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MODIFIABLE RISK FACTORS AFFECTING CORONARY ARTERY DISEASE SEVERITY AND ITS ASSESSMENT THROUGH CT CORONARY ANGIOGRAPHY

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Abstract

Background: *Coronary artery disease (CAD) has been ranked as a major cause of morbidity and mortality across the world, and its pathogenesis is propagated by modifiable risk factors. Controllable risks of diabetes, hypertension, smoking behaviour, cholesterol and obesity greatly influence the level of severity and the advancement of CAD.*

Objective: *The study aimed to assess CAD using CT coronary angiography (CTCA) and determine the influence of modifiable risk factors on CAD severity.*

Methodology: *This cross-sectional analytical study was carried out at the Army Cardiac Hospital, Lahore, including patients aged 18 years and above who presented with chest pain. A sample was selected using a non-probability convenient sampling design with a total of 246 patients, with the prevalence of CAD being 20 percent and a confidence interval of 95 percent. The CT coronary angiography was performed to diagnose CAD by using a 128-slice GE CT scan machine. Data was collected after developing the detailed proforma containing modifiable risk factors and CAD patterns. To get the association of risk factors and CAD severity, logistic regression and the Chi-square tests were performed in SPSS version 26.*

Results: *In this study, 174 males and 72 females, aged 21-80 years, presented with chest pain. 130 out of a total of 246 participants had a family history. The results indicated that there was a significant correlation of diabetes (p = 0.039) and hypertension (p = <0.001) with the presence of CAD. Logistic regression analysis shows the strongest predictors of CAD were smoking and hypertension, having p values of 0.056 and <0.001, respectively. The statistically significant relationships with lesion severity and plaque burden were found for diabetes mellitus (p values 0.010, 0.008), hypertension (p values <0.001, <0.001) and total cholesterol (p = 0.001, <0.001), respectively. Vessel calcium scoring was strongly associated with diabetes and hypertension, with p-values of 0.008 and 0.034 in the given order. CAD RADS scoring was significantly associated with plaque burden (p = <0.001) in CTCA.*

Conclusion: *The study highlights significant associations between coronary artery disease and key risk factors, including diabetes, hypertension, smoking, and elevated cholesterol levels. Hypertension and smoking emerged as the strongest predictors of CAD. Plaque burden and lesion severity were notably influenced by these factors. CT coronary angiography proved effective in assessing disease severity and risk.*

Keywords: *CT coronary angiography, Coronary artery disease, Dyslipidaemia, Hypertension, Modifiable risk factors, Plaque burden, Smoking*

INTRODUCTION

The leading cause of mortality and morbidity in the world is Coronary Artery Disease (CAD), which is mostly caused by atherosclerosis. Computed tomography angiography (CTA) has become an important and effective tool for non-invasive diagnosis, through which abnormalities in the coronary arteries can be detected effectively (1). CT angiography can give visualization of coronary artery structures, detailed information about where the plaque is located and how much of an artery is narrowed due to plaque, hence facilitating early detection of CAD without the insertion of a catheter as in traditional angiography (2).

Diagnosis and prevention of CAD are based on the knowledge of modifiable risk factors, which include diabetes, hypertension, dyslipidaemia, obesity, and smoking. Several studies have shown that modification of lifestyle, such as routine exercise, can decrease the risk of calcification of coronary arteries and enhance plaque structure (3). CTCA is significant in measuring coronary artery calcification, plaque characteristics, and obstructive and non-obstructive atherosclerosis so that medical professionals can assess the cardiovascular risk and prevent it. Cardiovascular diseases (CVDs) present a national health issue in Pakistan, with prevalence and soaring death rates. In a survey conducted recently, 18.9% of the population stated to had CVDs (4). The severity of CAD can be determined with the help of CTCA-based imaging, relieving the threat of the acute coronary event. It has also been found that high cholesterol level is a risk factor for plaque rupture, as high levels of cholesterol result in the formation of non-calcified plaque, and therefore, intervention and interventional procedures and use of medications are needed to reduce lipid levels (5).

CTCA is not only useful in the diagnosis of CAD, but it is also useful to define the risk, especially of those individuals with cardiovascular risk factors that can be modified, like high blood pressure and diabetes. It assists in early pathophysiological alterations, like stiffness of arteries and endothelial dysfunction, which are also linked to faster atherogenesis (6). These early detections allow healthcare professionals to initiate the right interventions early enough and improve cardiovascular outcomes. According to the World Health Organization (WHO), CAD is a leading cause of death in the world as it is the cause of about 30% of world deaths in terms of burden, and a lot is experienced in low- and middle-income countries such as Pakistan. CTCA presents a life-saving method, which can identify CAD in its early stage and allow taking countermeasures before the manifestation of the morbidity and mortality (7). However, considerable numbers of studies should be conducted regarding the population-specific strategies to increase CTCA use in areas with insufficient healthcare resources.

Atherosclerosis, the leading cause of CAD, is silent in most cases and has no obvious symptoms. CTCA plays a crucial role in the early detection of subclinical conditions, helping to prevent the progression of CAD. Studies show that 20-30% of individuals with non-obstructive CAD have plaques that can lead to severe, irreversible damage if the condition remains untreated. Early intervention is vital to halt the disease's progression and avoid irreversible harm (8). With the help of CTCA, such plaques can be identified, and timely preventive measures, i.e, lifestyle changes, medications, or early interventions can be done. Other recent developments in CTCA technology have sought to engage inflammation as one of the most important factors that lead to CAD. The volume of perivascular fat, which covers the coronary arteries, is a risk contributor to acute coronary events. Choosing CTCA will allow recognizing this fat that will give clinicians a new source to evaluate residual risk and enhance the management of CAD (9).

Scientific investigations on CAD risk factors through CTCA imaging have not fully established the direct cause-and-effect relationships that exist between risk factors and coronary plaque development patterns. The epidemiology of CAD feature distribution as revealed by angiography, specifically with the use of computed tomography angiography (CTCA), has not been checked out completely (10). Considering the possibility of varying differences in disease presentation and risks factor interactions, this study will give insights into the angiographic data of CAD in the Pakistani population and provide insight into the influence of modifiable risk factors on the severity of the disease. This study aims to analyse the features of coronary artery disease using CTCA and the severity of disease influenced by modifiable risk factors.

MATERIALS AND METHODS

In this study, a cross-sectional analytical design was utilized in the examination of the prevalence and severity of coronary artery disease (CAD) with the help of CT coronary angiography (CTCA). The Army Cardiac Hospital Lahore, being a well-established clinical setup dealing with cardiac care, was used to collect the data. The sample size was determined by using the result of the prevalence of CAD, which is reported to be around 20% among the general population. The formula for sample size was:

$$n = \frac{Z_{1-\alpha}^{2} P(1-P)}{d^{2}}$$

$Z(1-\alpha/2) = 1.96$ at a 95% confidence interval (11)

The participants were selected with the use of non-probability convenience sampling. The research was carried out in the period of 6 months and started following the acceptance of the synopsis of the study.

The study involved participants who were more than 18 years old and who were seeking hospital care because of chest pains. These persons were made aware of the rationale of the study, and written consent was taken from all the participants. Chest pain in patients was an included study criterion. Exclusion criteria were persons with renal impairment (glomerular filtration rate (GFR) <30ml/min), pregnant and persons with a known contraindication to contrast, where there is the possibility of confounding of results or providing an additional risk during the imaging procedure.

Several steps were prepared for data collection. Patients were advised to fast for 4 to 6 hours before the procedure, and patients with a normal creatinine level (less than 1.2 mg/dl) were said to qualify to be included. Data acquisition was performed using a 128-slice GE CT scanner. Patients lay in a supine position, and a localizer scan was obtained to outline the area of interest. A non-contrast scan was first undertaken to discover any plaques that had been calcified. After that, a non-ionic low-osmolar contrast agent was intravenously injected (by a power injector) at 4-6 ml/sec to make visualizing the coronary vasculature easier. The amount, extent, and nature of CAD were determined in terms of the known standards on coronary artery disease. An elaborate proforma was developed to capture different aspects affecting the development of CAD. This consisted of the demographics and the modifiable risk factors such as smoking, hypertension, diabetes mellitus, dyslipidemia, and obesity. The proforma also recorded the coronary angiographic characteristics, such as the plaque morphology, lesion severity, and plaque burden, which play an important role in comprehending the level of CAD.

Analysis of the collected data was done on the Statistical Package for Social Sciences (SPSS) version 26. Quantitative data that summarised the size of sample differences, age, and employed descriptive statistics of mean and standard deviation. Qualitative data were presented in terms of frequency and percentage. The chi-square test was used to evaluate relationships between categorical variables, and logistic regression analysis was used to highlight important risk factors that influence the presence of CAD. All statistical tests were conducted at the 5% level of significance, which proves the strength of the results and their possible course of action in clinical practice.

RESULTS

This cross-sectional analytical study was conducted at the Army Cardiac Hospital, Lahore, to evaluate the association between modifiable risk factors and the severity of coronary artery disease (CAD) in patients presenting with chest pain. A total of 246 patients were enrolled, comprising 174 males and 72 females, with an age range of 21 to 80 years. CT coronary angiography (CTCA) was employed as the primary diagnostic modality to assess coronary artery involvement.

Of the total participants, 130 (52.8%) reported a positive family history of CAD. CTCA findings revealed that 210 patients (85.4%) demonstrated radiological evidence of CAD. The study further explored correlations between modifiable lifestyle and medical risk factors—including smoking, hypertension, diabetes, dyslipidemia, and obesity—and the presence and severity of CAD as assessed by plaque burden, lesion severity, and calcium scoring..

Fig. 1 illustrates the frequency distribution of key modifiable risk factors among the study participants. The majority of individuals were non-diabetic (65.4%), non-hypertensive (58.1%), non-smokers (70.3%), and exhibited normal total cholesterol levels (80.5%). However, a notable proportion of the cohort (43.1%) was found to have dyslipidemia and were classified as overweight based on BMI criteria. These findings underscore the heterogeneity of cardiovascular risk profiles within the study population.

The analysis demonstrates a statistically significant association between diabetes mellitus and the presence of coronary artery disease (CAD), with 37.1% of diabetic individuals exhibiting CAD ($p = 0.039$). Similarly, hypertension shows a strong and highly significant correlation with CAD, as 63.8% of hypertensive patients were found to have the disease ($p < 0.001$). In contrast, the relationship between smoking status and CAD was not statistically significant ($p = 0.156$), despite 68.6% of non-smokers and 13.8% of current smokers being affected. Other modifiable risk factors, including elevated total cholesterol, dyslipidemia, and increased body mass index (BMI), did not show statistically significant associations with

CAD, with p-values of 0.406, 0.382, and 0.885 respectively, suggesting a weaker or non-existent correlation in this cohort (Table I).

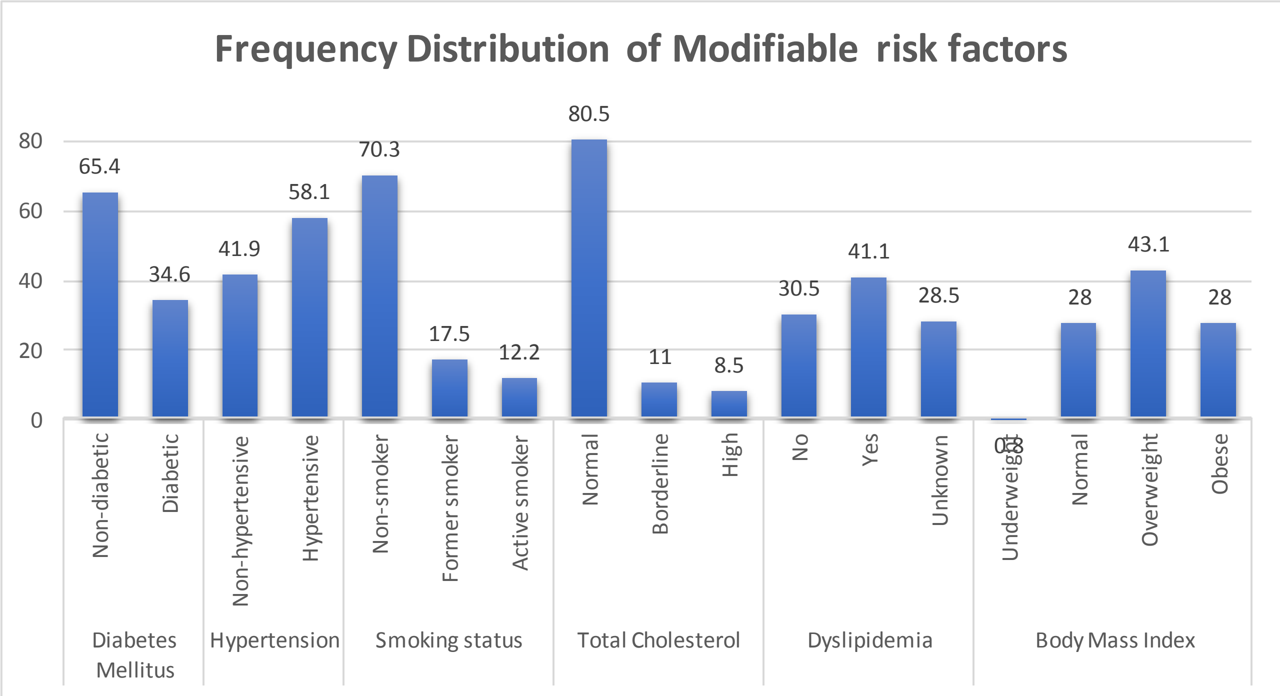

Fig. 1. Frequency distribution of modifiable risk factors

Table I. Association between modifiable risk factors and presence of CAD

Modifiable risk factors	Categories	Presence of CAD n (%)		Total	Chi square	P value
		Absent	Present			
Diabetes Mellitus	Non diabetic	29(80.6)	132(62.9)	161(65.4)	4.257	0.039*
	Diabetic	7(19.4)	78(37.1)	85(34.6)		
Hypertension	Non-hypertensive	27(75.0)	76(36.2)	103(41.9)	19.018	<0.001*
	Hypertensive	9(25.0)	134(63.8)	143(58.1)		
Smoking Status	Non-smoker	29(80.6)	144(68.6)	173(70.3)	3.710	0.156
	Former smoker	6(16.7)	37(17.6)	43(17.5)		
	Active smoker	1(2.8)	29(13.8)	30(12.2)		
Total Cholesterol	Normal	31(86.1)	167(79.5)	198(80.5)	1.805	0.406
	Borderline	4(11.1)	23(11.0)	27(11.0)		
	High	1(2.8)	20(9.5)	21(8.5)		
Dyslipidemia	No	13(36.1)	62(29.5)	75(30.5)	1.923	0.382
	Yes	11(30.6)	90(42.9)	101(41.1)		
	Unknown	12(33.3)	58(27.6)	70(28.5)		
Body Mass Index	Underweight	0(0.0)	2(1.0)	2(0.8)	0.651	0.885
	Normal	10(27.8)	59(28.1)	69(28.0)		
	Overweight	17(47.2)	89(42.4)	106(43.1)		
	Obese	9(25.0)	60(28.6)	69(28.0)		
Total		36(100)	210(100)	246(100)		

The distribution of coronary artery disease (CAD) across the major coronary vessels reveals that the left anterior descending (LAD) artery is the most frequently affected vessel, with 78 patients demonstrating significant involvement. Additionally, 38 patients exhibited multi-vessel disease, indicating the presence of CAD in more than one coronary artery. In comparison, the right coronary artery (RCA) and left circumflex artery (LCx) showed lower frequencies of involvement, suggesting a more localized pattern of atherosclerotic burden. This vessel-specific distribution is visually summarized in Fig.2.

The analysis of clinical features and associated risk factors in patients with coronary artery disease (CAD) demonstrates that single-vessel stenosis is the most prevalent angiographic finding, whereas double- and triple-vessel involvement occurs less frequently. Regarding plaque morphology, calcified plaques were the most commonly observed, followed by soft and mixed plaques, highlighting their significant role in the pathogenesis of CAD. Lesion severity was predominantly mild, with most cases falling within the 25–49% stenosis range. Furthermore, plaque burden assessment revealed a predominance of mild plaque burden in 149 patients (60.6%), with progressively fewer cases classified as moderate, severe, and extensive. These findings are illustrated in Fig. 3.

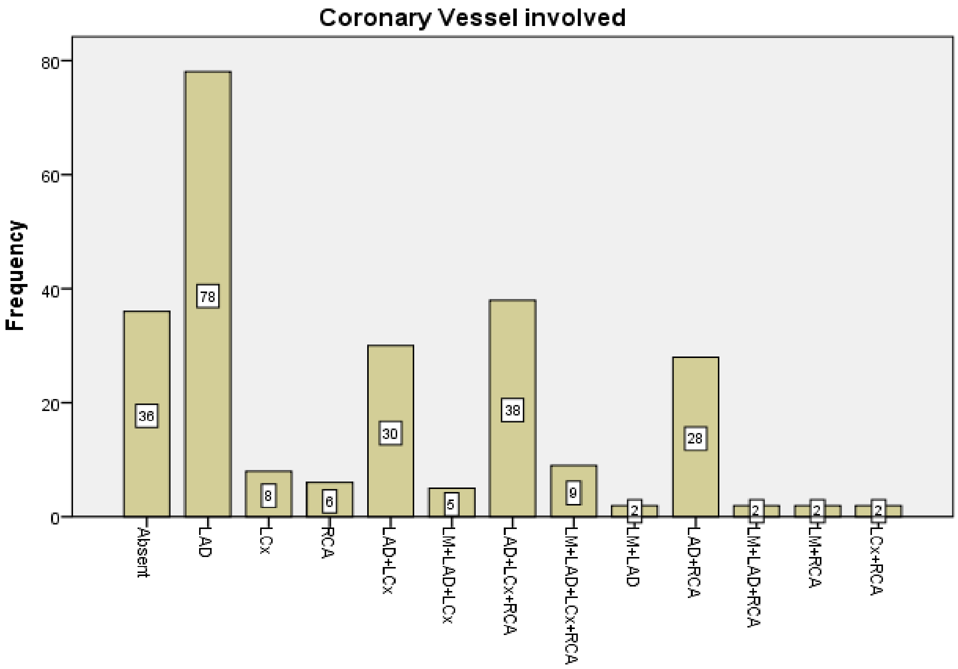

Fig. 2. Involvement of coronary vessel

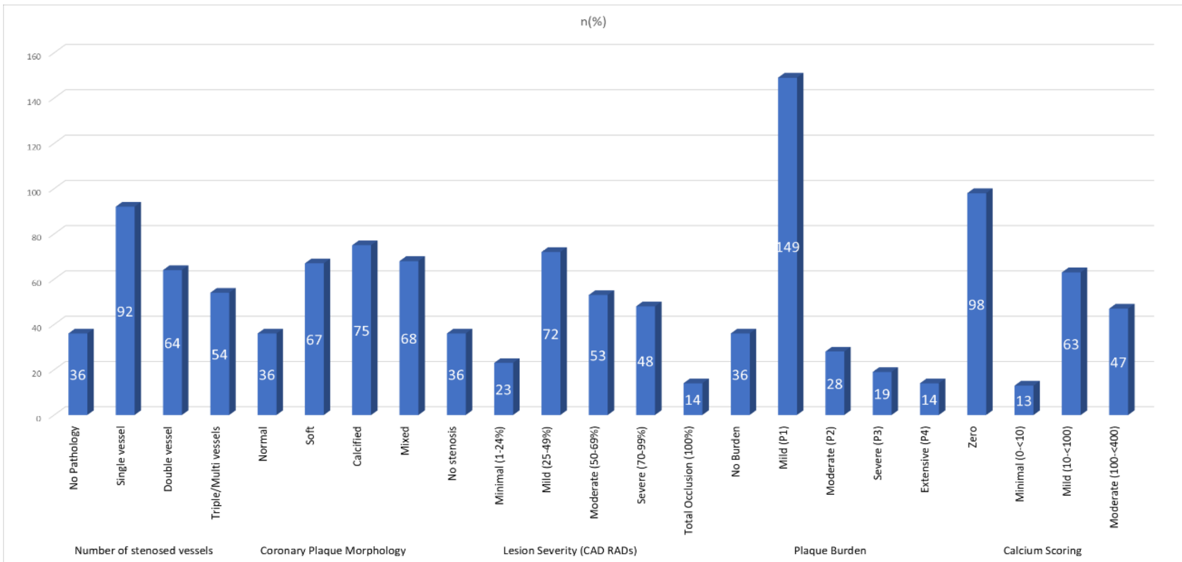

Fig. 3. CAD clinical features and risk factors

The logistic regression analysis identifies key modifiable risk factors associated with the presence of coronary artery disease (CAD). Among these, hypertension and smoking status emerge as the most significant predictors, with hypertension demonstrating a highly significant association ($p < 0.001$) and smoking showing a borderline significance ($p = 0.056$). In contrast, diabetes mellitus, total cholesterol levels, and dyslipidaemia exhibit weaker associations with CAD, as reflected by p-values greater than 0.05, indicating insufficient statistical significance. Furthermore, the body mass index (BMI) categories—ranging from underweight to obese—are characterized by minimal standard errors and an overall non-significant p-value ($p = 0.999$), suggesting that BMI may have a limited role in predicting CAD severity in this cohort. These findings are detailed in Table II.

Table II. Logistic regression analysis of factors associated with coronary artery disease presence

Variables in the Equation								
Variables	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
							Lower	Upper

Diabetes Mellitus (Diabetic)	0.734	0.475	2.383	1	0.123	2.0830	0.820	5.289
Hypertension (Hypertensive)	1.889	0.445	18.053	1	<0.001*	6.615	2.767	15.814
Smoking status (Non-smokers)			3.724	2	0.155			
Former Smokers	0.226	0.521	0.189	1	0.664	1.254	0.452	3.479
Active Smokers	2.058	1.079	3.639	1	0.056*	7.830	0.945	64.881
Total Cholesterol (Normal)			1.092	2	0.579			
Borderline	-0.140	0.647	0.047	1	0.829	0.869	0.245	3.088
High	1.099	1.103	0.993	1	0.319	3.002	0.346	26.081
Dyslipidemia (No)			1.679	2	0.432			
Yes	0.436	0.495	0.776	1	0.378	1.547	0.586	4.081
Unknown	-0.212	0.488	0.189	1	0.663	0.809	0.311	2.105
BMI			2.508	3	0.474			
BMI (Normal)	-17.962	28263.693	0.000	1	0.999	0.000	0.000	.
Overweight	-18.658	28263.693	0.000	1	0.999	0.000	0.000	.
Obese	-18.057	28263.693	0.000	1	0.999	0.000	0.000	.
Constant	18.703	28263.693	0.000	1	0.999	132637826.593		

The relationship between various modifiable risk factors and the extent of coronary artery stenosis in patients with coronary artery disease (CAD) was assessed. Among non-diabetic individuals, single-vessel involvement was more prevalent, whereas diabetic patients demonstrated a higher proportion of triple or multivessel disease; however, this difference did not reach statistical significance (p = 0.073). A strong and statistically significant association was observed between hypertension and elevated total cholesterol levels with severe CAD, as both factors were linked to a higher prevalence of double and triple-vessel disease (p < 0.001 and p = 0.008, respectively). In contrast, smoking status, dyslipidaemia, and body mass index (BMI) categories showed no statistically meaningful relationship with the number of affected vessels, with p-values of 0.085, 0.422, and 0.827, respectively. These results suggest that hypertension and hypercholesterolemia are the most reliable predictors of extensive coronary artery involvement. The detailed distribution is provided in Table III.

Table III. Association of modifiable risk factors with no. of stenosed vessels

Modifiable risk factors	**Categories**	**No. of stenosed vessels n (%)**				**Total**	**Chi Square**	**P Value**
		No pathology	**Single vessel**	**Double vessel**	**Triple/Multi vessel**			
Diabetes Mellitus	Non-diabetic	29 (80.6)	61 (66.3)	42 (65.6)	29 (53.7)	161 (65.4)	6.958	0.073
	Diabetic	7 (19.4)	31 (33.7)	22 (34.4)	25 (46.3)	85 (34.6)		
Hypertension	Non-hypertensive	27 (75.0)	41 (44.6)	21 (32.8)	14 (25.9)	103 (41.9)	24.307	<0.001
	Hypertensive	9 (25.0)	51 (55.4)	43 (67.2)	40 (74.1)	143 (58.1)		
Smoking Status	Non-smoker	29 (80.6)	65 (70.7)	43 (67.2)	36 (66.7)	173 (70.3)	11.103	0.085
	Former smoker	6 (16.7)	10 (10.9)	15 (23.4)	12 (22.2)	43 (17.5)		
	Active smoker	1 (2.8)	17 (18.5)	6 (9.4)	6 (11.1)	30 (12.2)		

Total Cholesterol	Normal	31 (86.1)	77 (83.7)	56 (87.5)	34 (63.0)	198 (80.5)	17.294	0.008
	Borderline	4 (11.1)	10 (10.9)	4 (6.2)	9 (16.7)	27 (11.0)		
	High	1 (2.8)	5 (5.4)	4 (6.2)	11 (20.4)	21 (8.5)		
Dyslipidemia	No	13 (36.1)	29 (31.5)	22 (34.4)	11 (20.4)	75 (30.5)	6.009	0.422
	Yes	11 (30.6)	39 (42.4)	23 (35.9)	28 (51.9)	101 (41.1)		
	Unknown	12 (33.3)	24 (26.1)	19 (29.7)	15 (27.8)	70 (28.5)		
Body Mass Index	Underweight	0 (0.0)	0 (0.0)	1 (1.6)	1 (1.9)	2 (0.8)	5.083	0.827
	Normal	10 (27.8)	29 (31.5)	17 (26.6)	13 (24.1)	69 (28.0)		
	Overweight	17 (47.2)	35 (38.0)	31 (48.4)	23 (42.6)	106 (43.1)		
	Obese	9 (25.0)	28 (30.4)	15 (23.4)	17 (31.5)	69 (28.0)		
Total		36 (100)		210 (100)		246 (100)		

The association between modifiable risk factors and coronary plaque morphology was evaluated in patients with coronary artery disease (CAD). A statistically significant relationship was observed between hypertension and plaque morphology, with hypertensive individuals showing a notably higher prevalence (70%) of calcified and mixed plaques ($p < 0.001$). In contrast, although diabetic participants exhibited a greater tendency toward mixed plaques, this association did not reach statistical significance ($p = 0.063$). Furthermore, no meaningful associations were found between plaque morphology and smoking status ($p = 0.301$), dyslipidaemia ($p = 0.427$), total cholesterol levels ($p = 0.070$), or body mass index (BMI) ($p = 0.525$), indicating a limited role of these variables in determining plaque composition. Further details are presented in Table IV.

Table IV. Association of modifiable risk factors with coronary plaque morphology

Modifiable risk factors	Categories	Coronary plaque morphology n (%)				Total	Chi Square	P Value
		Normal	Soft	Calcified	Mixed			
Diabetes Mellitus	Non-diabetic	29 (80.6)	47 (70.1)	47 (62.7)	38 (55.9)	161 (65.4)	7.296	0.063
	Diabetic	7 (19.4)	20 (29.9)	28 (37.3)	30 (44.1)	85 (34.6)		
Hypertension	Non-hypertensive	27 (75.0)	28 (41.8)	29 (38.7)	19 (27.9)	103 (41.9)	21.971	<0.001
	Hypertensive	9 (25.0)	39 (58.2)	46 (61.3)	49 (72.1)	143 (58.1)		
Smoking Status	Non-smoker	29 (80.6)	47 (70.1)	50 (66.7)	47 (69.1)	173 (70.3)	7.225	0.301
	Former smoker	6 (16.7)	8 (11.9)	15 (20.0)	14 (20.6)	43 (17.5)		
	Active smoker	1 (2.8)	12 (17.9)	10 (13.3)	7 (10.3)	30 (12.2)		
Total Cholesterol	Normal	31 (86.1)	57 (85.1)	63 (84.0)	47 (69.1)	198 (80.5)	11.652	0.070
	Borderline	4 (11.1)	7 (10.4)	7 (9.3)	9 (13.2)	27 (11.0)		
	High	1 (2.8)	3 (4.5)	5 (6.7)	12 (17.6)	21 (8.5)		
Dyslipidemia	No	13 (36.1)	16 (23.9)	26 (34.7)	20 (29.4)	75 (30.5)	5.968	0.427
	Yes	11 (30.6)	28 (41.8)	29 (38.7)	33 (48.5)	101 (41.1)		
	Unknown	12 (33.3)	23	20	15	70 (28.5)		

			(34.3)	(26.7)	(22.1)			
Body Mass Index	Underweight	0 (0.0)	0 (0.0)	0 (0.0)	2 (2.9)	2 (0.8)	8.092	0.525
	Normal	10 (27.8)	19 (28.4)	24 (32.0)	16 (23.5)	69 (28.0)		
	Overweight	17 (47.2)	30 (44.8)	27 (36.0)	32 (47.1)	106 (43.1)		
	Obese	9 (25.0)	18 (26.9)	24 (32.0)	18 (26.5)	69 (28.0)		
Total		36 (100)		210 (100)		246 (100)		

The correlation between the severity of coronary artery disease (CAD) lesions and various modifiable risk factors was analyzed to determine potential predictors of disease progression. A statistically significant association was observed with diabetes mellitus ($p = 0.010$), hypertension ($p < 0.001$), and elevated total cholesterol levels ($p = 0.001$), indicating that these factors are closely linked to increased lesion severity. In contrast, smoking status did not show a significant relationship ($p = 0.587$), as lesion severity appeared relatively consistent across non-smokers, former smokers, and current smokers. Similarly, dyslipidaemia ($p = 0.562$) and body mass index (BMI) categories ($p = 0.405$) did not demonstrate significant correlations with lesion severity. The comprehensive findings are summarized in Table V.

Table V. Associtaion of modifiable risk factors with lesion severity

Modifiable Risk factors	Categories	Lesion severity n (%)						Total	Chi Square	P Value
		No stenosis	Min (1-24%)	Mild (25-49%)	Moderate (50-69%)	Severe (70-99%)	T.O (100%)			
Diabetes Mellitus	Non-diabetic	29 (80.6)	13 (56.5)	48 (66.7)	40 (75.5)	26 (54.2)	5 (35.7)	161 (65.4)	15.021	0.010
	Diabetic	7 (19.4)	10 (43.5)	24 (33.3)	13 (24.5)	22 (45.8)	9 (64.3)	85 (34.6)		
Hypertension	Non-hypertensive	27 (75.0)	12 (52.2)	29 (40.3)	20 (37.7)	11 (22.9)	4 (28.6)	103 (41.9)	25.787	<0.001
	Hypertensive	9 (25.0)	11 (47.8)	43 (59.7)	33 (62.3)	37 (77.1)	10 (71.4)	143 (58.1)		
Smoking Status	Non-smoker	29 (80.6)	17 (73.9)	50 (69.4)	36 (67.9)	29 60.4)	12 (85.7)	173 (70.3)	8.425	0.587
	Former smoker	6 (16.7)	3 (13.0)	14 (19.4)	9 (17.0)	10 (20.8)	1 (7.1)	43 (17.5)		
	Active smoker	1 (2.8)	3 (13.0)	8 (11.1)	8 (15.1)	9 (18.8)	1 (7.1)	30 (12.2)		
Total Cholesterol	Normal	31 (86.1)	20 (87.0)	61 (84.7)	44 (83.0)	33 (68.8)	9 (64.3)	198 (80.5)	30.759	0.001
	Borderline	4 (11.1)	1 (4.3)	9 (12.5)	2 (3.8)	11 (22.9)	0 (0.0)	27 (11.0)		
	High	1 (2.8)	2 (8.7)	2 (2.8)	7 (13.2)	4 (8.3)	5 35.7)	21 (8.5)		
Dyslipidemia	No	13 (36.1)	7 (30.4)	23 (31.9)	12 (22.6)	13 (27.1)	7 (50.0)	75 (30.5)	8.682	0.562
	Yes	11 (30.6)	9 (39.1)	31 (43.1)	22 (41.5)	22 (45.8)	6 (42.9)	101 (41.1)		
	Unknown	12 (33.3)	7 (30.4)	18 (25.0)	19 (35.8)	13 (27.1)	1 (7.1)	70 (28.5)		
Body Mass Index	Underweight	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.9)	1 (2.1)	0 (0.0)	2 (0.8)	15.653	0.405
	Normal	10 (27.8)	7 (30.4)	15 (20.8)	22 (41.5)	12 (25.0)	3 (21.4)	69 (28.0)		
	Overweight	17 (47.2)	9 (39.1)	32 (44.4)	20 (37.7)	24 (50.0)	4 (28.6)	106 (43.1)		
	Obese	9	7	25	10	11	7	69		

	(25.0)	(30.4)	(34.7)	(18.9)	(22.9)	(50.0)	(28.0)
Total	36 (100)			210 (100)			246 (100)

An analysis of the association between modifiable risk factors and plaque burden among patients with coronary artery disease (CAD) highlights several significant relationships. Diabetic patients exhibited a markedly higher prevalence of severe and extensive plaque burden compared to non-diabetics, with a statistically significant p-value of 0.008. Hypertension and elevated total cholesterol levels were also significantly associated with increased plaque burden (both p < 0.001), reinforcing their role in disease progression. In contrast, smoking status and dyslipidaemia did not show significant associations with plaque burden (p = 0.492 and p = 0.341, respectively). Similarly, body mass index (BMI) categories were not significantly correlated with the extent of plaque accumulation (p = 0.747). Detailed results are presented in Table VI.

Table VI. Association of modifiable risk factors with plaque burden

Modifiable Risk Factors	Categories	Plaque Burden n (%)					Total	Chi Square	P Value
		No Burden	Mild (P1)	Moderate (P2)	Severe (P3)	Extensive (P4)			
Diabetes Mellitus	Non-diabetic	29 (80.6)	96 (64.4)	21 (75.0)	11 (57.9)	4 (28.6)	161 (65.4)	13.730	0.008
	Diabetic	7 (19.4)	53 (35.6)	7 (25.0)	8 (42.1)	10 (71.4)	85 (34.6)		
Hypertension	Non-hypertensive	27 (75.0)	60 (40.3)	6 (21.4)	7 (36.8)	3 (21.4)	103 (41.9)	23.800	<0.001
	Hypertensive	9 (25.0)	89 (59.7)	22 (78.6)	12 (63.2)	11 (78.6)	143 (58.1)		
Smoking Status	Non-smoker	29 (80.6)	104 (69.8)	19 (67.9)	11 (57.9)	10 (71.4)	173 (70.3)	7.417	0.492
	Former smoker	6 (16.7)	23 (15.4)	6 (21.4)	6 (31.6)	2 (14.3)	43 (17.5)		
	Active smoker	1 (2.8)	22 (14.8)	3 (10.7)	2 (10.5)	2 (14.3)	30 (12.2)		
Total Cholesterol	Normal	31 (86.1)	127 (85.2)	20 (71.4)	12 (63.2)	8 (57.1)	198 (80.5)	34.451	<0.001
	Borderline	4 (11.1)	15 (10.1)	3 (10.7)	5 (26.3)	0 (0.0)	27 (11.0)		
	High	1 (2.8)	7 (4.7)	5 (17.9)	2 (10.5)	6 (42.9)	21 (8.5)		
Dyslipidemia	No	13 (36.1)	46 (30.9)	5 (17.9)	4 (21.1)	7 (50.0)	75 (30.5)	9.011	0.341
	Yes	11 (30.6)	60 (40.3)	15 (53.6)	9 (47.4)	6 (42.9)	101 (41.1)		
	Unknown	12 (33.3)	43 (28.9)	8 (28.6)	6 (31.6)	1 (7.1)	70 (28.5)		
Body Mass Index	Underweight	0 (0.0)	2 (1.3)	0 (0.0)	0 (0.0)	0 (0.0)	2 (0.8)	8.470	0.747
	Normal	10 (27.8)	44 (29.5)	9 (32.1)	3 (15.8)	3 (21.4)	69 (28.0)		
	Overweight	17 (47.2)	62 (41.6)	11 (39.3)	12 (63.2)	4 (28.6)	106 (43.1)		
	Obese	9 (25.0)	41 (27.5)	8 (28.6)	4 (21.1)	7 (50.0)	69 (28.0)		
Total		36 (100)		210 (100)			246 (100)		

The findings presented in Table VII highlight the association between modifiable risk factors and coronary vessel calcium scores, ranging from zero (no calcification) to extensive (≥1000). Diabetes mellitus and hypertension demonstrate statistically significant correlations with calcium scoring, with p-values of 0.008 and 0.034, respectively. These results suggest that individuals with these conditions are more likely to exhibit moderate to severe vascular calcification, contributing to increased plaque deposition and arterial stiffness. While elevated cholesterol, dyslipidaemia, smoking status, and higher BMI categories are observed to have a tendency toward greater calcium scores, these associations are not statistically significant.

Table VII. Association of modifiable risk factors with calcium score in vessels

Modifiable risk factors	Categories	Calcium Score in vessels n (%)						Total	Chi Square	P Value
		Zero	Minimal (0-<10)	Mild (10-<100)	Moderate (100-<400)	Severe (400-<1000)	Extensive (≥1000)			
Diabetes Mellitus	Non-diabetic	74 (75.5)	9 (69.2)	41 (65.1)	28 (59.6)	8 (40.0)	1 (20.0)	161 (65.4)	15.485	0.008
	Diabetic	24 (24.5)	4 (30.8)	22 (34.9)	19 (40.4)	12 (60.0)	4 (80.0)	85 (34.6)		
Hypertension	Non-hypertensive	52 (53.1)	5 (38.5)	24 (38.1)	17 (36.2)	3 (15.0)	2 (40.0)	103 (41.9)	12.041	0.034
	Hypertensive	46 (46.9)	8 (61.5)	39 (61.9)	30 (63.8)	17 (85.0)	3 (60.0)	143 (58.1)		
Smoking Status	Non-smoker	73 (74.5)	10 (76.9)	43 (68.3)	31 (66.0)	14 (70.0)	2 (40.0)	173 (70.3)	11.637	0.310
	Former smoker	13 (13.3)	2 (15.4)	10 (15.9)	11 (23.4)	6 (30.0)	1 (20.0)	43 (17.5)		
	Active smoker	12 (12.2)	1 (7.7)	10 (15.9)	5 (10.6)	0 (0.0)	2 (40.0)	30 (12.2)		
Total Cholesterol	Normal	84 (85.7)	11 (84.6)	48 (76.2)	38 (80.9)	14 (70.0)	3 (60.0)	198 (80.5)	16.363	0.90
	Borderline	10 (10.2)	0 (0.0)	10 (15.9)	5 (10.6)	2 (10.0)	0 (0.0)	27 (11.0)		
	High	4 (4.1)	2 (15.4)	5 (7.9)	4 (8.5)	4 (20.0)	2 (40.0)	21 (8.5)		
Dyslipidemia	No	28 (28.6)	7 (53.8)	17 (27.0)	12 (25.5)	8 (40.0)	3 (60.0)	75 (30.5)	16.305	0.091
	Yes	37 (37.8)	5 (38.5)	32 (50.8)	16 (34.0)	9 (45.0)	2 (40.0)	101 (41.1)		
	Unknown	33 (33.7)	1 (7.7)	14 (22.2)	19 (40.4)	3 (15.0)	0 (0.0)	70 (28.5)		
Body Mass Index	Underweight	0 (0.0)	0 (0.0)	1 (1.6)	1 (2.1)	0 (0.0)	0 (0.0)	2 (0.8)	12.481	0.642
	Normal	27 (27.6)	5 (38.5)	23 (36.5)	10 (21.3)	3 (15.0)	1 (20.0)	69 (28.0)		
	Overweight	46 (46.9)	4 (30.8)	23 (36.5)	23 (48.9)	9 (45.0)	1 (20.0)	106 (43.1)		
	Obese	25 (25.5)	4 (30.8)	16 (25.4)	13 (27.7)	8 (40.0)	3 (60.0)	69 (28.0)		
Total		98 (100)			148 (100)			246 (100)		

Table VIII illustrates a statistically significant association between lesion severity (CAD RADs) and plaque burden, supported by a Pearson Chi-Square value of 410.547 and a p-value of <0.001. Among the patients, 36 had no plaque burden, corresponding to 0% stenosis. In the minimal stenosis category (1–24%), 15.4% of patients exhibited mild plaque burden. For mild stenosis (25–49%), a majority of patients (66) showed mild plaque burden, with a few having moderate plaque. In the moderate stenosis group (50–69%), most patients (39) had mild plaque, while a substantial portion (12) had moderate plaque levels. Severe stenosis (70–99%) was predominantly associated with severe plaque burden, though 13 patients in this group exhibited moderate plaque as well. In the total occlusion category (100%), 9 patients had extensive plaque, and the remainder had either moderate or severe plaque burdens. These findings indicate that as lesion severity increases, the extent of plaque burden also tends to rise significantly.

Table VIII. Association between lesion severity (CAD RADS) and plaque burden

Lesion Severity (CAD RADS)	Plaque Burden					Total	Chi square	P value
	No Burden	Mild (P1)	Moderate (P2)	Severe (P3)	Extensive (P4)			
No stenosis (0%)	36 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	36 (14.6)	410.547[a]	<0.001
Minimal (1-24%)	0 (0.0)	23 (15.4)	0 (0.0)	0 (0.0)	0 (0.0)	23 (9.3)		
Mild (25-49%)	0 (0.0)	66 (44.3)	5 (17.9)	1 (5.3)	0 (0.0)	72 (29.3)		
Moderate (50-69%)	0 (0.0)	39 (26.2)	12 (42.9)	2 (10.5)	0 (0.0)	53 (21.5)		
Severe (70-99%)	0 (0.0)	21 (14.1)	9 (32.1)	13 (68.4)	5 (35.7)	48 (19.5)		
Total occlusion (100%)	0 (0.0)	0 (0.0)	2 (7.1)	3 (15.8)	9 (64.3)	14 (5.7)		
Total	36 (100.0)		210 (100)			246 100.0 %		

DISCUSSION

Results of the data collected from the Army Cardiac Hospital Lahore, demonstrated the correlation between modifiable risk factors and coronary artery disease (CAD) among a sample of patients with complaints of chest pain. There were 246 participants in the study; 174 out of 246 were males, and 72 were females, with age ranges between 21 and 80 years. To assess the occurrence of CAD and its relation to such risk factors as diabetes mellitus, hypertension, smoking, total cholesterol levels, dyslipidaemia, and body mass index (BMI), the research employed coronary CT angiography (CTCA). The results depicted some essential correlations and gave knowledge of how modifiable risk factors cause the progression of CAD. The percentage of participants having CAD was 85.4% and this number was 210. This included 130 individuals who have a family history of CAD, which is an important non-modifiable risk factor in the development of the disease. The results of the research regarding the frequency distribution of the modifiable risk factors have shown that the dominant nosology of the patients was the non-diabetic (65.4%), hypertensive (58.1%) and non-smokers (70.3%), as well as the normal total cholesterol (80.5%). Nevertheless, dyslipidaemia (41.1%) and overweight (43.1%) made a strong impression in the cohort.

In the context of the modifiable risk factors statistical analysis of this study, the result of the logistic regression test showed that there were significant relations with both hypertension and smoking. Hypertension was the strongest risk factor of CAD, as subjects who had hypertension were more than sixfold more likely to develop CAD than subjects who were non-hypertensive ($p < 0.001$). The literature backed this discovery, as the works of Muhammad Zain ul Abidin et al. (2020) stated that the presence of hypertension strongly correlates with the development of CAD. Mechanisms of the involvement of hypertension in CAD comprise augmented vascular resistance, vascular endothelial derangement and thrombotic platelet aggregation and all of them contribute to the development of atherosclerotic plaques (12). These data indicate that blood pressure control should be given much attention in prevention of CAD.

The smoking practice was also noted to be an important modifiable risk factor, but it did not have any correlation with the vessel involvement or the plaque burden during the analysis of the study. The patterns of CAD where active smokers had a higher CAD prevalence (12.2%) than non-smokers (74.5%) were similar to the research carried out by Manfrini et al. (2020) in that there is a higher relative risk of obstructive CAD in active smokers. Although data on the statistical significance of smoking as a risk factor of CAD were not as impressive as those in hypertension, it still plays a significant role in incidence of CAD, especially in the determination of plaque and stenosis (13).

The p-value of the study is 0.123, which did not reveal the existence of a significant correlation between diabetes mellitus and the presence of CAD, proving that diabetes might not have such high independent influence on the occurrence of CAD as it is believed. This finding is in contrast to the findings of other authoritative studies that have brought out the elevated risk of CAD in persons with diabetes, including the one conducted by Bittencourt *et al.* (2014), which saw the existence of poor glycemic control on the development of coronary lesions (14). Nonetheless, the research found an association between diabetes and extent of CAD lesions especially on diabetics (p = 0.010) which showed greater extent of stenosis and also high incidence of mixed and calcified lesions. The present result correlates with the study by Manfrini *et al.* (2020), who stated that diabetes, particularly in female patients, intensifies CAD.

Regarding dyslipidemia, the research study depicted that there was no significant relationship with prevalence of CAD as far as dyslipidemia is concerned (p = 0.382), but 43.1 percent of the cohort had dyslipidemia. This observation can be based on the complicated picture of the lipid disorders due to some factors such as LDL cholesterol and HDL cholesterol level are major factors contributing to formation of plaque, as reported by Wu et al. (2024). The authors discovered that the patients with CAD have a high cholesterol level without any statistical relation. On the same note, total cholesterol failed to demonstrate a substantial correlation with the extent of CAD in the present research (p = 0.319). These facts are supported by those presented by the work carried out by Mannsverk *et al.* (2016) who concluded that enhancement in the levels of cholesterol may not related to the frequencies of cardiovascular diseases although more research would need to be done to illustrate the importance of lipid measures how they contribute to the extent of CAD (15).

The BMI measurement did not show significant results relating to the association of obesity to CAD, whichever BMI group measured, p-values were 0.999. This finding is comparable to those of the earlier studies that concluded that BMI alone may not be sufficient enough indicator of CAD risk but rather that visceral fat and waist circumference may prove to be more indicative of the onset of CAD. We may suppose that due to the complexity of metabolic systems, the presence of BMI does not show a stronger connection with the severity of CAD, which Malakar *et al.* (2019) discussed because insulin resistance and inflammation instead of BMI as a center of the issue with CAD (16).

In the case of the coronary vessels' participation, hypertension rose again as the most dominant factor. The presence of multiple vessel involvement especially the Left Anterior Descending (LAD) artery stood higher among the hypertensive patients’ group. Such an observation coincided with the literature, which found the increase in likelihood in hypertensive people developing multi-vessel disease and more severe coronary artery lesions (18). Smoking, in contrast, was not significantly related to vessel involvement, which is perhaps indicative of the fact that the effects of smoking on coronary arteries are more complicated and that long term exposure or interaction with additional risk factors may be required to cause damage (17). Regarding the morphology, the study has revealed that hypertension was strongly related to the presence of calcified and mixed plaques (p = <0.001) that are usually present in later stages of CAD. This concurs with the research at Bittencourt *et al.* (2014), which determined that poorer glycaemic control in diabetes is one of the factors contributing to worse coronary lesions (14). Likewise, existence of calcified plaques was prevalent among hypertensive patients further heightening the need to control blood pressure in order to prevent the development and growth of plaques.

The research, too, identified a very strong relationship of total cholesterol concentrations (p <0.001) and hypertension (p <0.001) with the occurrence of high plaque burden which affirms the status of dyslipidaemia in the precipitation of CAD. Dyslipidaemia was not significantly related however to plaque morphology or severity in this study. This finding can be attributed to the very complicated effect of added metabolic components in dyslipidaemia individuals, inflammatory markers, and a state of oxidative stress, which were not directly measured in the present study. Finally, our review also surpasses the work by Khan *et al.* (2024) that targeted the effectiveness of AI-QCT in the measurement of plaque burden (18). Although Khan *et al.* 2024, concluded that AI-QCT is very effective in determining plaque volume measurement, in our research, we go further into the contribution of modifiable risk factors that can predict the coronary plaque appearance and lesion severity; the risk factors included hypertension, diabetes, cholesterol, smoking, and BMI. We have also determined that hypertension and diabetes also play a significant role in the mixed and quantification plaques and also the severity of the lesions that occur due to CAD with diabetes being specially linked to severe stenosis and difficulty or inability to pass through the blockage. Diabetes and hypertension show the statistically significant association with plaque burden as their p values are 0.008 and <0.001 respectively. These factors also show significant correlation with calcium scores in vessels i.e, p values are 0.008 and 0.034 for diabetes and hypertension respectively. It was nowhere as obvious as with hypertension or smoking, perhaps because of the complexity of the conditions and the likelihood of hidden confounding factors. This detailed methodology augments clinical importance of such findings in that it directly associates the risk factors with the developments in CAD.

The absence of significant relations between diabetes, dyslipidemia and body mass index (BMI) implies that the use of these factors in isolation may not adequately predict CAD until other underlying conditions are put into consideration. Future longitudinal studies and a much broader, more diverse sample would help to get a better idea of how the change in these risk factors can modify the development of CAD. The research highlights the necessity of early detection, intervention, in controlling the hypertension, smoking, and levels of cholesterol. Preventive methods such as lifestyle changes and preventive care should be considered to lower the CAD burden especially on the risk groups.

CONCLUSION

This study emphasizes the importance of modifiable risk factors, especially high blood pressure, smoking, and cholesterol levels, in the development and progression of the coronary artery disease (CAD). Hypertension was established as the most powerful predictor as hypertensive patients have more than six times the likelihood of developing CAD than the non-hypertensive ones. Smoking was also proven to have significant effects on the development of CAD as active smokers had an even greater frequency of plaques and stenosis. Although diabetes mellitus, dyslipidaemia, and BMI were somehow linked to CAD, they were not to the extent of hypertension and smoking, which may be related to the complexity of the mentioned conditions and other limiting factors. Although smoking, cholesterol levels, dyslipidaemia, and body mass index also showed increasing trends in calcium scores and plaque burden, these associations did not reach statistical significance.

Additionally, a strong relationship was observed between lesion severity (CAD-RADS classification) and plaque burden. Patients without stenosis showed no plaque, while increasing lesion severity was accompanied by proportionally higher plaque accumulation, with the most extensive burden observed in total occlusion cases.

Overall, this study confirms the utility of CTCA in characterizing CAD and underscores the impact of modifiable risk factors—particularly diabetes and hypertension—on disease progression. These findings support the need for early identification and control of risk factors to reduce CAD burden.

Authors' contribution:

NA and TB Conceptualization & supervision; ZS Research work, writing initial manuscript & data acquisition; SA and AA Data analysis, statistical analysis & editing of manuscript.

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