

## Original Research Article

# A STUDY OF MEAN CAROTID INTIMA-MEDIA THICKNESS IN PATIENT WITH TYPE 2 DIABETES MELLITUS(T2DM) IN A TERTIARY CARE HOSPITAL

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## ABSTRACT

**Background:** Type 2 diabetes mellitus (T2DM) is associated with accelerated atherosclerosis and an increased risk of cardiovascular disease. Carotid intima-media thickness (CIMT) is a validated, non-invasive marker of subclinical atherosclerosis. This study aimed to evaluate CIMT and determine its association with glycemic control and other cardiovascular risk factors in patients with T2DM.

**Materials and Methods:** A cross-sectional observational study was conducted among 60 adults with T2DM attending the Department of General Medicine, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, between May 2024 and October 2025. Demographic, clinical, anthropometric, biochemical, and lipid profile data were collected. CIMT was measured using B-mode ultrasonography. Correlation analysis and multivariable linear regression were performed to identify independent predictors of CIMT.

**Results:** The mean age was  $52.4 \pm 9.1$  years, and 56.7% of participants were male. The mean HbA1c was  $8.1 \pm 1.1\%$ , and the mean duration of diabetes was  $8.2 \pm 4.3$  years. Mean CIMT increased significantly with worsening glycemic control, longer diabetes duration, and hypertension (all  $p < 0.001$ ). CIMT showed significant positive correlations with age ( $r=0.48$ ), duration of diabetes ( $r=0.62$ ), HbA1c ( $r=0.54$ ), BMI ( $r=0.41$ ), LDL cholesterol ( $r=0.46$ ), and systolic blood pressure ( $r=0.44$ ). Multivariable regression identified HbA1c, duration of diabetes, age, BMI, systolic blood pressure, and LDL cholesterol as independent predictors of increased CIMT.

**Conclusion:** Poor glycemic control and longer duration of diabetes were the strongest determinants of increased CIMT. Routine CIMT assessment may facilitate early detection of subclinical atherosclerosis and improve cardiovascular risk stratification in patients with T2DM.

**Keywords:** Type 2 diabetes mellitus; Carotid intima-media thickness; Glycated hemoglobin; Atherosclerosis; Cardiovascular risk; Glycemic control.

## INTRODUCTION

Type 2 diabetes mellitus (T2DM) is one of the most prevalent non-communicable diseases worldwide and represents a major public health challenge because of its rapidly increasing incidence and its strong association with cardiovascular morbidity and mortality.<sup>[1]</sup> According to the International Diabetes

Federation (IDF), the global burden of diabetes continues to rise, with low- and middle-income countries contributing the majority of affected individuals. India, often referred to as the "diabetes capital of the world," has witnessed a substantial increase in the prevalence of T2DM over the past two decades, placing enormous pressure on the healthcare system and increasing the burden of diabetes-related vascular complications.<sup>[2,3]</sup> T2DM is characterized by

insulin resistance and progressive pancreatic  $\beta$ -cell dysfunction, resulting in chronic hyperglycemia and multiple metabolic abnormalities.<sup>[4]</sup> Persistent hyperglycemia promotes oxidative stress, chronic low-grade inflammation, endothelial dysfunction, and dyslipidemia, all of which accelerate the development of vascular complications. Consequently, cardiovascular disease remains the leading cause of death among individuals with T2DM, accounting for a significant proportion of diabetes-related mortality.<sup>[5,6]</sup> Epidemiological studies have consistently demonstrated that patients with diabetes have a two- to four-fold higher risk of developing coronary artery disease, stroke, and peripheral arterial disease compared with the non-diabetic population.<sup>[5,7]</sup> Atherosclerosis is a chronic inflammatory disease involving endothelial injury, lipid deposition, smooth muscle proliferation, and progressive thickening of the arterial wall. In patients with T2DM, the atherosclerotic process begins early and progresses rapidly because of the combined effects of hyperglycemia, insulin resistance, oxidative stress, and associated metabolic risk factors such as hypertension, obesity, and dyslipidemia.<sup>[6]</sup> Therefore, early identification of subclinical atherosclerosis is essential for timely intervention and reduction of future cardiovascular events. Although coronary angiography remains the reference standard for assessing coronary artery disease, its invasive nature, procedural risks, and high cost limit its use as a screening tool, particularly in asymptomatic individuals. Consequently, there has been increasing interest in non-invasive methods capable of identifying early vascular changes before the occurrence of overt cardiovascular disease.<sup>[8]</sup> Among the available imaging modalities, high-resolution B-mode ultrasonographic measurement of carotid intima-media thickness (CIMT) has emerged as a reliable, reproducible, and cost-effective surrogate marker of systemic atherosclerosis.<sup>[9,10]</sup> Carotid intima-media thickness represents the combined thickness of the intimal and medial layers of the carotid artery and reflects the structural changes occurring during early atherosclerosis. Numerous histopathological and clinical studies have validated CIMT as an indicator of generalized atherosclerotic burden. Besides measuring arterial wall thickness, carotid ultrasonography also enables visualization of plaque formation and arterial morphology without exposing patients to radiation or contrast agents, making it suitable for routine clinical practice.<sup>[9-11]</sup> Several studies have consistently reported significantly higher CIMT values in patients with T2DM compared with healthy individuals, indicating accelerated vascular aging even before the appearance of clinically evident cardiovascular disease.<sup>[12-14]</sup> Increased CIMT has been shown to correlate with the duration of diabetes, poor glycemic control, hypertension, dyslipidemia, obesity, smoking, and other cardiovascular risk factors.<sup>[13,14]</sup> Furthermore, longitudinal studies have demonstrated that increased CIMT independently predicts future

myocardial infarction, stroke, and cardiovascular mortality, emphasizing its value in cardiovascular risk stratification.<sup>[11]</sup> In India, where diabetes develops at a younger age and cardiovascular complications occur earlier than in many Western populations, identification of subclinical atherosclerosis assumes particular importance. Despite growing evidence supporting the clinical utility of CIMT, data regarding its distribution among patients with T2DM in many tertiary care settings remain limited. Therefore, the present study was undertaken to assess the mean carotid intima-media thickness in patients with type 2 diabetes mellitus attending a tertiary care hospital, thereby contributing to the early identification of individuals at increased cardiovascular risk.

## MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of General Medicine, Sathagiri Institute of Medical Sciences and Research Centre (SIMS & RC), Bengaluru, India, over an 18-month period from May 2024 to October 2025. Patients were recruited from both the outpatient and inpatient services of the department.

**Study Population:** The study included 60 consecutive adult patients diagnosed with Type 2 Diabetes Mellitus (T2DM). The sample size was determined a priori and a total of 60 participants were enrolled to improve the precision of the study estimates.

**Eligibility Criteria:** Patients aged  $\geq 18$  years with a confirmed diagnosis of T2DM who provided written informed consent were eligible for inclusion. Patients with autoimmune vasculitis, hepatic failure, renal failure, malignancy, autoimmune disorders, or those receiving statin therapy were excluded because these conditions could independently influence carotid intima-media thickness (CIMT).

**Data Collection:** After obtaining written informed consent, demographic and clinical information including age, sex, duration of diabetes, hypertension, smoking status, and dyslipidemia were recorded using a structured data collection form. A detailed clinical evaluation was performed to identify diabetes-related complications, including microvascular complications (neuropathy, nephropathy, and retinopathy) and macrovascular complications (coronary artery disease and peripheral arterial disease). Anthropometric measurements and cardiovascular examination were performed according to standard clinical protocols.

**Laboratory and Radiological Assessment:** Fasting blood glucose, glycated hemoglobin (HbA1c), and fasting lipid profile were measured using standard laboratory methods. Carotid intima-media thickness was assessed using a Voluson S8 ultrasound system equipped with a high-frequency linear transducer. All examinations were performed by a senior radiologist using a standardized B-mode ultrasonography

protocol. Participants were examined in the supine position with the neck slightly extended and rotated contralaterally. Longitudinal images of the common carotid artery demonstrating the characteristic double-line pattern formed by the lumen–intima and media–adventitia interfaces were obtained, and a single CIMT measurement was recorded for each participant.

**Statistical Analysis:** Data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality and presented as mean  $\pm$  standard deviation (SD) or median with interquartile range (IQR), as appropriate, while categorical variables were expressed as frequencies and percentages. Differences in mean CIMT between two groups were analyzed using the independent Student's t-test, whereas comparisons among multiple groups were performed using one-way analysis of variance (ANOVA) followed by Tukey's honestly significant difference (HSD) post hoc test. Associations between categorical variables were evaluated using the Chi-square test. Pearson correlation analysis was used to assess the relationship between CIMT and continuous variables including age, duration of diabetes, HbA1c, body mass index (BMI), low-density lipoprotein cholesterol (LDL-C), and systolic blood pressure. Multivariable linear regression analysis was performed to identify independent predictors of CIMT. A two-tailed p-value  $<0.05$  was considered statistically significant.

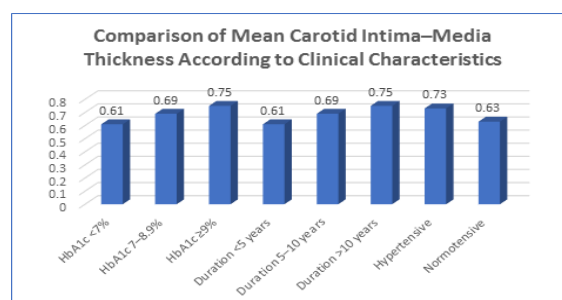
**Ethical Considerations:** The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Institutional Ethics Committee approval was obtained before commencement of the study, and written informed consent was obtained from all participants prior to enrollment.

## RESULTS

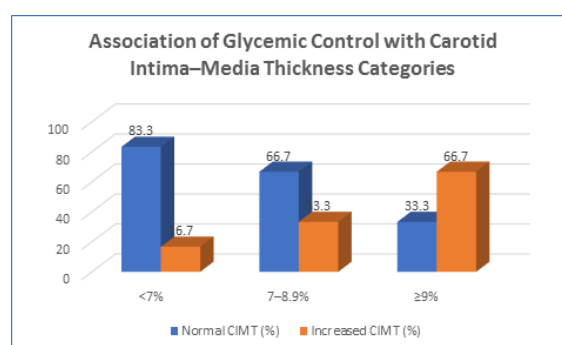
The mean HbA1c level of the study participants was  $8.1 \pm 1.1\%$ , with a median value of  $8.0\%$  (IQR:  $7.3\text{--}9.0\%$ ). Only  $20.0\%$  of patients had adequate glycemic control with HbA1c  $<7\%$ , whereas  $50.0\%$  had HbA1c levels between  $7\%$  and  $8.9\%$ , and  $30.0\%$  had HbA1c  $\geq 9\%$ . The mean total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride levels were  $198 \pm 36$  mg/dL,  $122 \pm 28$  mg/dL,  $41 \pm 7$  mg/dL, and  $176 \pm 52$  mg/dL, respectively. Neuropathy was the most common diabetic complication, affecting  $31.7\%$  of patients, followed by retinopathy in  $28.3\%$ , nephropathy in  $23.3\%$ , coronary artery disease in  $18.3\%$ , and peripheral arterial disease in  $10.0\%$  [Table 2]

Mean carotid intima–media thickness (CIMT) increased significantly with worsening glycemic control. Patients with HbA1c  $<7\%$  had a mean CIMT of  $0.61 \pm 0.05$  mm, compared with  $0.69 \pm 0.06$  mm among those with HbA1c between  $7\%$  and  $8.9\%$ , and  $0.75 \pm 0.05$  mm among those with HbA1c  $\geq 9\%$  ( $F=29.8$ ;  $p<0.001$ ). A similar progressive increase in

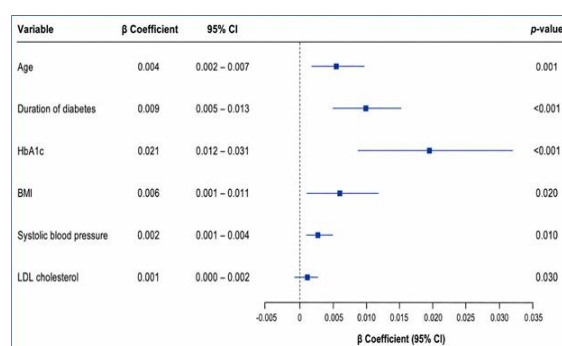
CIMT was observed with increasing duration of diabetes. The mean CIMT was  $0.61 \pm 0.05$  mm in patients with diabetes duration of  $<5$  years,  $0.69 \pm 0.06$  mm in those with a duration of  $5\text{--}10$  years, and  $0.75 \pm 0.05$  mm in those with diabetes for  $>10$  years ( $F=29.8$ ;  $p<0.001$ ). Hypertensive patients had a significantly greater mean CIMT than normotensive patients ( $0.73 \pm 0.06$  mm versus  $0.63 \pm 0.05$  mm;  $t=6.79$ ;  $p<0.001$ ) [Table 3]. Correlation analysis demonstrated that CIMT had significant positive correlations with all the evaluated clinical and biochemical variables. The strongest correlation was observed with the duration of diabetes ( $r=0.62$ ;  $p<0.001$ ), followed by HbA1c ( $r=0.54$ ;  $p<0.001$ ), age ( $r=0.48$ ;  $p<0.001$ ), LDL cholesterol ( $r=0.46$ ;  $p<0.001$ ), systolic blood pressure ( $r=0.44$ ;  $p<0.001$ ), and BMI ( $r=0.41$ ;  $p=0.001$ ) [Table 4]



**Figure 1: Comparison of Mean Carotid Intima–Media Thickness According to Clinical Characteristics**



**Figure 2: Association of Glycemic Control with Carotid Intima–Media Thickness Categories**



**Figure 3: Multivariable Linear Regression Analysis for Predictors of Carotid Intima–Media Thickness**

The proportion of patients with increased CIMT rose progressively with worsening glycemic control. Increased CIMT was observed in only  $16.7\%$  of

patients with HbA1c <7%, compared with 33.3% of those with HbA1c between 7% and 8.9% and 66.7% of those with HbA1c ≥9%. Conversely, normal CIMT was present in 83.3%, 66.7%, and 33.3% of patients in the respective HbA1c categories. The association between glycemic-control category and CIMT category was statistically significant ( $\chi^2=16.3$ ;  $p<0.001$ ) [Table 5 and Figure 2] On multivariable linear regression analysis, age, duration of diabetes, HbA1c, BMI, systolic blood pressure, and LDL cholesterol remained significant independent

predictors of CIMT. Each additional year of age was associated with a 0.004-mm increase in CIMT (95% CI: 0.002–0.007;  $p=0.001$ ), while each additional year of diabetes duration was associated with a 0.009-mm increase (95% CI: 0.005–0.013;  $p<0.001$ ). Each 1% increase in HbA1c was associated with a 0.021-mm increase in CIMT (95% CI: 0.012–0.031;  $p<0.001$ ). BMI, systolic blood pressure, and LDL cholesterol were also independently associated with greater CIMT [Table 6 and Figure 3].

**Table 1: Baseline Demographic and Clinical Characteristics of the Study Participants (N = 60)**

| Variable                                | Value            |
|-----------------------------------------|------------------|
| Age (years), Mean ± SD                  | 52.4 ± 9.1       |
| Median (IQR)                            | 53 (45–60)       |
| Age range (years)                       | 35–70            |
| Male sex, n (%)                         | 34 (56.7)        |
| Female sex, n (%)                       | 26 (43.3)        |
| Duration of diabetes (years), Mean ± SD | 8.2 ± 4.3        |
| Median (IQR)                            | 8 (4–12)         |
| Duration <5 years, n (%)                | 18 (30.0)        |
| Duration 5–10 years, n (%)              | 23 (38.3)        |
| Duration >10 years, n (%)               | 19 (31.7)        |
| BMI (kg/m <sup>2</sup> ), Mean ± SD     | 27.8 ± 3.4       |
| Median (IQR)                            | 27.5 (25.2–30.1) |
| Normal BMI, n (%)                       | 14 (23.3)        |
| Overweight, n (%)                       | 28 (46.7)        |
| Obese, n (%)                            | 18 (30.0)        |
| Hypertension, n (%)                     | 36 (60.0)        |
| Normotensive, n (%)                     | 24 (40.0)        |

**Table 2: Glycemic Status, Lipid Profile, and Diabetic Complications (N = 60)**

| Variable                             | Value         |
|--------------------------------------|---------------|
| HbA1c (%), Mean ± SD                 | 8.1 ± 1.1     |
| Median (IQR)                         | 8.0 (7.3–9.0) |
| HbA1c <7%, n (%)                     | 12 (20.0)     |
| HbA1c 7–8.9%, n (%)                  | 30 (50.0)     |
| HbA1c ≥9%, n (%)                     | 18 (30.0)     |
| Total cholesterol (mg/dL), Mean ± SD | 198 ± 36      |
| LDL cholesterol (mg/dL), Mean ± SD   | 122 ± 28      |
| HDL cholesterol (mg/dL), Mean ± SD   | 41 ± 7        |
| Triglycerides (mg/dL), Mean ± SD     | 176 ± 52      |
| Neuropathy, n (%)                    | 19 (31.7)     |
| Nephropathy, n (%)                   | 14 (23.3)     |
| Retinopathy, n (%)                   | 17 (28.3)     |
| Coronary artery disease, n (%)       | 11 (18.3)     |
| Peripheral arterial disease, n (%)   | 6 (10.0)      |

**Table 3: Comparison of Mean Carotid Intima–Media Thickness According to Clinical Characteristics**

| Variable            | Mean CIMT (mm) ± SD | Test Statistic | p-value |
|---------------------|---------------------|----------------|---------|
| HbA1c <7%           | 0.61 ± 0.05         | F=29.8         | <0.001  |
| HbA1c 7–8.9%        | 0.69 ± 0.06         |                |         |
| HbA1c ≥9%           | 0.75 ± 0.05         |                |         |
| Duration <5 years   | 0.61 ± 0.05         | F=29.8         | <0.001  |
| Duration 5–10 years | 0.69 ± 0.06         |                |         |
| Duration >10 years  | 0.75 ± 0.05         |                |         |
| Hypertensive        | 0.73 ± 0.06         | t=6.79         | <0.001  |
| Normotensive        | 0.63 ± 0.05         |                |         |

**Table 4: Correlation Between Carotid Intima–Media Thickness and Clinical Variables**

| Variable                | Pearson's r | p-value |
|-------------------------|-------------|---------|
| Age                     | 0.48        | <0.001  |
| Duration of diabetes    | 0.62        | <0.001  |
| HbA1c                   | 0.54        | <0.001  |
| BMI                     | 0.41        | 0.001   |
| LDL cholesterol         | 0.46        | <0.001  |
| Systolic blood pressure | 0.44        | <0.001  |

**Table 5: Association of Glycemic Control with Carotid Intima–Media Thickness Categories**

| HbA1c Category | Normal CIMT, n (%) | Increased CIMT, n (%) | Total, n (%) | p-value |
|----------------|--------------------|-----------------------|--------------|---------|
| <7%            | 10 (83.3)          | 2 (16.7)              | 12 (100.0)   | <0.001  |
| 7–8.9%         | 20 (66.7)          | 10 (33.3)             | 30 (100.0)   |         |
| ≥9%            | 6 (33.3)           | 12 (66.7)             | 18 (100.0)   |         |
| Chi-square     |                    | 16.3                  |              |         |

**Table 6: Multivariable Linear Regression Analysis for Predictors of Carotid Intima–Media Thickness**

| Variable                | β Coefficient | 95% CI      | p-value |
|-------------------------|---------------|-------------|---------|
| Age                     | 0.004         | 0.002–0.007 | 0.001   |
| Duration of diabetes    | 0.009         | 0.005–0.013 | <0.001  |
| HbA1c                   | 0.021         | 0.012–0.031 | <0.001  |
| BMI                     | 0.006         | 0.001–0.011 | 0.020   |
| Systolic blood pressure | 0.002         | 0.001–0.004 | 0.010   |
| LDL cholesterol         | 0.001         | 0.000–0.002 | 0.030   |

## DISCUSSION

The present cross-sectional observational study evaluated carotid intima–media thickness (CIMT) and its association with glycemic control, duration of diabetes, obesity, hypertension, lipid profile, and diabetic complications in patients with type 2 diabetes mellitus (T2DM). Our findings demonstrated that increasing age, longer duration of diabetes, poor glycemic control, obesity, hypertension, and elevated LDL cholesterol were significantly associated with increased CIMT, highlighting their contribution to subclinical atherosclerosis.

The study population had a mean age of  $52.4 \pm 9.1$  years, with a slight male predominance (56.7%). These findings are comparable with Kayastha et al,<sup>[15]</sup> and Bhattarai et al,<sup>[16]</sup> who reported similar mean ages among diabetic patients. Kota et al,<sup>[17]</sup> observed significantly higher CIMT in diabetic patients than healthy controls, while Winckler et al,<sup>[18]</sup> identified advancing age as a major determinant of CIMT progression, supporting our observation that vascular changes worsen with age. The mean duration of diabetes was  $8.2 \pm 4.3$  years, and approximately 70% of participants had diabetes for more than five years. Mean CIMT increased significantly from  $0.61 \pm 0.05$  mm in patients with diabetes duration <5 years to  $0.75 \pm 0.05$  mm in those with duration >10 years ( $p < 0.001$ ). Similar associations between longer diabetes duration and increased CIMT have been reported by Wagenknecht et al,<sup>[14]</sup> Kota et al,<sup>[17]</sup> and Rad et al,<sup>[19]</sup> suggesting cumulative hyperglycemic exposure accelerates atherosclerotic changes. Poor glycemic control was strongly associated with CIMT in our study. Mean HbA1c was  $8.1 \pm 1.1\%$ , with 80% of patients having HbA1c >7%. CIMT increased progressively with worsening glycemic control, and patients with HbA1c ≥9% had the highest prevalence of increased CIMT (66.7%). Comparable findings were reported by Kumar et al,<sup>[20]</sup> Olt et al,<sup>[21]</sup> and Dehdashti Sharokh et al,<sup>[22]</sup> all of whom demonstrated significant positive relationships between HbA1c and CIMT. Obesity was highly prevalent, with 76.7% of participants being overweight or obese. BMI showed a significant positive correlation with CIMT and remained an

independent predictor. Similar findings have been reported by Bažadona et al,<sup>[23]</sup> Zahra et al,<sup>[24]</sup> and the systematic review by Mashaba et al,<sup>[25]</sup> confirming obesity as an important contributor to vascular injury in T2DM. Hypertension was present in 60% of patients, and hypertensive individuals had significantly greater CIMT than normotensive participants. These findings agree with Al-Auqbi et al,<sup>[26]</sup> Mirza et al,<sup>[27]</sup> and Baroncini et al,<sup>[28]</sup> who demonstrated that hypertension markedly accelerates carotid atherosclerosis in diabetic patients. Likewise, elevated LDL cholesterol correlated positively with CIMT, consistent with studies by Mashaba et al,<sup>[25]</sup> Bažadona et al,<sup>[23]</sup> and Aggarwal et al.<sup>[29]</sup> Neuropathy (31.7%), retinopathy (28.3%), and nephropathy (23.3%) were the most frequent diabetic complications, comparable with observations by Venguidesvarane et al,<sup>[30]</sup> and Khan et al,<sup>[31]</sup> Correlation and multivariable regression analyses identified duration of diabetes and HbA1c as the strongest independent predictors of CIMT, followed by age, BMI, systolic blood pressure, and LDL cholesterol. Similar findings were reported by Shukla et al,<sup>[32]</sup> Pramayudha et al,<sup>[33]</sup> and Soliman et al,<sup>[34]</sup> Overall, our findings support previous evidence that CIMT is a reliable, non-invasive marker for early detection of subclinical atherosclerosis and cardiovascular risk stratification in patients with T2DM.

## CONCLUSION

The present study demonstrated that carotid intima–media thickness (CIMT) was significantly associated with poor glycemic control, longer duration of diabetes, hypertension, higher body mass index, elevated LDL cholesterol, and advancing age among patients with type 2 diabetes mellitus. Duration of diabetes and HbA1c emerged as the strongest independent predictors of increased CIMT, highlighting the cumulative impact of chronic hyperglycemia on subclinical atherosclerosis. These findings support the use of CIMT as a simple, non-invasive, and reliable marker for early detection of cardiovascular risk in patients with T2DM. Early optimization of glycemic control and aggressive management of modifiable cardiovascular risk

factors may help reduce the progression of atherosclerosis and improve long-term cardiovascular outcomes.

**Limitation of the Study:** This study has certain limitations. First, the relatively small sample size from a single tertiary care center may limit the generalizability of the findings to the broader diabetic population. Second, the cross-sectional study design precludes establishing a causal relationship between risk factors and increased carotid intima-media thickness. Finally, CIMT was measured at a single time point without longitudinal follow-up, preventing assessment of disease progression or the impact of therapeutic interventions over time.

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