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Association of Carotid Intima Media Thickness with the Extent of Angiographically Proven Coronary Artery Disease

Jahanzeb Ibrahim, Elishba Qazi, Zuhoor Uddin, Kalsoom Nawab, Sahib Noor, Laila Haleem

ABSTRACT

Objective: To evaluate the association between carotid intima-media thickness (CIMT) with the extent of angiographically proven coronary artery disease (CAD).

Study Design and Setting: A cross-sectional study was conducted at Khyber Teaching Hospital (KTH), Peshawar.

Methodology: The study included 65 patients receiving diagnostic coronary angiography and was conducted from 1st April 2025 to 30th September 2025. Cardiovascular risk factors were among the baseline clinical and demographic features that were documented. High-resolution B-mode ultrasonography was used to measure CIMT. The degree of CAD was classified as either no CAD, single-vessel, two-vessel, or three-vessel disease, and its severity was measured using the Gensini score. Regression and correlation models were used to examine relationships between CIMT and angiographic results. The diagnostic accuracy of CIMT for predicting obstructive CAD was assessed using receiver operating characteristic (ROC) curve analysis.

Results: The mean age of participants was 56.2 ± 9.8 years, with 64.6% males. Obstructive CAD was present in 75.4% of patients. CIMT increased progressively with the extent of CAD, from 0.68 ± 0.10 mm in patients without CAD to 1.02 ± 0.13 mm in those with three-vessel disease ($p < 0.001$). CIMT showed a strong positive correlation with the Gensini score ($p < 0.001$). ROC analysis demonstrated good diagnostic accuracy (AUC = 0.82).

Conclusion: CIMT is a reliable, non-invasive marker that correlates with the severity of CAD and can aid in early risk stratification and clinical decision-making.

Keywords: Carotid Intima-Media Thickness; Coronary Angiography; Coronary Artery Disease; Gensini Score; Risk Factors; Ultrasonography

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INTRODUCTION

A significant portion of the burden of atherosclerotic cardiovascular disease, which is still the primary cause of morbidity and mortality globally, is attributed to coronary artery disease (CAD). Therefore, early detection of subclinical atherosclerosis is essential for risk assessment, primary prevention, and prompt intervention. It has long been suggested that carotid intima-media thickness (CIMT), which is determined by B-mode ultrasonography of the common carotid artery, is a noninvasive proxy for systemic atherosclerosis and vascular aging. As a simple, reproducible imaging metric, CIMT promises to bridge the gap between traditional risk scoring and direct assessment of arterial pathology, and has consequently been the subject of intense study for its association with the presence and extent of angiographically proven CAD.[1, 2]

Physiologically, CIMT reflects both adaptive and pathological changes in the arterial wall, including medial hypertrophy, intimal thickening, and early atheroma formation. These changes often mirror coronary atherosclerosis because they share systemic risk factors like hypertension, dyslipidaemia, diabetes, and smoking. Drive

diffuse arterial remodeling. However, CIMT and coronary plaque differ in some pathobiological attributes. CIMT measures wall thickness but does not fully capture plaque burden, composition, or vulnerability, which are features that determine clinical events. Accordingly, the debate has shifted from whether CIMT correlates with cardiovascular risk to how well it predicts the presence, extent, and anatomical severity of CAD on coronary angiography.[2, 3]

Epidemiologic cohort studies and meta-analyses have repeatedly shown that increased CIMT is associated with higher rates of cardiovascular events and incremental risk over conventional factors in some populations.[4, 5] Nevertheless, the magnitude and clinical utility of that association vary by age, measurement protocol (mean vs. maximum CIMT, which arterial segment is measured), and whether carotid plaque is present, plaque burden often being a stronger predictor than intima-media thickening alone. Recent systematic appraisals emphasize that heterogeneity in CIMT definitions and measurement techniques limits direct comparability between studies and complicates translation into practice.[6, 7]

When investigators have compared CIMT directly with angiographic findings, results are mixed but informative. Higher CIMT values are linked to the presence of obstructive CAD and multivessel disease, according to many cross-sectional studies that involve individuals who have been sent for coronary angiography. This supports the idea that CIMT serves as a measure of generalized atherosclerotic load. At the same time, other studies demonstrate only weak or modest correlations, particularly when plaque characteristics or coronary calcification are considered, underscoring that a thickened intima-media is only one facet of a complex systemic process [7, 8]

Investigators have explored combining CIMT with biochemical markers, like lipid parameters, inflammatory indices, or homocysteine, and with carotid plaque assessment to improve the prediction of angiographic CAD and acute coronary syndromes. Several contemporary cohorts have demonstrated that models incorporating carotid plaque presence or plaque burden outperform models using CIMT alone for identifying significant coronary stenosis. Moreover, subgroup analyses suggest age-dependent differences in predictive power; CIMT may be more informative in middle-aged individuals than in the very young or elderly, where competing structural and degenerative changes confound interpretation.[7, 9, 10]

Clinical implications stem from these empirical observations. If CIMT reliably reflects the extent of angiographic CAD, it could be used to triage symptomatic patients, refine risk estimates in persons with intermediate risk scores, or monitor response to therapies that target atherosclerotic progression. Conversely, if the association is weak or inconsistent, overreliance on CIMT could misclassify patients and divert resources from more informative tests like coronary CT angiography or invasive angiography when clinically indicated. Therefore, clarifying the strength and determinants of the CIMT–CAD relationship, particularly in populations undergoing coronary angiography, is essential.[11, 12]

Coronary artery disease (CAD) is still one of the leading causes of death globally, and preventing negative consequences and managing the condition effectively depend on early detection. A non-invasive indicator of subclinical atherosclerosis, carotid intima-media thickness (CIMT), has drawn more attention as a possible proxy for coronary atherosclerotic load. Assessing CIMT may assist in identifying those at risk of substantial CAD without the need for invasive procedures because the pathophysiological causes of atherosclerosis in the carotid and coronary arteries are similar. The purpose of this study was to assess the relationship between the degree of angiographically confirmed coronary artery disease and carotid intima-media thickness.

METHODOLOGY

This analytical cross-sectional study was conducted in the Department of Radiology, Khyber Teaching Hospital, Peshawar, over a period of six months from 1st April 2025 to 30th September 2025. The objective of the study was to determine the association between carotid intima-media thickness (CIMT) and the extent of angiographically proven coronary artery disease (CAD). Ethical approval for the study was obtained from the Institutional Research and Ethics Board of Khyber Medical College, under approval number 281/DME/KMC dated 21 March 2025.

The sample size was calculated using OpenEpi software based on the expected prevalence of obstructive coronary artery disease of 20%, a confidence level of 95%, and a margin of error of 8%. The sample size of the study was 96 patients, but a total of 65 patients were included in the study through the study period owing to time and resource constraints. [13]

A consecutive sampling technique was employed. Patients aged 18 years and above, referred for diagnostic coronary angiography due to suspected CAD, were enrolled. Exclusion criteria included a history of coronary interventions such as coronary artery bypass grafting or percutaneous coronary intervention, known carotid artery disease or prior carotid intervention, poor acoustic windows on ultrasound, pregnancy, severe systemic illness such as end-stage renal or liver disease, and inability to cooperate with the procedure. Arterial diseases like cardiomyopathies, chronic obstructive pulmonary disease, lung diseases affecting the coronary circulations especially hypoxic diseases and valvular heart diseases were also excluded.

A structured proforma was used to gather data. Before registration, all individuals gave their informed consent. Age, sex, and cardiovascular risk factors like hypertension, diabetes mellitus, dyslipidemia, smoking history, and family

history were recorded, as well as clinical presentations such as stable angina or acute coronary syndrome. Additionally documented were baseline laboratory tests such as the fasting lipid profile and the fasting blood glucose, or HbA1c. To ensure temporal consistency, carotid ultrasonography was done either two weeks after the surgery or before angiography, whenever feasible. A consultant radiologist of over five years' experience in vascular ultrasonography performed all ultrasound tests and was blinded to angiographic results.

Carotid intima-media thickness was measured using high-resolution B-mode ultrasonography with a 7.5–12 MHz linear array transducer. Patients were examined in a supine position with the head slightly extended and rotated contralaterally. Measurements were taken from the far wall of the common carotid artery, one centimeter proximal to the carotid bulb, on both sides. A minimum of three measurements was obtained from each side, and the mean CIMT was calculated. Maximum CIMT and the presence of focal carotid plaques, defined as focal wall thickening of 1.5 mm or greater than 50% of the surrounding intima-media thickness, were also documented. All ultrasound examinations were performed by an experienced radiologist who was blinded to the angiographic results in order to reduce observer bias.

Coronary angiography was performed using the standard Judkins technique.[14] All angiograms were reviewed by a consultant interventional cardiologist who was blinded to the CIMT findings. These procedures were conducted by skilled consultant interventional cardiologists in accordance with the standard practice in the clinics; but, all angiographic results were analyzed and confirmed by one consultant interventional cardiologist to ensure consistency in evaluation. Coronary artery disease severity was quantified by two approaches: first, by the number of vessels showing 50% or greater luminal stenosis, categorized as single-, double-, or triple-vessel disease; and second, by calculating the Gensini score[15], which provides a weighted estimate of both the severity and anatomical location of stenotic lesions, thereby reflecting the overall burden of coronary atherosclerosis. A score was assigned to each constriction in a coronary artery according to the percentage of blockage (e.g., 25, 50, 75, 90, 99, and 100). The scores were then weighted based on the position of the blockage in the coronary arteries and all the scores were summed to come up with the total Gensini score which is proportional to the severity of the coronary artery disease in general.

Data analysis was conducted using SPSS version 26. Continuous variables such as age, body mass index, CIMT, and Gensini score were expressed as mean with standard deviation or as median with interquartile range, depending on their distribution. Categorical variables, including sex, diabetes mellitus, hypertension, smoking, and the presence of CAD, were expressed as frequencies and percentages. The association between CIMT and angiographic extent of blockage was assessed using Pearson correlation coefficients. Comparisons of CIMT across categories of CAD were evaluated using analysis of variance or the Kruskal–Wallis test. Multivariable linear regression analysis was performed to assess the independent association between CIMT and the Gensini score after adjusting for conventional risk factors. Logistic regression was also applied to evaluate CIMT as a predictor of obstructive CAD, defined as luminal stenosis of 50 percent or greater. The diagnostic performance of CIMT was further assessed by receiver operating characteristic curve analysis. A p-value =0.05 was considered statistically significant.

RESULTS

A total of 65 patients were included in the study with a mean age of 56.2 ± 9.8 years. Among them, 64.6% were male, and 35.4% were female, with a mean body mass index (BMI) of 27.4 ± 3.9 kg/m². Regarding cardiovascular risk factors, 60.0% were hypertensive, while 43.1% had diabetes mellitus. Dyslipidemia was observed in 52.3%, and 29.2% were current smokers. A positive family history of coronary artery disease (CAD) was present in 32.3%, whereas 67.7% reported no such history. Upon angiographic evaluation, 75.4% of patients had obstructive CAD (=50% stenosis), whereas 24.6% did not have any obstructive disease. In terms of vessel involvement, 24.6% of patients had no angiographically visible CAD, whereas 27.7% had single-vessel disease, 23.1% had two-vessel disease, and 24.6% had three-vessel disease. The study population's median Gensini score was 42 (IQR: 28–68). (Table 1) The mean CIMT values for patients without CAD were 0.68 ± 0.10 mm, those with single-vessel disease were 0.82 ± 0.12 mm, and those with two- and three-vessel disease were 0.91 ± 0.11 mm and 1.02 ± 0.13 mm, respectively. Post-hoc analysis confirmed that CIMT was significantly higher in all CAD categories compared to patients without CAD, and the difference between groups was statistically significant ($p < 0.001$). (Table 2) The Gensini score, which measures the angiographic severity of coronary artery disease, showed a high positive correlation with carotid intima-media thickness (CIMT). There was a higher association between the maximum CIMT and the Gensini score ($r = 0.66$, $p < 0.001$) than there was between the mean CIMT and the Gensini score ($p < 0.001$)**.** (Table 3) (Figure 1) CIMT was identified as an independent variable significantly associated with the severity of coronary artery disease in the multivariable linear regression analysis. A 5.8-point increase in the Gensini score was statistically linked to every 0.1 mm increase in CIMT ($p < 0.001$). Higher Gensini scores were also substantially correlated with increasing age ($p = 0.009$). Clinical risk variables independently linked to severe CAD included smoking ($p = 0.021$), diabetes mellitus ($p = 0.028$), and hypertension ($p = 0.019$). (Table 4) ROC curve analysis was used to evaluate the diagnostic efficacy of carotid intima-

media thickness (CIMT) for predicting obstructive coronary artery disease (CAD). As shown in the image, CIMT demonstrated strong discriminative ability to distinguish between patients with and without obstructive CAD, with an area under the curve (AUC) of 0.82. With a positive predictive value (PPV) of 89.1% and a negative predictive value (NPV) of 57.7%, the ideal cutoff value of 0.85 mm produced a sensitivity of 78.6% and a specificity of 75.0%. (Figure 2) CIMT was independently associated with the presence of obstructive coronary artery disease (=50% stenosis) in the logistic regression analysis. The risks of obstructive CAD increased 1.48 times for every 0.1 mm rise in CIMT ($p < 0.001$). Additionally, there was a significant correlation between age and the risks of obstructive CAD, with the odds rising by 5% for each extra year of age ($p = 0.046$). (Table 5)

Table 1. Baseline Demographic, Clinical, and Angiographic Characteristics of Study Participants (n = 65)

Variable	n (%) / Mean ± SD
Age (years)	56.2 ± 9.8
Sex	
Male	42 (64.6)
Female	23 (35.4)
BMI (kg/m²)	27.4 ± 3.9
Hypertension	
Yes	39 (60.0)
No	26 (40.0)
Diabetes Mellitus	
Yes	28 (43.1)
No	37 (56.9)
Dyslipidemia	
Yes	34 (52.3)
No	31 (47.7)
Current Smoker	
Yes	19 (29.2)
No	46 (70.8)
Family History of CAD	
Positive	21 (32.3)
Negative	44 (67.7)
Obstructive CAD (=50% stenosis)	
Present	49 (75.4)
Absent	16 (24.6)
Number of Vessels Involved	
None	16 (24.6)
Single-vessel	18 (27.7)
Two-vessel	15 (23.1)
Three-vessel	16 (24.6)
Gensini Score Median (IQR)	42 (28–68)

Table 2. Association of of CIMT across the Extent of Coronary Artery Disease

CAD Category	Mean CIMT (mm) ± SD	p-value*
No CAD (n=16)	0.68 ± 0.10	
Single-vessel (n=18)	0.82 ± 0.12	0.002
Two-vessel (n=15)	0.91 ± 0.11	<0.001
Three-vessel (n=16)	1.02 ± 0.13	<0.001

*ANOVA test used for comparison across groups. Post hoc analysis showed significant differences between the no-CAD group and each CAD group.

Table 3. Correlation between Carotid Intima-Media Thickness and Gensini Score

Variable	Correlation Coefficient (r)
Mean CIMT (mm) vs. Gensini score	0.61 (Pearson)
Maximum CIMT (mm) vs. Gensini score	0.66 (Pearson)

Figure 1: Scatter plot showing the correlation between mean carotid intima-media thickness (CIMT) and Gensini score.

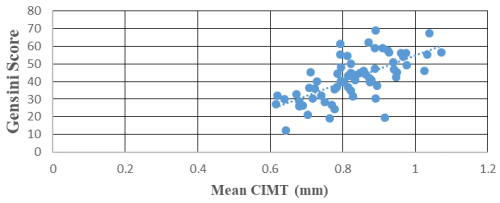


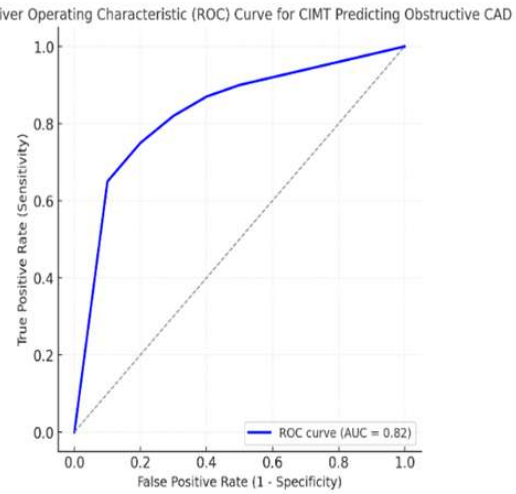
Table 4. Multivariable Linear Regression Analysis of Factors Associated with Gensini Score

Predictor Variable	â (95% CI)	p-value
CIMT (per 0.1 mm increase)	5.8 (3.1 – 8.5)	<0.001
Age (years)	0.42 (0.11 – 0.74)	0.009
Male (gender)	4.6 (–3.2 – 12.3)	0.24
Hypertension	7.1 (1.2 – 13.0)	0.019
Diabetes mellitus	6.4 (0.7 – 12.1)	0.028
Dyslipidemia	3.9 (–2.4 – 10.2)	0.22
Smoking	5.5 (0.9 – 10.1)	0.021

Table 5. Logistic Regression for CIMT as Predictor of Obstructive CAD (=50% stenosis)

Predictor Variable	Odds Ratio (OR)	95% CI	p-value
CIMT (per 0.1 mm increase)	1.48	1.21 – 1.80	<0.001
Age (per year)	1.05	1.00 – 1.10	0.046
Hypertension	1.82	0.74 – 4.50	0.19
Diabetes mellitus	2.05	0.84 – 5.01	0.11
Smoking	1.67	0.63 – 4.41	0.30

Figure 2: The ROC curve showing the diagnostic performance of CIMT in predicting obstructive CAD, with an AUC of 0.82



DISCUSSION

In this study, CIMT demonstrated a strong and independent association with the angiographic burden of coronary atherosclerosis. Mean CIMT correlated positively with the Gensini score (r = 0.61), and maximum CIMT correlated even more strongly (r = 0.66). Each 0.1 mm increment in CIMT was associated with a 5.8-point increase in Gensini score in multivariable analysis, and CIMT predicted obstructive CAD with an AUC of 0.82 (optimal cutoff 0.85 mm; sensitivity 78.6%, specificity 75.0%). These results indicate that higher carotid wall thickness closely parallels more extensive coronary disease in a population referred for coronary angiography.

Our findings are concordant with a number of contemporary studies that have reported meaningful associations between carotid arterial indices and coronary atherosclerotic burden. A prospective single-centre study by Verma and Katyal (2022) in a South Asian population found that mean CIMT was higher in patients with angiographically confirmed CAD and that CIMT independently predicted the presence of CAD, although their report suggested a weaker or inconsistent relationship with angiographic severity indices (Gensini and SYNTAX) in some analyses.[16] In contrast, several other cohorts have reported stronger, graded relationships between CIMT and severity measures: a multicentre series of patients reported higher mean CIMT in those with multivessel disease and showed moderate discriminatory performance (AUCs in the 0.70–0.80 range) for detecting significant coronary stenoses, findings that closely mirror our observed AUC of 0.82 and the stepwise increase in mean CIMT across none to single to two and to three-vessel disease in our data.[4, 17]

Differences between studies in effect sizes and diagnostic accuracy likely reflect methodological and population heterogeneity. Measurement protocols like mean versus maximum CIMT, site of measurement, common carotid vs bulb/internal carotid, sonographer experience, and whether carotid plaque was separately assessed are all important modifiers. Studies that incorporate plaque assessment or plaque burden commonly report that plaque variables add incremental predictive value and often outperform CIMT alone for detecting severe or complex coronary disease. For instance, population analyses and clinical cohorts from the last several years emphasize that focal carotid plaque, rather than diffuse intima-media thickening alone, may better reflect advanced atherosclerotic burden and improve agreement with coronary imaging. Our protocol recorded plaque presence but focused the primary analyses on CIMT. The relatively high AUC we observed suggests that in our sample, CIMT captured a substantial portion of systemic atherosclerotic burden, yet augmenting models with plaque metrics might further enhance discrimination, as other investigators have reported.[17, 18]

The magnitude of the association we observed (â = 5.8 Gensini points per 0.1 mm CIMT) is clinically interpretable: small measurable changes in arterial wall thickness correspond to meaningful differences in angiographic disease burden. This parallels the pattern reported in other mid-sized angiographic cohorts in which per-unit increases in CIMT were associated with higher coronary scores or greater odds of multivessel disease. Still, not all studies found identical results. Verma et al. 2022 reported that while CIMT predicted the presence of CAD, its correlation with severity scores was weak in their sample, an observation that cautions against universal generalization and highlights the role of sample composition, age distribution, and prevalence of risk factors.[16]

Our multivariable models also showed that traditional risk factors, age, hypertension, diabetes, and smoking, retained independent associations with angiographic severity, consistent with large contemporary cohorts. Importantly, CIMT remained an independent predictor even after adjusting for these covariates, supporting its added value beyond routine clinical risk markers. This supports the potential role of CIMT as a non-invasive adjunct to risk stratification in patients with intermediate clinical suspicion where the pretest probability of CAD is uncertain. Several recent studies recommend precisely this pragmatic use, combining carotid imaging with clinical and biochemical markers to improve patient selection for downstream testing.[17, 19, 20]

In practical terms, the present results add to the accumulating evidence that CIMT is a useful, widely available, low-cost imaging biomarker that correlates with the extent of coronary atherosclerosis. Our data suggest an optimal CIMT cutoff (0.85 mm) with good sensitivity and specificity for obstructive CAD in this referral population, a performance similar to several contemporary reports. Still, clinicians should interpret CIMT alongside clinical risk factors and, where available,

carotid plaque assessment or coronary CT imaging rather than as a standalone gatekeeper. Future research should focus on larger, multicentre cohorts with standardized CIMT and plaque protocols, combined prediction models that formally integrate CIMT, plaque burden, biomarkers, and clinical scores, and prospective studies assessing whether CIMT-guided diagnostic strategies improve patient-centred outcomes or reduce unnecessary invasive testing.

The results of this study demonstrate the significance of carotid intima-media thickness (CIMT), a straightforward, affordable, and non-invasive indicator that has a good correlation with the prevalence of coronary atherosclerosis. When deciding whether to do invasive coronary angiography on patients with intermediate cardiovascular risk, CIMT assessment may be used as an additional tool for risk stratification in standard clinical practice. The observed cutoff value of 0.85 mm, with good sensitivity and specificity, suggests that CIMT can help identify patients more likely to harbor significant coronary artery disease (CAD), thereby guiding earlier preventive interventions or more targeted diagnostic testing.

Furthermore, CIMT may provide additional value beyond established risk scores like Framingham or ASCVD calculators because it continues to be an independent predictor of CAD severity even after controlling for traditional risk factors. Incorporating CIMT into clinical algorithms could allow earlier identification of high-risk individuals who might otherwise be underestimated by traditional scoring systems alone. In addition, CIMT screening may be especially relevant in resource-limited settings where advanced imaging modalities such as coronary CT angiography or invasive angiography are not readily available.

A surrogate marker for evaluating the efficacy of preventive measures, including statin therapy, antihypertensives, and lifestyle changes, may also be provided by routine CIMT monitoring. However, CIMT should not be viewed as a replacement for established diagnostic modalities; rather, it should be integrated with clinical history, risk factor assessment, and, where indicated, imaging of carotid plaque or coronary arteries. In this way, CIMT can enhance early detection, improve patient selection for invasive testing, and ultimately contribute to reducing the burden of cardiovascular morbidity and mortality.

Limitations: There are limitations to consider when comparing our results to the literature. First, our sample size (n = 65) is modest compared with some larger cohorts; while our estimates were statistically robust, smaller samples are more susceptible to sampling variability and may overestimate effect sizes. Second, temporal proximity between CIMT measurement and angiography was controlled but not identical in every subject; although we limited the interval to two weeks, interval events or medical therapy changes could alter plaque dynamics. Third, measurement heterogeneity remains a universal caveat; different sonographers, ultrasound systems, and measurement protocols across studies complicate direct head-to-head comparisons. Finally, most prior studies and ours are observational and cross-sectional, limiting causal inference about progression; longitudinal studies that track CIMT change and progressive coronary plaque development would be most informative.

CONCLUSION:

Carotid intima-media thickness (CIMT) is shown in this study to be an independent predictor of angiographic severity in addition to being substantially correlated with the existence and severity of coronary artery disease (CAD). Its potential as a non-invasive proxy for the burden of coronary atherosclerosis is shown by the steady increase in CIMT readings across single-, double-, and triple-vessel disease. Crucially, CIMT is a useful clinical tool for CAD risk stratification and early diagnosis, as evidenced by the diagnostic accuracy indicated by an AUC. Routine cardiovascular risk assessment that includes CIMT monitoring may help identify high-risk individuals early, lessen the need for invasive procedures, and ultimately direct preventive measures to lessen the burden of coronary artery disease.

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Authors Contribution:
Jahanzeb Ibrahim: Intro, Literature Review, data collection, Data analysis.
Elishba Qazi: Literature Review, data collection, result.
Zuhoor Uddin: Literature Review, data collection, Data analysis.
Kalsoom Nawab: Review the article, Result and Data analysis.
Sahib Noor: Literature Review, Data Collection.
Laila Haleem: Data Collection, Data analysis.

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