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Relation of Pericardial Adipose Tissue Thickness by Echocardiography to Coronary Artery Disease

Kawa Wsu Hassan [1], Omeed Faeq Othman[2]

[1]MD, MBChB, FEBC (Card), FIBMS (med), Dept. of Sulaymaniyah Cardiac Hospital.

Kawahassan79@gmail.com

[2]MSc. Cardiology, Dept. of Sulaymaniyah Cardiac Hospital. Omeedfothman@yahoo.com

Kawahassan79@gmail.com

Abstract

Background and Objectives: Coronary artery disease (CAD) continues to be a predominant cause of global morbidity. This study aimed to investigate the association between the thickness of pericardial adipose tissue (PAT) and the intensity and spread of CAD.

Methods: A cross-sectional observational study was conducted at Sulaimanyah Cardiac Hospital from January to December 2023. A total of 100 patients undergoing their first coronary angiography due to chest pain were recruited. The collection of data encompassed interviews, physical assessments, laboratory tests, electrocardiograms, echocardiograms, and coronary angiographies.

Results: Patients were categorized based on PAT thickness into two groups: PAT ≤ 0.3 cm 20 (20%) and PAT > 0.3 cm 80 (80%). Significant findings included higher rates of systemic hypertension (p=0.02), Waist circumference (WC) (p< 0.001), metabolic syndrome (p=0.003), atypical chest pain (p<0.01), abdominal obesity (p<0.01), and ischemic ECG finding (p=0.02) in the thicker PAT group. Echocardiographic and angiographic assessments revealed a significant association between increased PAT thickness and greater prevalence and severity of CAD. Notably, the presence of CAD correlated significantly with PAT thickness (r=0.350, p<0.001).

Conclusion: Increased PAT thickness is associated with the severity and extent of CAD, suggesting that PAT could serve as a potential marker for cardiovascular risk stratification.

Keywords: Abdominal Obesity, Cardiovascular Risk, Coronary Angiography, Hypertension, Metabolic Syndrome.

1. Introduction

Coronary artery disease (CAD) is primarily caused by the buildup of atherosclerotic plaque within the coronary arteries, which are essential for delivering oxygenated blood to the heart muscle (1). This buildup results in the narrowing of the arteries, limiting blood flow and oxygen supply (2). CAD is a major cause of mortality and is responsible for a significant number of lost Disability Adjusted Life Years (DALYs) globally, particularly in low- and middle-income nations. It is

estimated that CAD contributes to approximately 7 million deaths and 129 million DALYs each year (3).

Aside from established risk factors including hypertension, diabetes, and smoking, the occurrence of CAD is also impacted by several intricate variables, including obesity and metabolic disorders (4). Precisely identifying CAD is essential for initiating effective treatment regimens. Diagnostic techniques encompass non-invasive procedures such as electrocardiograms (ECG), stress tests, and sophisticated imaging modalities such as coronary angiography (CA), CT angiography, and MRI (5, 6). Echocardiography, in particular, stands out due to its non-invasive nature, accessibility (7), , and cost-effectiveness, and recent advancements have enabled its use in measuring PAT thickness, positioning it as a promising biomarker for CAD (8).

The visceral fat deposit around the heart known as PAT has been linked to the pathophysiology of CAD through mechanisms involving local inflammation and adipokine secretion that may affect coronary atherogenesis. Therefore, measuring the thickness of PAT through echocardiography potentially offers a unique window into assessing CAD risk, providing an integrative marker of metabolic risk and cardiovascular health (9). A research done by Szabo et al. (2024) indicated that the quantity and nature of PAT distinguish persons with prevalent heart failure (HF) and predict the incidence of future HF (10). The study of Muzurović et al. (2021) also shows that modification of epicardial adipose tissue (EAT)/PAT can be a potential therapeutic target to reduce the burden of cardiovascular disease (CVD) (11).

Despite these advances, significant gaps remain in our understanding of the relationship between PAT thickness and CAD. Most existing studies have utilized CT or MRI for the measurement of PAT, which, while effective, exposes patients to ionizing radiation. Additionally, because limited studies have been done in this field; The purpose of this research was to investigate the correlation between the degree and severity of CAD as determined by CA and PAT thickness as determined by two-dimensional echocardiography.

2. Methods and Materials

2.1. Study Design and Setting

This cross-sectional observational study was conducted at Sulaymaniyah Cardiac Hospital. The study was carried out over one year, from January 2023 to December 2023.

2.2. Participants

This study involved 100 patients who had been admitted for their first invasive CA procedures because of chest pain. The participants were chosen through Convenience sampling technique. On the basis of PAT thickness all patients were classified into two PAT thickness groups. PAT thickness of 0. 3 cm has been used as the cut-off for categorizing our sample patients. Group A (GA): patients with a mean PAT thickness equal to ≤ 0.3 cm. Group B (GB): patients with a mean PAT thickness of > 0.3 cm (12).

The investigation included adults >18 years who were admitted to the coronary care unit with their first episode of chest pain and who agreed to participate in the study. Individuals were excluded if

they had renal failure, infections, a history of stroke, anemia, uncontrolled hypertension, electrolyte imbalances, psychological or systemic illnesses, life-threatening conditions, refused treatment, exhibited digitalis intoxication, had allergies to contrast media, vascular disease, HF, coagulopathies, an aortic aneurysm, pericardial effusion, severe valvular disease, or if their echocardiographic imaging was of poor quality.

2.3. Data Collection Methods

Data was gathered via patient interviews, physical examinations, laboratory analyses, electrocardiography, and echocardiography. Laboratory evaluations included checks for fasting plasma glucose, lipid profiles, serum creatinine, and hemoglobin. Patients were evaluated for chest pain severity, ranging from atypical to stable and unstable angina, using the Killip classification to categorize HF severity in cases of acute myocardial infarction. Data collection involved patient interviews, physical examinations, lab tests, electrocardiography, and echocardiography. Lab tests included measurements of fasting plasma glucose, lipid panels, serum creatinine, and hemoglobin levels. Patient evaluations also considered the severity of chest pain and Killip classification for HF severity during acute myocardial infarction. Metabolic syndrome (MS) was determined based on the presence of at least three of the following five components. Firstly, an elevated WC is considered a risk factor, with the threshold set at > 102 cm for men and > 88 cm for women. Secondly, elevated triglycerides, ≥ 150 mg/dL. Thirdly, reduced HDL cholesterol, less than 40 mg/dL. Fourthly, elevated blood pressure, ≥ 130/85 mm Hg, or the use of medication for hypertension. Lastly, elevated fasting glucose, ≥ 100 mg/dL, or the use of medication for hyperglycemia (13). Physical evaluations also included blood pressure measurements, arterial pulse checks, heart auscultation, and WC measurements.

2.3.1. Electrocardiography and Echocardiography

A 12-lead ECG was performed using a GE MAC 2000 ECG machine to identify ischemic changes. Echocardiographic studies were conducted using commercially available systems (Philips EPIQ 7). The measurements consisted of assessing the thickness of the PAT on the right ventricle's free wall in the still images of the two-dimensional echocardiogram. These images were obtained at end-diastole and viewed from both the parasternal long-axis and short-axis perspectives. The diastolic function of the left ventricle was evaluated using pulse wave Doppler of the mitral inflow. Ejection fraction (EF) was measured to evaluate systolic left ventricular function. Wall motion and myocardial thickening were assessed to detect any wall motion abnormality.

2.3.2. Coronary Angiography

The CA was performed using either a trans-femoral or trans-radial approach on a Siemens Artis zee system, ensuring at least two orthogonal plane views of each coronary vessel. The interpretations of the angiograms were conducted by independent, blinded specialists. The presence of significant coronary lesions and the extent of vessel involvement, categorized as 'single', 'two', or 'multiple', were determined based on the degree of luminal stenosis. A stenosis of greater than 50% in the left main coronary artery or over 70% in other major epicardial arteries was considered significant (14). The lesions were further classified according to the American

College of Cardiology/American Heart Association (ACC/AHA) lesion type classification system (15).

2.4. Ethical Considerations

The research received approval from the hospital's ethics committee. All participants provided informed permission after receiving a detailed explanation of the study's aim, methods, and possible hazards.

2.5. Statistical Analysis

The data were analyzed using the Statistical Package for Social Sciences (SPSS) version 17. The frequencies were used to define the categorical data, and Chi-square or Fischer's exact tests were used to compare them, depending on the circumstances. Mean and Standard Deviation were used to characterize continuous variables, and they were compared using the Mann-Whitney and t-test. The strength of the relationships between the variables was assessed using Pearson's correlation coefficients at a significance threshold of $p < 0.05$, indicating statistical significance.

3. Results

The participants were categorized into two groups according on their PAT thickness. The percentage of PAT thickness showed that 20 (20%) patients had PAT ≤ 0.3 cm and 80 (80%) had PAT > 0.3 cm (Figure 1).

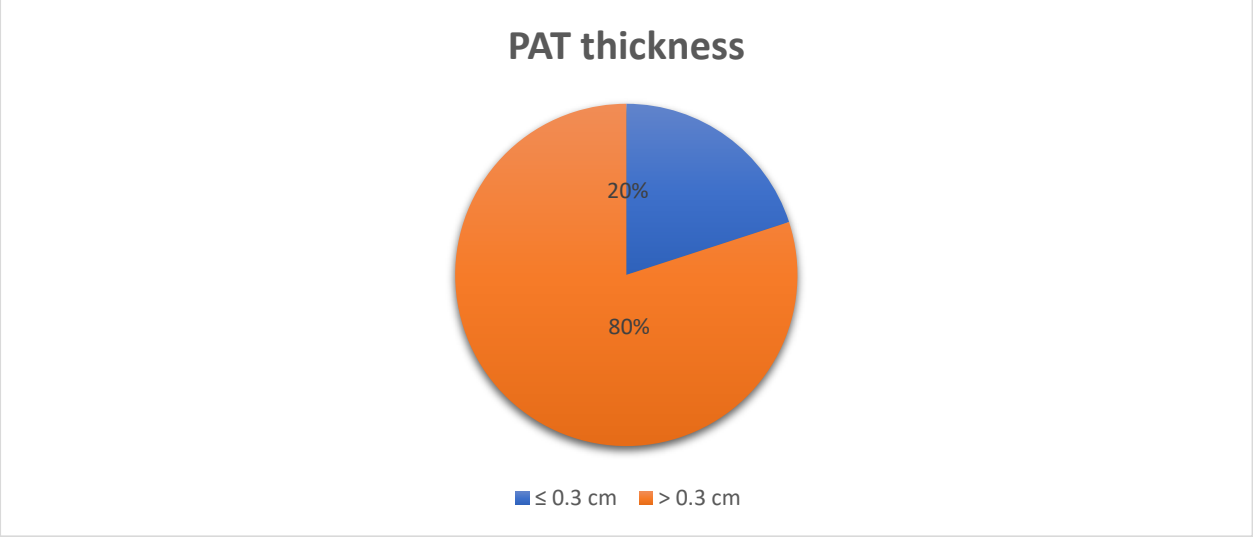

Figure (1): PAT thickness percentage of the two studied groups

The mean age was 55.5 ± 10 years for GA and 58.15 ± 9.1 years for GB, with no notable difference (p=0.257). The sex distribution was similar, with males comprising 15 (75%) of GA and 59 (73%) of GB (p=0.57), while females comprised 5 (25%) of GA and 21 (26.3%) of GB. GB had 59 cases (73%) of systemic hypertension, but GA had only 7 cases (35%) (p≤0.02). Three (15%) of GA's population had abdominal obesity, while fifty (65%) of GB had it (p≤0.01). In GB (101.4 ± 7.13 cm) compared to GA (94.65 ± 6.59 cm), the mean WC was substantially higher (p≤0.001). In GA, 2 (10%) patients satisfied at least three of the criteria for MS, according to the findings of the

analysis of the MS variable. While in GB, 43 (53.75%) patients had this criterion (p≤0.003). In addition, the results of examining the variables of smoking, family history of CAD, DM, and dyslipidemia between the two groups were not notable significant (p>0.05) (Table 1).

Clinically, atypical chest pain was significantly more common in GA at 10 (50%) versus 8 (10%) in GB (p≤0.01). Stable angina was observed in 8 (40%) of GA and 49 (61%) of GB (p=0.07), and unstable angina in 2 (10%) of GA and 23 (29%) of GB (p=0.06). Killip class I was recorded in 18 (90%) of GA and 61(76.3%) of GB (p=0.147), while Killip class II was 2 (10%) in GA and 19 (23.8%) in GB. Ischemic ECG findings were significantly higher in GB at 54 (67.5%) compared to 8 (40%) in GA (p≤0.02) (Table 1).

Table (1): Demographic and clinical characteristic of the whole studied population.

Variable		Group A (N=20)	Group B (N=80)	P. Value
Demographic Characteristic				
Age (Mean ± SD)		**55.5± 10**	**58.15 ± 9.1**	**0.257**
Sex	**Male**	**15 (75%)**	**59 (73%)**	**0.57**
	Female	**5 (25%)**	**21 (26.3%)**	
Smoking		**9 (45%)**	**37 (46.3%)**	**0.56**
Family History Of CAD		**3 (15%)**	**20 (25%)**	**0.26**
Systemic HTN		**7 (35%)**	**59 (73%)**	**0.02**
DM		**8 (40%)**	**36 (45%)**	**0.44**
Dyslipidemia		**8 (40%)**	**48 (60%)**	**0.17**
Abdominal Obesity		**3 (15%)**	**50 (65%)**	**≤ 0.01**
WC (Mean ± SD)		**94.65± 6.59**	**101.4 ± 7.13**	**≤ 0.001**
MS	**0**	**5 (25%)**	**5 (6.25%)**	**≤ 0.003**
	1	**7 (35%)**	**9 (11.25%)**	
	2	**6 (30%)**	**23 (28.75%)**	
	3	**1 (5%)**	**15 (18.75%)**	
	4	**1 (5%)**	**22 (27.5%)**	
	5	**0 (0%)**	**6 (7.5%)**	
Clinical And Electrocardiographic Characteristic				
Atypical Chest Pain		**10 (50%)**	**8 (10%)**	**≤ 0.01**
Stable Angina		**8 (40%)**	**49 (61%)**	**0.07**
Unstable Angina		**2 (10%)**	**23 (29%)**	**0.06**
Killip Class	**I**	**18 (90%)**	**61 (76.3%)**	**0.147**
	II	**2 (10%)**	**19 (23.8%)**	
Ischemic ECG Finding		**8 (40%)**	**54 (67.5%)**	**≤ 0.02**

Table 2 shows the echocardiographic findings of systolic and diastolic function of the two studied groups. There was no discernible disparity between the two groups in terms of EF and diastolic dysfunction.

Table (2): Echocardiographic Assessment of Left Ventricular Function.

Variable	Group A	Group B	P Value
EF, Mean±SD	**59.85 ± 7.18**	**59.21 ± 7.72**	**0.52**
Diastolic Dysfunction	**5 (25%)**	**34 (42%)**	**0.11**

Concerning the findings of CA, there was a significantly greater proportion of individuals displaying normal coronary arteries in GA, with 9 participants (45%), in comparison to GB, with 5 participants (6.3%) (p≤0.01). Furthermore, there was a notably lower occurrence of LAD involvement in GA, with 4 participants (20%) as opposed to GB, with 40 participants (50%) (p≤0.02). The incidence of proximal or osteal LAD involvement was nil (0%) in GA, whereas it was markedly higher at 22 participants (27.5%) in GB (p≤0.01). Similarly, while diagonals were not involved in GA, they were present in 15 participants (18.75%) of GB (p≤0.02). LCX involvement was identified in 3 participants (15%) of GA and 35 participants (43.75%) of GB (p=0.04 The results of the number of diseased blood vessels showed that the largest number of participants 9 (45%) in GA had Normal and One vessel. On the other hand, in GB, 43 (53.8%) patients had two vessels (p≤0.01). The results of ACC/AHA lesion classifications showed that the largest number of participants (45%) in GA had normal vessels and in GB 44 (55%) patients had lesion type B (p≤ 0.01).

Table (3): Statistical Analysis of CA Findings and Vessel Disease Severity in Relation to PAT Thickness.

Variable	Group A (N=20)	Group B (N=80)	P. Value
Coronary Angiographic			
Normal CA	**9 (45%)**	**5(6.3%)**	**≤ 0.01**
LMA	**0 (0%)**	**5 (6.3%)**	**0.31**
LAD	**4 (20%)**	**40 (50%)**	**≤ 0.02**
Proximal Or Osteal LAD	**0 (0%)**	**22 (27.5%)**	**≤ 0.01**
Diagonals	**0 (0%)**	**15 (18.75%)**	**≤ 0.02**
LCX	**3 (15%)**	**35 (43.75%)**	**≤ 0.04**
RCA	**5 (25%)**	**35 (43.75%)**	**0.19**
PDA	**1 (5%)**	**12 (15%)**	**0.18**
Number Of Diseased Blood Vessels			
Normal	**9 (45%)**	**5 (6.3%)**	**≤ 0.01**
One Vessel	**9 (45%)**	**15 (18.8%)**	
Two Vessels	**2 (10%)**	**43 (53.8%)**	
Three Or More Vessels	**0 (0%)**	**17 (21.3%)**	
ACC/AHA Lesion Type			
Normal Vessel	**9 (45%)**	**5 (6.3%)**	**≤ 0.01**
Lesion Type A	**5 (25%)**	**11 (13.8%)**	
Lesion Type B	**6 (30%)**	**44 (55%)**	
Lesion Type C	**0 (0%)**	**20 (25%)**	

Figure 2 shows that the median PAT was thicker in patients with CAD (0.49 cm) than those with normal coronary arteries (0.35 cm). Patients with three or more vessels of CAD had significantly thicker PAT than those with two vessels, a single vessel, and a normal coronary artery. Median

PAT thickness was 0.55 cm in patients with three or more vessel CAD, 0.5 cm in those with two vessel CAD, 0.4 cm in single vessel disease, and 0.3 cm in those with normal coronary arteries.

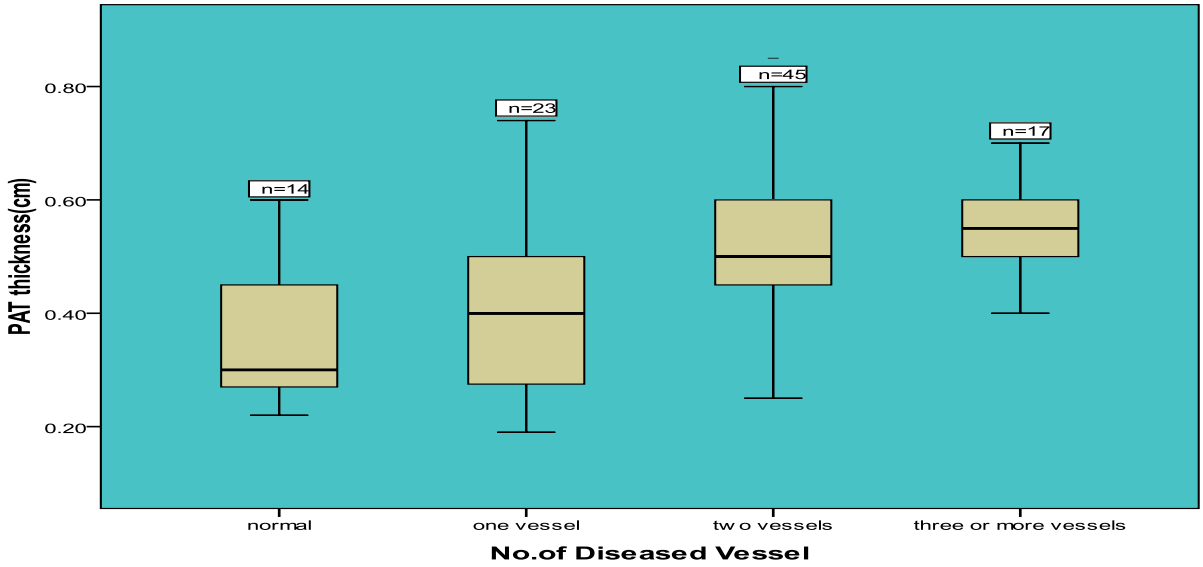

Figure (2): Comparison of the PAT thickness according to the number of diseased coronary arteries.

Table 4 shows the correlations between PAT thickness measured via two-dimensional echocardiography and various risk factors and manifestations of CAD as assessed by CA. Hypertension showed a positive correlation with PAT thickness (r=0.213, p≤0.03). A strong correlation was noted with abdominal obesity (r=0.410, p≤0.001) and WC (WC; r=0.424, p≤0.001), indicating that increased central adiposity is significantly associated with greater PAT thickness. Similarly, the presence of MS was strongly correlated with increased PAT thickness (r=0.406, p≤0.001). Additionally, ischemic ECG findings were positively correlated with PAT thickness (r=0.229, p≤0.02). In terms of the presence and extent of CAD, overall CAD presence correlated significantly with PAT thickness (r=0.350, p≤0.001), along with specific coronary artery involvements such as the LAD artery (r=0.254, p≤0.01), the left circumflex artery (LCX; r=0.273, p≤0.006), and the diagonals (r=0.266, p≤0.007). Moreover, the number of diseased vessels was strongly correlated with PAT thickness (r=0.494, p≤0.001). The study also found significant correlations between PAT thickness and ACC/AHA lesion type (r=0.398, p≤0.001).

Table (4): Statistically Significant Correlations Between PAT Thickness and CAD Risk Factors and Indicators

PAT Thickness	**R**	***P Value***

Risk Factors Of CAD		
Family History Of CAD	**0.171**	**0.09**
Smoking	**-0.057**	**0.571**
Diabetes Mellitus (DM)	**-0.018**	**0.856**
HTN	**0.213**	**≤ 0.03**
Dyslipidemia	**0.162**	**0.12**
Sex	**0.084**	**0.407**
Abdominal Obesity	**0.410**	**≤ 0.001**
WC	**0.424**	**≤ 0.001**
MS	**0.406**	**≤ 0.001**
Ischemic ECG Findings		
Ischemic ECG Finding	**0.229**	**≤ 0.02**
Presence And Extent Of CAD		
CAD	**0.350**	**≤ 0.001**
LMA	**0.082**	**0.415**
LAD	**0.254**	**≤ 0.01**
Proximal Or Osteal LAD	**0.073**	**0.470**
LCX	**0.273**	**≤ 0.006**
Diagonals	**0.266**	**≤ 0.007**
RCA	**0.021**	**0.837**
PDA	**0.133**	**0.188**
No. Of Diseased Vessels	**0.494**	**≤ 0.001**
ACC/AHA Lesion Type		
ACC/AHA Lesion Type	**0.398**	**≤ 0.001**

In the multiple logistic analysis of various risk factors for CAD, several factors demonstrated statistically significant associations. Age was a notable predictor with an odds ratio (OR) of 36.889 (95% CI: 7.716-17.63), indicating that older age significantly increased the likelihood of CAD (p≤0.001). Smoking also showed a notable association, with an OR of 6.286 (95% CI: 1.327-29.77) (p≤0.009). Similarly, DM was significantly related to CAD, with an OR of 5.727 (95% CI: 1.209-27.133) (p≤0.014). Hypertension was another significant factor, with an OR of 17.455 (95% CI: 3.619-84.181) (p<0.001). Dyslipidemia had a significant OR of 11.423 (95% CI: 2.359-55.326) (p≤0.001). The WC showed a notable association with an OR of 3.311 (95% CI: 0.962-11.391) (p≤0.045). Finally, PAT thickness was a highly significant predictor of CAD, with an OR of 12.273 (95% CI: 3.470-43.403) (p≤0.001) (Table 5).

Table (5): Multiple logistic analysis of different risk factors in CAD.

Risk Factors	Odd Ratio (95% CI)	P Value
Age	**36.889 (7.716-17.63)**	**≤ 0.001**
Smoking	**6.286 (1.327- 29.77)**	**≤ 0.009**

Family History	**1.938 (0.401-9.371)**	**0.325**
DM	**5.727 (1.209-27.133)**	**≤ 0.014**
Hypertension	**17.455 (3.619-84.181)**	**≤ 0.001**
Dyslipidemia	**11.423 (2.359-55.326)**	**≤ 0.001**
WC	**3.311 (0.962-11.391)**	**≤ 0.045**
PAT Thickness	**12.273 (3.470- 43.403)**	**≤ 0.001**

4. Discussion

The investigation aimed to investigate the link between the thickness of PAT and the extent and presence of CAD, as identified through CA. The findings demonstrated a robust relationship between a higher PAT thickness and an increased severity of CAD. Specifically, those with thicker PAT frequently exhibited more severe forms of CAD, characterized by broader coronary artery involvement and a greater number of affected arteries. Additionally, a thicker PAT was linked to several cardiovascular risk factors, including high blood pressure, central obesity, and MS, which are all recognized as factors that can lead to the progression of coronary artery disease.

Pericardial adipose tissue (PAT), a crucial ectopic fat storage site, is linked to heightened risks of CVD. PAT consists primarily of EAT, which is located between the heart's myocardium and the visceral layer of the pericardium, and paracardial adipose tissue (16). Previous studies have established a reliable relationship between the measurement of PAT thickness via echocardiography and MRI, suggesting that echocardiography is a dependable method for estimating the thickness of PAT (17).

This study investigated the relationship between PAT thickness and conventional cardiovascular risk factors. The findings indicated a significant relationship between PAT thickness and systemic hypertension. This observation is consistent with the results reported by Guan et al. (2021), and Iacobellis (2009), who found that both intrathoracic and pericardial adipose tissue were associated with hypertension (18, 19). Abdominal obesity and WC showed strong relationship with PAT thickness. These findings are in line with prior research indicating that central adiposity is a major determinant of PAT. A study by Bai et al. (2023) corroborates these results, emphasizing that visceral adiposity, as indicated by WC and abdominal obesity, is closely linked to increased PAT (20).

Metabolic syndrome is a cluster of interconnected factors that increase the risk of CVD and type 2 diabetes. These factors include central obesity, hypertension, dyslipidemia, and impaired glucose tolerance (21). The PAT is the fat depot located between the parietal and visceral pericardium. Like EAT, PAT is associated with MS. A study conducted by Kim et al. (2018) found that higher PAT volume was independently associated with a greater prevalence of MS (22). Another research found that the amount of EAT was considerably greater in persons with MS compared to those without the condition. (17).

Moreover, ischemic ECG findings were positively correlated with PAT thickness, supporting the notion that increased PAT may be associated with myocardial ischemia. The association between

PAT and myocardial ischemia has been further supported by studies showing that increased EAT, is linked to coronary artery disease. EAT is thought to provide a direct source of free fatty acids to the myocardium and secrete inflammatory cytokines that may contribute to the development of atherosclerosis (23, 24).

Prior research has shown inconsistent findings on the relationship between pericardial fat and angiographic CAD, perhaps because to variations in measuring methodologies and study cohorts. Jeong et al. (2007) discovered a direct relationship between the thickness of pericardial fat and the degree of CAD in individuals who were mostly not obese but already had CAD (25). Conversely, Chaowalit et al. (2006) did not observe an association between epicardial fat thickness and angiographic CAD (26). A recent study conducted by Greif et al. (2009) used cardiac multidetector computed tomography (MDCT) to investigate the relationship between pericardial fat and coronary plaques. The findings of the study indicated a positive relationship between the quantity of pericardial fat and the existence of coronary plaques (27). Taguchi et al. (2001) discovered a significant relationship between the amount of fat around the heart (pericardial fat) and the occurrence of CAD in Japanese individuals who were not obese (28).

The present study revealed a significant positive relationship between pericardial adipose tissue (PAT) thickness and ACC/AHA lesion type. The median PAT thickness was greater in patients with lesion type C compared to those with lesion types B, A, and normal coronary arteries. These observations indicate that PAT is related to significant coronary lesion types, implying that pericardial fat might have unfavorable direct perivascular influences on coronary atherogenesis through inflammation (29, 30). To sum up, in the current research, investigating various risk factors for CAD, it is found that PAT thickness is a definitive risk factor linked with CAD.

5. Conclusions

The findings of the current research pointed towards a direct proportional relationship between the PAT thickness and the extent as well as the frequency of CAD with many other cardiovascular risk factors such as hypertension, abdominal obesity, and MS. Such outcomes accentuate the necessity of comprehending the role of PAT thickness for the assessment of cardiovascular health and risk differentiation processes in clinical practice. In the same way, the PAT thickness was found to increase with more severe CAD, suggesting that a targeted approach to the central fat accumulation is necessary within the general strategy, aimed at prevention and cessation of CVDs.

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