p < 0.001). (Figure 2)

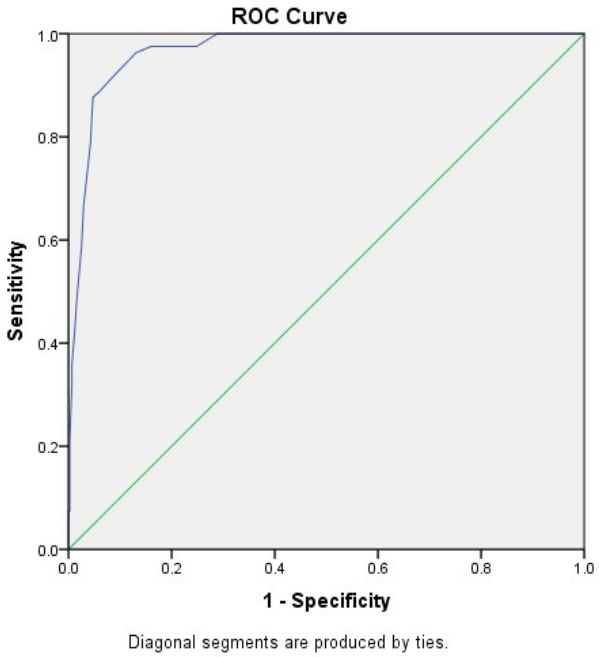

Figure 2: ROC curve for EFT predicting obstructive CAD

(AUC: 0.969, CI: 0.95-0.98, P value <0.001)

3.6. Bivariate Analysis

Bivariate analysis of factors associated with obstructive CAD showed that older age, male sex, hypertension, diabetes, dyslipidemia, higher epicardial fat thickness, and elevated coronary calcium scores are significantly associated with obstructive CAD in this cohort.(Table 3)

Table 3: Bivariate analysis of risk factors for obstructive CAD

Variables	Categories	Obstructive CAD		Total, n (%)	p-value
		No, n (%)	Yes, n (%)		
Age (years)	Mean± SD	53.3± 12.4	61.3± 10.5	60 ± 10	<0.001*
Sex	Female	242 (88%)	33 (12%)	275(100%)	0.02
	Male	201 (81%)	48(19%)	249 (100%)	
Hypertension	No	298 (88%)	41 (12%)	339 (100%)	0.004
	Yes	145 (79%)	40 (21%)	185 (100%)	
Diabetes	No	385(87%)	56(13%)	441 (100%)	<0.001
	Yes	58(70%)	25 (30%)	83 (100%)	
Family H/o CAD	No	438 (85%)	80 (15%)	518 (100%)	0.9 **
	Yes	1 (17%)	5 (83%)	6 (100%)	
Smoking	No	392 (85%)	68 (15%)	460 (100%)	0.25
	Yes	51 (80%)	13 (20%)	64 (100%)	
Obesity	No	403 (84%)	75 (16%)	478 (100%)	0.63
	Yes	40 (87%)	6(13%)	46 (100%)	
Dyslipidemia	No	398 (87%)	61 (13%)	459 (100%)	< 0.001
	Yes	45 (69%)	20 (30%)	65 (100%)	

EFT ≥ 3.15	No	385 (99%)	3 (1%)	388 (100%)	<0.001
	Yes	58(43%)	78 (57%)	136 (100%)	
CACS	0	338 (97%)	9(3%)	347 (100%)	<0.001**
	1-99	81 (73%)	30 (27%)	111 (100%)	
	100-399	18 (41%)	26 (59%)	44 (100%)	
	≥400	6 (27%)	16 (73%)	22 (100%)	

- * Independent T test applied
- **Fisher's exact test applied

3.7. Multivariate logistic regression analysis of factors associated with obstructive CAD

Epicardial Fat Thickness (EFT ≥3.15 mm) and higher Coronary Artery Calcium Scores (CACS ≥100) as independent predictors of obstructive coronary artery diseases. Participants with EFT ≥3.15 mm had 91.3-fold higher adjusted odds (95% CI: 23.3–357.0, $p < 0.001$) compared to the EFT< 3.15mm. A graded association was observed for CACS, with the strongest risk in the ≥400 subgroup (AOR = 7.08, 95% CI: 1.55–32.42, $p = 0.012$). Traditional risk factors (age, sex, hypertension, diabetes, dyslipidemia) were not statistically significant.(Table 4)

Table 4: Multivariable Logistic Regression analysis of factors associated with obstructive CAD

Variables	Categories	Beta	Adjusted Odds Ratio (AOR)	95% CI	p-value
Epicardial fat thickness (mm)	<3.15		-		<0.001
	≥ 3.15	4.51	91.3	23.3-357.0	
Age, in years		0.008	0.992	0.95-1.02	0.631
Sex	Female		-		
	Male	0.144	1.15	0.57-2.30	0.683
Hypertension	No		-		
	Yes	0.322	1.381	0.68-2.76	0.364
Diabetes	No		-		
	Yes	0.294	0.745	0.33-1.65	0.470
Dyslipidemia	No		-		
	Yes	0.148	1.160	0.46-2.91	0.752
CACS (AU)	0		-		
	1-99	0.532	1.73	0.57-5.003	0.3
	100-399	1.247	3.48	1.07-11.24	0.037
	≥ 400	1.957	7.08	1.54-32.42	0.012

3.8. CT coronary artery calcium score & epicardial fat thickness

The study showed a moderate positive correlation between coronary artery calcium (CAC) score, expressed in Agatston Units (AU), and mean epicardial fat thickness (EFT) measured on CT. The Pearson correlation coefficient ($r = 0.566$) indicates that as EFT increases, the CAC score also tends to rise, reflecting a direct relationship between these two parameters. The correlation is statistically significant ($p < 0.001$) as shown in Figure 3.

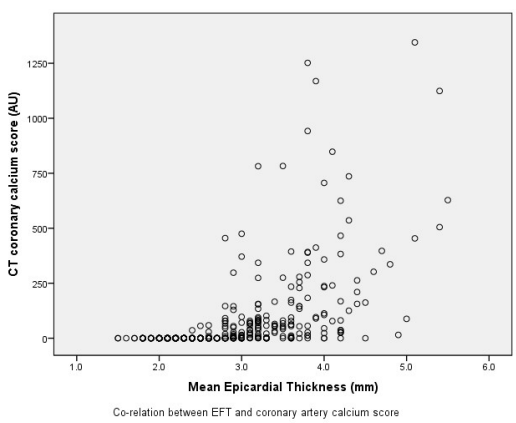

Figure 3: Co-relation between CT coronary calcium score (AU) and mean EFT (mm) (Pearson's co-relation coefficient r= 0.566, $p < 0.001$)

The study revealed mean epicardial fat thickness across ascending coronary calcium categories. Patients with zero calcium had the lowest EFT (2.37 ± 0.40 mm), while those with severe calcification (CACS ≥ 400) demonstrated the highest values (4.10 ± 0.78 mm). The differences among groups were statistically significant (ANOVA, $p < 0.001$). These findings indicate that higher EFT is strongly associated with increasing coronary atherosclerotic burden, as reflected by CT calcium scoring. (Table 5)

Table 5: Mean EFT (mm) in different CT coronary calcium score (CACS) category

CACS (AU)	Mean EFT (mm)	N (%)	Std. Dev.	P value
CACS:0	2.377	347 (66.2)	0.403	
CACS:1-99	3.266	111(21.2)	0.490	
CACS:100-399	3.736	44 (8.4%)	.5181	<0.001
CACS: ≥ 400	4.095	22 (4.2%)	.7847	
Total	2.752	524 (100%)	.7153	

Discussion

In this study of 524 patients undergoing CT coronary angiography, epicardial fat thickness (EFT) and coronary artery calcium score (CACS) were found to be strongly associated with the presence and severity of obstructive coronary artery disease (CAD). These findings support the growing body of evidence suggesting that epicardial adiposity, as measured non-invasively by CT, is not only a marker of metabolic risk but also an independent predictor of coronary atherosclerosis.

Patients with obstructive CAD having significantly higher values of EFT compared to those without disease. Moreover, there is a graded relationship between EFT and severity of CAD, suggesting a dose–response relationship between EFT and atherosclerotic burden. The association was further confirmed when stratified by vessel score, where EFT progressively increased from single to triple vessel disease.

ROC curve analysis demonstrated that an EFT threshold of 3.15 mm optimally predicted obstructive CAD with excellent discriminative accuracy (AUC 0.969, sensitivity 96.3%, specificity 86.9%). Importantly, EFT ≥3.15 mm remained an independent predictor of CAD in multivariate analysis (AOR 91.3, CI 23.3–357.0, $p < 0.001$), outperforming traditional risk factors such as age, hypertension, diabetes, and dyslipidemia. These findings highlight EFT as a robust imaging biomarker for coronary risk stratification.

Our results are consistent with prior studies. Bachar et al. evaluated 190 asymptomatic patients using 64-slice MDCT and reported that EFT was significantly higher in patients with obstructive CAD.[14] They identified an optimal EFT cutoff of 2.4 mm with sensitivity of 80% and specificity of 73% for predicting significant stenosis, with EFT >2.4 mm being an independent predictor of CAD (OR 10.8, CI 4.9–24.8, $p < 0.001$). Although their cutoff was lower than ours (2.4 mm vs. 3.15 mm), the overall trend remains concordant, supporting the utility of EFT as a reliable predictor of CAD across different populations.

Samy et al. reported a significantly higher optimal EFT threshold of 7.2 mm (sensitivity 67%, specificity 67.4%) for predicting obstructive CAD, measured at the right ventricular free wall using 256-slice MDCT.[18] Similarly, Gać et al. identified an EFT cut-off ≥ 16.7 mm as highly sensitive (98.4%) for detecting high-risk CAD based on calcium scoring, while a value ≤ 8.7 mm was most specific for excluding CAD.[19] Taha et al. evaluated epicardial fat volume (EFV) instead of thickness, reporting optimal thresholds of 124 ml and significant correlations with CAD severity.[20]

Maimaituxun et al found that local peri-coronary fat thickness surrounding the LAD was significantly greater in patients with CAD (6.86 ± 2.19 mm vs. 5.45 ± 2.16 mm, $p < 0.001$), while no significant association was observed with RCA or LCX fat thickness or total fat volume.[21] Furthermore, LAD-specific EAT thickness correlated with CAD severity ($r = 0.239$) and improved predictive performance when combined with the Framingham Risk Score. Compared with our findings, which used a global mean EFT measurement along right ventricular free wall, their study underscores the pathophysiological relevance of regional fat depots. Nonetheless, both approaches demonstrate that excess epicardial fat exerts a measurable influence on coronary atherosclerosis and could be integrated into risk prediction models.

Moreover, mean EFT also increased progressively with higher calcium categories, demonstrating a moderate positive correlation . In line with established epidemiology, we found that older age, male sex, hypertension, diabetes, and dyslipidemia were significantly associated with obstructive CAD in Bivariate analysis. However, in multivariate models, these factors lost statistical significance once EFT and CACS were included. This finding suggests that imaging biomarkers, which directly reflect underlying atherosclerotic activity, may provide stronger predictive value compared to conventional risk factors, especially in populations with multiple overlapping comorbidities.

Table no 6 shows comparison of various studies which showed relation of epicardial fat thickness or volume with coronary artery disease.

Table 6: Comparison of Epicardial Fat Measurements in different studies in predicting CAD

Author (Year)	Study Population	Method of Measurement	Cut-off Value	Key Findings
This study (2025, Nepal)	524 patients, undergoing CTCA	EFT at RV free wall	≥3.1 mm	EFT significantly higher in CAD; correlated with CAD severity and CACS and independent predictor of obstructive CAD
Samy et al.[18] (2020, Egypt)	100 patients undergoing 256-slice MDCT	EFT at RV free wall & PCFT	≥7.2 mm (EFT) (sensitivity 67%, specificity 67.4%)	EFT & PCFT higher in obstructive CAD; correlated with CACS.
Taha et al.[20] (2019, Egypt)	180 patients referred for CTCA	Epicardial fat volume (EFV)	≥124 ml	EFV strongly correlated with CACS, CAD severity, and number of vessels involved; good diagnostic accuracy.
Gać et al.[19] (2021, Poland)	101 patients undergoing 128-slice MSCT	EATT & PATT	≥16.7 mm-specific ≤8.7 mm-sensitive	EATT and PATT correlated with CACS and high-risk CAD; much higher cut-offs than current thesis.
Aydin et al.[22] (2019, Turkey)	145 patients with risk factors for CAD	EFT & PCFT	Not specified	EFT correlated with BMI, HTN, DM, cholesterol, and calcified plaque; peri-coronary fat associated with CAD.
Lee et al.[23] (2010, South Korea)	252 patients with CACS=0	EFT at RV free wall	No significant cut-off	EFT not different between obstructive vs non-obstructive CAD in zero calcium population.
Bachar et al.[14] (2012, Israel)	190 asymptomatic patients with one additional risk factors referred for CTCA	EFT by 64 slice MDCT at RV free wall	≥ 2.4mm for obstructive CAD (sensitivity-80%, specificity-73%)	EFT was an independent predictor of CAD; patients with CAD had significantly thicker EFT compared to those without.
Maimaituxun et al.[21] (2018, Japan)	197 patients undergoing 320-slice CTCA	Localized EFT (LAD, LCX, RCA) & EFV	No absolute cut-off; EAT-LAD thickness significant	EAT around LAD higher in CAD (6.86±2.19 vs 5.45±2.16 mm; p<0.001); correlated with CAD presence (r=0.276) and severity (r=0.239); LAD-EFT improved prediction beyond Framingham risk score.
Demircelik et.al[15] (2014, Turkey)	131 patients undergoing 64 slice CTCA	EFT and PCFT	PCFT: 13.8mm (sensitivity 72.2 % specificity 68.1 %) EFT: 6.8cm (sensitivity :73.5%, specificity:69.3%)	Mean PCFT and EFT were significantly higher in those with obstructive atherosclerosis group compared with no atherosclerosis and non-obstructive group

Conclusion

Measurement of the epicardial fat thickness is an easy and effective way to assess the risk of obstructive coronary artery disease. Epicardial fat thickness along with CT coronary calcium scores are independent predictor of obstructive coronary artery disease.

Limitations

There are few limitations to this study. This was a single center-based study. CT coronary angiography report was not compared with invasive coronary artery angiography.

Conflicts of Interest: The authors declare no conflict of interest.

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References

1. Vaduganathan M, Mensah GA, Turco JV, Fuster V, Roth GA. The global burden of cardiovascular diseases and risk: a compass for future health. American College of Cardiology Foundation Washington DC; 2022. p. 2361-71.
2. Maskey A, Sayami A, Pandey MR. Coronary artery disease: an emerging epidemic in Nepal. Nepalese Heart Journal. 2003;2(2):2-6.
3. Vaidya A, Pokharel P, Nagesh S, Karki P, Kumar S, Majhi S. Prevalence of coronary heart disease in the urban adult males of eastern Nepal: a population-based analytical cross-sectional study. Dharan: BP Koirala Institute of Health Sciences. 2009.
4. Meiburger KM. Coronary artery disease severity, its predictors, and the increasingly important role of noninvasive imaging. International Journal of Cardiology. 2018;257:312-3.
5. Loh WJ, Chang X, Aw TC, Phua SK, Low AF, Chan MY-Y, et al. Lipoprotein (a) as predictor of coronary artery disease and myocardial infarction in a multi-ethnic Asian population. Atherosclerosis. 2022;349:160-5.
6. Iacobellis G, Willens HJ. Echocardiographic epicardial fat: a review of research and clinical applications. Journal of the American Society of Echocardiography. 2009;22(12):1311-9.
7. Iwasaki K, Matsumoto T, Aono H, Furukawa H, Samukawa M. Relationship between epicardial fat measured by 64-multidetector computed tomography and coronary artery disease. Clinical cardiology. 2011;34(3):166-71.
8. Marchington JM, Mattacks CA, Pond CM. Adipose tissue in the mammalian heart and pericardium: structure, foetal development and biochemical properties. Comparative Biochemistry and Physiology Part B: Comparative Biochemistry. 1989;94(2):225-32.
9. Iacobellis G. Local and systemic effects of the multifaceted epicardial adipose tissue depot. Nature Reviews Endocrinology. 2015;11(6):363-71.
10. Sacks HS, Fain JN, Holman B, Cheema P, Chary A, Parks F, et al. Uncoupling protein-1 and related messenger ribonucleic acids in human epicardial and other adipose tissues: epicardial fat functioning as brown fat. The Journal of Clinical Endocrinology & Metabolism. 2009;94(9):3611-5.
11. Iacobellis G, Pistilli D, Gucciardo M, Leonetti F, Miraldi F, Brancaccio G, et al. Adiponectin expression in human epicardial adipose tissue in vivo is lower in patients with coronary artery disease. Cytokine. 2005;29(6):251-5.
12. Silaghi A, Achard V, Paulmyer-Lacroix O, Scridon T, Tassistro V, Duncea I, et al. Expression of adrenomedullin in human epicardial adipose tissue: role of coronary status. American Journal of Physiology-Endocrinology and Metabolism. 2007;293(5):E1443-E50.
13. Mazurek T, Zhang L, Zalewski A, Mannion JD, Diehl JT, Arafat H, et al. Human epicardial adipose tissue is a source of inflammatory mediators. Circulation. 2003;108(20):2460-6.
14. Bachar GN, Dicker D, Kornowski R, Atar E. Epicardial adipose tissue as a predictor of coronary artery disease in asymptomatic subjects. The American journal of cardiology. 2012;110(4):534-8.
15. Demircelik MB, Yilmaz OC, Gurel OM, Selcoki Y, Atar IA, Bozkurt A, et al. Epicardial adipose tissue and pericoronary fat thickness measured with 64-multidetector computed tomography: potential predictors of the severity of coronary artery disease. Clinics. 2014;69(6):388-92.
16. Moradi M, Talebi V. Evaluation of epicardial adipose tissue by coronary multi-detector computed tomography: An independent predictor of obstructive coronary artery disease. Egyptian Journal of Radiology and Nuclear Medicine. 2023;54(1):68.
17. Mancio J, Azevedo D, Saraiva F, Azevedo AI, Pires-Morais G, Leite-Moreira A, et al. Epicardial adipose tissue volume assessed by computed tomography and coronary artery disease: a systematic review and meta-analysis. European Heart Journal-Cardiovascular Imaging. 2018;19(5):490-7.
18. Samy NI, Fakhry M, Farid W. Relation between epicardial adipose tissue thickness assessed by multidetector computed tomography and significance of coronary artery disease. World Journal of Cardiovascular Diseases. 2020;10(02):91.
19. Gać P, Macek P, Poręba M, Kornafel-Flak O, Mazur G, Poręba R. Thickness of epicardial and pericoronary adipose tissue measured using 128-slice MSCT as predictors for risk of significant coronary artery diseases. Irish Journal of Medical Science (1971-). 2021;190(2):555-66.
20. Taha DA, El Shafey RAA, Hamesa MF, Abu-Dewan KAE-W, Nagy HA. Relationship between epicardial fat volume measured by multi-detector computed tomography and coronary artery disease. Egyptian Journal of Radiology and Nuclear Medicine. 2021;52(1):235.
21. Maimaituxun G, Shimabukuro M, Fukuda D, Yagi S, Hirata Y, Iwase T, et al. Local Thickness of Epicardial Adipose Tissue Surrounding the Left Anterior Descending Artery Is a Simple Predictor of Coronary Artery Disease—New Prediction Model in Combination With Framingham Risk Score—. Circulation Journal. 2018;82(5):1369-78.
22. Aydın AM, Kayalı A, Poyraz AK, Aydın K. The relationship between coronary artery disease and pericoronary epicardial adipose tissue thickness. Journal of International Medical Research. 2015;43(1):17-25.
23. Lee HY, Song IS, Yoo SM, Rho JY, Moon JY, White CS. Can the extent of epicardial adipose tissue thickness or the presence of descending thoracic aortic calcification predict significant coronary artery stenosis in patients with a zero coronary calcium score on multi-detector CT? Atherosclerosis. 2010;212(2):495-500.