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Carotid artery doppler study in patients of myocardial infarction and its correlation with other atherosclerotic risk factors: A cross-sectional study

Krishna Somani[1], Preeti Gupta[1,*] and Trupti Pisal[1]

[1]Department of Medicine, Jagjivan Ram Hospital, Mumbai, Maharashtra 400008, India

Abstract

Background: Carotid Doppler (CD) findings, including stenosis, increased carotid intima-media thickness (CIMT) and plaques, are linked to a higher risk of myocardial infarction (MI). Atherosclerotic risk factors play a key role in these changes. However, limited research has examined the association of these CD characteristics with risk factors in Indian patients with MI. This study evaluated the association of atherosclerotic risk factors with CD findings and the association between CIMT and coronary angiography (CAG) findings in MI patients.

Materials and methods: This cross-sectional study was conducted over 24 months from February 2022 to January 2023 in the Cardiology department of a tertiary care hospital. A total of 50 patients with acute MI were included, and sociodemographic data, atherosclerotic risk factors, and biochemical parameters were recorded. All the patients underwent both CD and CAG.

Results: CD findings had a negative and significant correlation with high-density lipoprotein (HDL) ($r=-0.51$, $p<0.001$). The CD findings had positive, and significant correlations with systolic blood pressure ($r=0.34$, $p=0.017$), total cholesterol ($r=0.34$, $p=0.015$), waist circumference ($r=0.36$, $p=0.011$), serum triglycerides ($r=0.37$, $p=0.009$), and low-density lipoprotein (LDL) ($r=0.38$, $p=0.007$). The LDL/HDL ratio ($r=0.41$, $p=0.003$) and HbA1c ($r=0.72$, $p<0.001$) showed positive, and significant correlations with CD findings. CIMT >1 mm was significantly associated with triglycerides >200 mg/dL, HDL <40 mg/dL, obesity, and HbA1c ≥ 6.5% ($p<0.05$). Dyslipidemia and diabetes were significantly associated with the plaques ($p<0.05$). Mean CIMT was significantly associated with CAG findings ($p<0.001$).

Conclusion: CD findings, particularly CIMT and plaques, are significantly associated with dyslipidemia, diabetes, and CAD severity.

Keywords: carotid intima-media thickness; carotid Doppler ultrasound; coronary artery disease; myocardial infarction; plaques; stenosis

Introduction

Carotid artery disease is a key risk factor for cerebrovascular and cardiovascular events, including myocardial infarction (MI) [1]. Globally, MI prevalence is approximately 23.3% [2], while in India, the incidence is alarmingly high at 64.37 per 1000 individuals aged 29–69 years, contributing to 31.7% of all deaths [3]. Carotid plaques, defined as lesions measuring 1.5 mm to less than 50% lumen narrowing, increased carotid intima-media thickness (CIMT) exceeding 1 mm, and carotid stenosis of 50% or more, are strongly associated with coronary artery disease (CAD) and subsequent MI [1]. Globally, the prevalence of increased CIMT >1mm, carotid plaques, and carotid stenosis are 27.6%, 21.1%, and 1.5%, respectively [4].

***Corresponding author:** Dr. Preeti Gupta, MD Medicine, Senior Divisional Medical Officer, Department of Medicine, Jagjivan Ram Hospital, Mumbai, Maharashtra 400008, India. Email: preetigupta401@gmail.com

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Carotid Doppler (CD) ultrasound, a non-invasive imaging technique, offers indirect markers of systemic atherosclerosis. It provides valuable insights into carotid stenosis, CIMT, and plaque characteristics, essential for risk stratification in MI patients [5,6]. Thickened CIMT indicates localized changes in the common carotid artery (CCA) and correlates with generalized atherosclerosis. Studies link increased CIMT to higher MI risk, with contributing risk factors such as diabetes, hypertension, dyslipidemia, obesity, and smoking [7]. CD allows identification of hemodynamically significant stenosis and the characterization of plaques, thereby guiding appropriate medical or surgical interventions [8].

In contrast, coronary angiography (CAG) remains the gold standard for evaluating CAD, allowing direct visualization of coronary lesions and precise quantification of atherosclerosis severity [6,9]. However, this invasive method is associated with morbidity, particularly in patients with unstable hemodynamic variables [9]. CIMT is a robust, non-invasive predictor of MI, reflecting early atherosclerosis and cardiovascular risk [4]. Numerous studies have demonstrated a correlation between CIMT and the extent and severity of coronary atherosclerosis observed on CAG [10].

Despite existing literature, research exploring the association between CIMT and CAG findings in Indian patients with MI remains limited [11]. While CIMT's predictive value for cardiovascular events is well-documented, its relationship with coronary lesions in this demographic is underexplored [12]. Understanding this link is crucial for improving risk stratification and management strategies.

Thus, this study aimed to evaluate association of atherosclerotic risk factors with CD findings and assess the association between CIMT and CAG findings in MI patients.

Methodology

A cross-sectional study was conducted over 24 months from February 2022 to January 2023 in the Department of Cardiology, Jagjivan Ram Hospital, Mumbai. The study was approved by the Institutional Ethical Committee, and written informed consent was obtained from the patients. The study included patients aged >18 years admitted, diagnosed with acute MI, and underwent both CD and CAG. While patients with trauma or brain neoplasms, rheumatic heart diseases, connective tissue diseases, congenital heart diseases, acute or chronic kidney disease or with previous history of cerebrovascular accident and any MI were excluded from the study.

Study participants admitted to the Cardiology Department were identified and written informed consent was obtained. Subsequently, a questionnaire was filled from the patient's medical records, including demographic characters (age, gender), signs and symptoms at presentation, atherosclerotic risk factors (hypertension, diabetes mellitus, obesity, dyslipidemia, and smoking), anthropometric parameter (waist circumference), vital parameters (systolic [SBP] and diastolic blood pressure [DBP]), lipid profile (total cholesterol, triglyceride, low-density lipoprotein [LDL], high-density lipoprotein [HDL], and LDL/HDL ratio) and glycated hemoglobin (HbA1c). A waist circumference of more than 35 inches for women and more than 40 inches for men was considered as obesity. Lastly, a CD ultrasound of the carotid arteries was performed using a portable ultrasound machine (Sonos 5500, Hewlett Packard, Inc., Anaheim, CA, USA).

The diagnosis of acute MI was reached on the basis of: A documented history of ischemic chest discomfort; Evolutionary changes on serially obtained 12-leads electrocardiogram (ECG) tracings, including: ST-segment elevation of ≥2 mm in two or more contiguous chest leads and ST-segment elevation of ≥1 mm in two or more limb leads.; and A rise and fall in serum cardiac markers were observed [13]. Patients with a history of chest discomfort, elevated cardiac biomarkers (i.e., Troponin-T) after 6 hours of chest pain onset, and ST elevation as described above were labeled as STEMI. Anterior wall MI was diagnosed if ST-segment elevation of ≥2 mm was present in two or more contiguous precordial leads. Inferior wall MI was diagnosed if ST-segment elevation of ≥1 mm was present in two or more contiguous leads II, III, and augmented Vector Foot (aVF) [13].

Patients with a history of chest discomfort, elevated cardiac biomarkers (i.e., Troponin-T) after 6 hours of chest pain onset, and ECG findings of ST-segment depression or deep T-wave inversion in precordial or limb leads were labeled as NSTE-MI [13].

For patients whose Troponin-T was initially negative, the test was repeated after 12 hours. Patients with a history of chest discomfort, ECG findings of ST-segment depression or deep T-wave inversion in precordial or limb leads, and consistently negative Troponin-T results (both initially and after 12 hours) were labeled as unstable angina and were not included in the study [13].

Biochemical tests

Troponin T (qualitative assay) was performed at the

initial diagnosis of AMI and was repeated after 12 hours if the initial result was negative. The assay was carried out using the Trop T (NAC) act kit with the immune-inhibition/modified International Federation of Clinical Chemistry and Laboratory Medicine method on the Micro Lab 200 Merck analyzer. HbA1c levels were measured using the latex-enhanced immunoturbidimetric method. Total cholesterol was estimated by the Cholesterol Oxidase-Phenol Aminophenazone method, triglycerides by the Glycerol-3-Phosphate Oxidase-Phenol Aminophenazone method, LDL by the homogeneous colorimetric enzymatic assay, and HDL by the phosphor-tungstic acid precipitation method [12].

Electrocardiogram

A standard 12-lead ECG was obtained on admission and whenever post-infarction angina occurred. The ECG was performed using a single-channel ECG machine (Bharat Parenterals Limited).

Carotid doppler (CD) ultrasound

CD scanning was conducted using a high-resolution Sonos 5500 (Hewlett Packard, Inc., Anaheim, CA, USA) equipped with a duplex B-mode scanner and a linear-phased array transducer of 7.5 MHz frequency. Scanning was performed by a trained sonologist. Intima-media thickness (IMT) was measured as the distance between the leading edge of the first echogenic line off the far wall of the carotid artery (lumen-intima interface) and the leading edge of the second echogenic line (media-adventitia interface). IMT measurements were obtained at end-diastole (peak of the R wave) at three segments on each side: the distal 1 cm of the CCA just before the bifurcation, the carotid bifurcation, and the proximal 1 cm of the internal carotid artery. The average of all six measurements (right and left sides) was calculated to determine the mean CIMT. CIMT values above 0.8 mm were considered abnormal [14].

Plaques were identified as focal thickenings of >1.5 mm or focal widenings of the vessel wall by 50% relative to adjacent segments, with protrusion into the lumen, composed of calcified or non-calcified components [15]. The total number of plaques on both sides was counted.

To determine the degree of stenosis, the criteria recommended by the 2003 consensus conference of the Society of Radiologists in Ultrasound was applied [13]. The internal carotid artery (ICA) was analyzed for the degree of stenosis on both the right and left sides as follows: Normal: ICA peak systolic velocity (PSV) <125 cm/s with no plaque or intimal thickening visible; <50% stenosis: ICA PSV <125 cm/s with plaque or intimal thickening visible; 50–69% stenosis: ICA PSV 125–230 cm/s with visible plaque; and >70% stenosis to near occlusion: ICA PSV >230 cm/s with visible plaque and lumen narrowing [16].

Coronary angiography (CAG)

All patients were taken up for coronary angiography. Stenosis of 70% or greater of the arterial intraluminal diameter of the right coronary artery or the left anterior descending or circumflex branches of the left coronary artery was considered significant. Single-vessel disease (SVD), double-vessel disease (DVD), and triple-vessel disease (TVD) were defined as significant stenosis of one, two, and three coronary arteries, respectively. Those without 70% or greater stenosis in any of the arteries were classified as having non-significant CAD.

Outcome variables

CIMT measurements (main outcome variable) were obtained within a region free of plaque with a clearly identified double-line pattern on the far wall of the CCA at least 10 mm below its end. Proximal, mid, and distal measurements were recorded, and the mean was calculated. The IMT was defined as the distance between the inner echogenic line representing the intima-blood interface and the outer echogenic line representing the adventitia-media junction. The Mannheim CIMT and Plaque Consensus recommendations were applied to identify patients with abnormal CIMT using the average of values obtained from each CCA. A CIMT thickness of 1 mm or more was classified as high [17].

Sample size estimation

To achieve a statistically significant difference with 80% power, 5% level of significance, 0.75 effect size, and a 95% confidence interval, the required sample size was calculated to be 50 patients per group.

Statistical analysis

The data was analyzed with SPSS (IBM, Armonk, NY, USA) version 23.0 for Windows. The categorical and continuous variables are represented as frequency (percentage) and mean ± standard deviation, respectively. A chi-square test was used to assess association between various categorical variables. Pearson correlation test was used to assess correlation of CD findings with atherosclerotic risk factors. One-way ANOVA was used to evaluate association of CIMT with CAG findings. The $p < 0.05$ was considered statistically significant.

Results

The patients were predominantly male (82%) with a mean age of 55.3 ± 6.2 years. The most common symptoms were chest pain (98%), followed by tachycardia (62%) and sweating (20%). In terms of risk factors, diabetes was the most prevalent, affecting 74% of the patients, followed by hypertension (68%), dyslipidemia (54%), and obesity (52%). Smoking was reported in 32% of the patients. Regarding the ECG diagnosis, many patients had STEMI (60%), while non-STEMI was present in 40% of the cohort (Table 1).

Table 1: Demographic and clinical characteristics.

Characteristics	*Mean ± SD n = 50*
	55.3 ± 6.2
Gender, n (%)	
Female	9 (18)
Male	41 (82)
Symptoms and signs, n (%)	
Raised JVP	0 (0)
Basal crepitations	0 (0)
Breathlessness	4 (8)
Nausea	4 (8)
Vomiting	4 (8)
Palpitation	8 (16)
Sweating	10 (20)
Tachycardia	31 (62)
Chest pain	49 (98)
Risk factors, n (%)	
Smoking	16 (32)
Obesity	26 (52)
Dyslipidemia	27 (54)
Hypertension	34 (68)
Diabetes	37 (74)
ECG diagnosis, n (%)	
STEMI	30 (60)
Non-STEMI	20 (40)

Abbreviations: JVP: Jugular Venous Pressure; ECG: Electrocardiogram; STEMI: ST-Elevation Myocardial Infarction.

The mean CIMT was 1.02 ± 0.68 mm. The majority of patients had no plaques (30%), while 28% patients had one plaque. In terms of stenosis, 60% of patients had no stenosis and 38% patients had < 50% stenosis (Table 2).

Table 2: Carotid doppler findings.

Findings	*n = 50 (1.02 ± 0.68)*
Plaques, n (%)	
0	15 (30)
1	14 (28)
2	7 (14)
3	3 (6)
≥ 4	1 (2)
Stenosis, n (%)	
No stenosis	30 (60)
< 50	19 (38)
> 50	1 (2)

Abbreviations: CIMT: Carotid Intima-Media Thickness.

There was moderate negative correlation between HDL levels and CD findings (r = -0.51, p < 0.001). The CD findings had positive, weak, and significant correlations with SBP (r = 0.34, p = 0.017), total cholesterol (r = 0.34, p = 0.015), waist circumference (r = 0.36, p = 0.011), serum triglycerides (r = 0.37, p = 0.009), and LDL (r = 0.38, p = 0.007). The LDL/HDL ratio (r = 0.41, p = 0.003) and HbA1c (r = 0.72, p < 0.001) showed strong, positive, and significant correlations with CD findings. While age (r = 0.05, p = 0.750) and DBP (r = 0.22, p = 0.312) had very weak and non-significant correlation with CD findings (Table 3).

Table 3: Correlation of carotid doppler findings with atherosclerotic risk factors.

Factors	*Correlation coefficient*	*p value*
Age, years	0.05	0.750
DBP, mmHg	0.22	0.312
SBP, mmHg	0.34	0.017
Waist Circumference, cm	0.36	0.011
Total cholesterol, mg/dL	0.34	0.015
Serum triglyceride, mg/dL	0.37	0.009
LDL, mg/dL	0.38	0.007
HDL, mg/dL	-0.51	<0.001
LDL / HDL	0.41	0.003
HbA1c, %	0.72	<0.001

Abbreviations: HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; SBP: Systolic Blood Pressure: DBP: Diastolic Blood Pressure.

CIMT > 1 mm was significantly associated with triglycerides > 200 mg/dL (p = 0.003), HDL < 40 mg/dL (p = 0.002), obesity (p = 0.016), and HbA1c ≥ 6.5% (p <

0.001). While CIMT was not associated with LDL > 160 mg/dL (p = 0.21), smoking (p = 0.051), and hypertension (p = 0.318) (Table 4).

Table 4: Association of risk factors with mean CIMT.

Factors	*≤ 1 mm (n = 17)*	*> 1 mm (n = 33)*	*p value*
Triglyceride > 20 mg/dL	1 (5.88%)	16 (33.39%)	0.003
LDL > 160 mg/dL	2 (11.77%)	9 (27.27%)	0.21
HDL < 40 mg/dL	3 (17.65%)	21 (63.64%)	0.002
Obesity	7 (41.18%)	25 (75.76%)	0.016
Smoking	7 (41.18%)	8 (24.24%)	0.051
Hypertension	10 (58.82%)	24 (72.72%)	0.318
HbA1c > 6.5%	10 (58.82%)	3 (9.09%)	< 0.001

Abbreviations: HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein.

Dyslipidemia (p < 0.05) and diabetes (p < 0.05) were significantly associated with the presence of plaques. In contrast, obesity, smoking, and hypertension showed no significant association with plaque presence (p > 0.05) (Table 5).

Table 5: Association of risk factors with plaque.

Factors	*Present (n = 35)*	*Absent (n = 15)*	*p value*
Dyslipidemia	29 (82.86%)	8 (53.33%)	< 0.05
Obesity	20 (57.14%)	6 (40.00%)	> 0.05
Smoking	22 (62.86%)	11 (73.33%)	> 0.05
Hypertension	22 (62.86%)	11 (73.33%)	> 0.05
Diabetes	31 (88.57%)	6 (40.00%)	< 0.05

Mean CIMT was significantly associated with CAG findings, as determined by ANOVA (p < 0.001) (Table 6).

Table 6: Coronary angiographic findings with mean CIMT.

Findings	*Mean CIMT (mm)*	*n = 50*	*p value*
Not specified	0.75	9 (18%)	< 0.001
Single vessel disease	1	25 (50%)	
Double vessel disease	1.22	13 (26%)	
Triple vessel disease	1.27	3 (6%)	

Abbreviations: CIMT: Carotid intima-media thickness.

Discussion

The principal findings of the study revealed a significant correlation of CD findings with atherosclerotic risk factors, including waist circumference, SBP, total cholesterol, serum triglyceride, LDL, HDL, LDL/HDL ratio, and HbA1c levels. Plaque was significantly associated with dyslipidemia and diabetes. CIMT > 1 mm was significantly associated with triglyceride > 200 mg/dL, HDL <40 mg/dL, HbA1c ≥6.5%, and obesity. Finally, a significant relationship was identified between CAG findings and CIMT, with CIMT increasing in tandem with CAD severity.

The mean CIMT was 1.02 ± 0.68 mm, indicating a moderate level of atherosclerotic change. CIMT values above 1 mm are associated with increased cardiovascular risk, reflecting early-stage atherosclerosis, a known predictor of events such as MI. Prior studies reported mean CIMT values between 0.88 and 1.023 mm, signifying a high cardiovascular risk burden [8]. The higher prevalence of abnormal CIMT may stem from the use of a lower cutoff value (≥1 mm), classifying more individuals as abnormal. Similarly, the Rotterdam study by Meer et al., which followed 7,983 individuals >55 years for 4.6 years, found significantly higher CIMT in those who experienced MI [18]. The cutoff for thickened CIMT, used in the present study, was 1 mm, whereas Held et al. linked CIMT >1.02 mm to cardiovascular death or MI risk [19]. These differences could be ascribed to younger cohort (mean age 55 years) and higher prevalence of diabetes (78%) and hypertension (70%) [19].

The presence of plaque was strongly associated with dyslipidemia and diabetes. Carotid plaques are critical markers of systemic atherosclerosis, strongly associated with MI and cardiovascular events [20]. In the study cohort, carotid plaque presence was associated with dyslipidemia and diabetes, key risk factors for atherosclerosis and contributors to plaque formation in the carotid arteries. Dyslipidemia, marked by high triglycerides and low HDL, is a key contributor to atherosclerosis and cardiovascular events [21]. Similarly, diabetes accelerates atherosclerosis through endothelial dysfunction and chronic inflammation [22]. Prior studies have shown a linear correlation between carotid plaques and diabetes in patients at risk of carotid atherosclerosis and MI [23]. Additionally, Sung et al. reported elevated lipoprotein(a) levels (>50 mg/dL) and higher rates of diabetes and dyslipidemia in patients at risk of MI and cardiovascular diseases, further underscoring these associations [24]. The absence of significant associations between CIMT and LDL, smoking, or hypertension suggests these factors were either well-managed or less prominent in this cohort.

This highlights the importance of improving lipid profiles and glycemic control to reduce cardiovascular risk. Kota et al. also found a higher prevalence of increased CIMT in patients with diabetes, where CIMT >0.8 mm was strongly linked to cardiovascular diseases [14]. In contrast, Yusuf et al. reported that risk factors including smoking, hypertension, cholesterol and diabetes are significantly correlated with plaque formation, thereby increasing risk of MI [25].

In the present study, a moderate negative correlation was observed between HDL levels and CD findings, indicating that lower HDL levels were associated with more significant atherosclerotic changes in the carotid arteries. This aligns with existing literature demonstrating that low HDL cholesterol is a critical risk factor for atherosclerosis and cardiovascular diseases, including MI [26]. HDL plays a protective role in cardiovascular health by facilitating reverse cholesterol transport, reducing arterial plaque formation. Conversely, CD findings showed weak positive correlations with other atherosclerotic risk factors, suggesting that higher levels of these factors, such as hypertension, hyperlipidemia, and obesity, contribute to increased carotid atherosclerosis, consistent with findings from Song et al. [4].

Notably, HbA1c levels demonstrated a strong positive correlation with CD findings, emphasizing the role of diabetes and poor glycemic control in accelerating atherosclerosis through mechanisms like endothelial dysfunction and inflammation [22]. In contrast, age and DBP exhibited very weak correlations, unlike studies identifying age as a significant predictor of increased CIMT and carotid plaque formation, suggesting that the relationship between age and atherosclerosis may vary across populations [4]. The weak correlation, in this cohort, suggests that lifestyle and metabolic health factors likely had a greater impact on carotid atherosclerosis than chronological age alone [27].

The significant association between CD and CAG findings observed reflects shared pathophysiological mechanisms of atherosclerosis leading to acute MI. Systemic atherosclerosis, driven by factors like dyslipidemia, hypertension, diabetes, and lifestyle, contributes to plaque formation and arterial thickening in both coronary and carotid arteries [28]. This aligns with findings by Nowak et al., where CAG identified CAD, and CD ultrasound demonstrated carotid plaques and IMT, effectively discriminating patients with CAD [29].

In the present study, patients with non-significant CAD had a mean CIMT of 0.76 mm, with CIMT progressively increasing alongside CAD severity. This finding suggests that greater coronary involvement corresponds to more pronounced carotid atherosclerotic changes. Similarly, Coskun et al. observed a mean CIMT of 0.78 ± 0.21 mm in patients with non-critical coronary lesions, with CIMT ranging from 1.2 to 1.6 mm as CAD severity increased [10]. Gacoń et al. also highlighted CIMT as a significant predictor of CAD severity, emphasizing its utility as a cardiovascular risk marker [28]. However, Heriadi et al. found that although CIMT was elevated in patients with multi-vessel disease, it did not consistently correlate with stenosis severity assessed by the Gensini score, suggesting additional factors influencing CAD progression [30].

Despite significant association found between CAG findings and mean CIMT, underscoring the potential of CIMT as a non-invasive marker for assessing atherosclerotic burden in MI patients, there are few limitations to consider. Firstly, cross-sectional study design limits the ability to establish causality between the observed associations. Secondly, the sample size of 50 was not large enough to generalize the findings to the broader population, particularly in diverse ethnic groups. Thirdly, reliance on CAG findings may overlook other significant atherosclerotic changes that could be detected through advanced imaging techniques. Furthermore, potential confounding factors, such as medication use and lifestyle changes, were not controlled for, which could influence the results. Finally, the study may not account for the longitudinal progression of atherosclerosis, as it only provides a snapshot of the relationship between CIMT and coronary disease at a single point in time.

Conclusions

The study findings reveal a significant association between increased mean CIMT, plaque formation, and atherosclerotic risk factors, particularly dyslipidemia and diabetes. This underscores the importance of managing these risk factors in this cohort to mitigate the progression of carotid atherosclerosis and CAD, ultimately enhancing outcomes for patients with MI. Additionally, a significant association between CD findings, including mean CIMT, and the severity of CAD in patients with MI was observed. The results indicate that increased CIMT correlates with higher degrees of coronary artery involvement, suggesting that carotid ultrasound can serve as a valuable non-invasive tool for assessing cardiovascular risk.

Conflicts of interest

Authors declare no conflicts of interest.

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