

## CAROTID INTIMA-MEDIA THICKNESS IN PATIENTS WITH AND WITHOUT TYPE 2 DIABETES MELLITUS: A COMPARATIVE STUDY

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Received : 04/08/2026  
Received in revised form : 02/09/2026  
Accepted : 16/09/2026

### Keywords:

Carotid intima-media thickness; Type 2 diabetes mellitus; Subclinical atherosclerosis.

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DOI: 10.47009/jamp.2026.8.5.94

Source of Support: Nil,

Conflict of Interest: None declared

Int J Acad Med Pharm  
2026; 8 (5); 599-603



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### ABSTRACT

**Background:** Community-acquired pneumonia (CAP) remains an important cause of hospitalization, respiratory failure and mortality. Clinical severity scores and inflammatory biomarkers assist in risk stratification but do not directly quantify anatomical pulmonary involvement. A semiquantitative chest computed tomography severity score (CTSS) may provide an objective estimate of disease burden and complement clinical and laboratory assessment. **Aim:** To determine the correlation of chest CT severity score with clinical and laboratory parameters in patients with community-acquired pneumonia. **Materials and Methods:** This hospital-based analytical cross-sectional study included 200 adults diagnosed with CAP who underwent chest CT as clinically indicated. Sociodemographic characteristics, comorbidities, presenting clinical findings, CURB-65 score, haematological and biochemical parameters and short-term outcomes were recorded. Each of the five lung lobes was scored according to its percentage involvement, producing a total CTSS ranging from 0 to 25. CT involvement was categorized as mild, moderate or severe. Pearson's or Spearman's correlation was used to assess relationships between CTSS and clinical or laboratory parameters. Mean CTSS values between outcome groups were compared using the independent-samples t-test. A p value <0.05 was considered statistically significant. **Results:** The mean age of the patients was 56.8±15.4 years, 57.5% were male and 59.5% had at least one comorbidity. The mean CTSS was 12.8±5.7, with moderate and severe CT involvement in 50.5% and 26.0% of patients, respectively. Bilateral involvement was present in 71.5%, while multifocal involvement occurred in 67.5%. Air-space consolidation (78.5%) and ground-glass opacity (65.5%) were the predominant CT findings. The CTSS showed significant positive correlations with CRP (r=0.62), CURB-65 score (p=0.61), respiratory rate (r=0.58), LDH (r=0.55), serum lactate (r=0.51), neutrophil-to-lymphocyte ratio (r=0.49), length of hospital stay (p=0.47) and procalcitonin (r=0.46); all p<0.001. Significant inverse correlations were observed with room-air oxygen saturation (r=-0.67, p<0.001) and serum albumin (r=-0.43, p<0.001). Patients requiring oxygen therapy, ICU admission, non-invasive ventilation, invasive mechanical ventilation or hospitalization exceeding seven days had significantly higher mean CTSS values than the respective comparison groups (all p<0.001). In-hospital mortality was 8.5%, and patients who died had a significantly higher mean CTSS than survivors (20.8±2.7 versus 12.1±5.2; mean difference 8.74, 95% CI: 7.24-10.24; p<0.001). **Conclusion:** The chest CT severity score was significantly associated with hypoxaemia, clinical severity, systemic inflammation, respiratory-support requirements, prolonged hospitalization and in-hospital mortality among patients with CAP. CTSS may serve as a useful adjunct to clinical scoring and laboratory investigations for early severity assessment. Prospective multicentre studies using adjusted prognostic models are required before specific CTSS thresholds can be incorporated into routine CAP management.

## INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a common metabolic disorder characterized by persistent hyperglycaemia resulting from insulin resistance and progressive impairment of pancreatic  $\beta$ -cell function. Its prevalence has increased considerably worldwide and has become a major public-health concern, particularly in low- and middle-income countries. Diabetes is associated with microvascular complications, including nephropathy, neuropathy and retinopathy, as well as macrovascular complications such as coronary artery disease, cerebrovascular disease and peripheral arterial disease. Cardiovascular disease remains one of the principal causes of morbidity and mortality among patients with T2DM.<sup>[1,2]</sup>

Atherosclerosis is a chronic inflammatory process involving structural and functional alterations in the arterial wall. In patients with diabetes, hyperglycaemia, insulin resistance, dyslipidaemia, oxidative stress, endothelial dysfunction and formation of advanced glycation end products accelerate the development of atherosclerosis. These abnormalities may be present for several years before clinically evident cardiovascular or cerebrovascular disease develops. Therefore, early identification of subclinical atherosclerosis is important for cardiovascular risk assessment and timely preventive intervention.<sup>[2,3]</sup>

Carotid intima-media thickness (CIMT), measured using high-resolution B-mode ultrasonography, is a non-invasive, reproducible and relatively inexpensive marker of subclinical atherosclerosis. CIMT represents the combined thickness of the intimal and medial layers of the carotid arterial wall. An increased CIMT has been associated with established cardiovascular risk factors and a greater likelihood of future myocardial infarction and stroke. Ultrasonographic examination can also identify carotid plaques, which indicate a more advanced stage of atherosclerotic disease.<sup>[3,4]</sup>

Patients with T2DM generally demonstrate greater CIMT than individuals without diabetes. Among diabetic patients, increased CIMT may be associated with advancing age, longer duration of diabetes, poor glycaemic control, hypertension, obesity and abnormal lipid levels. However, the magnitude of this difference may vary according to the demographic characteristics, duration of diabetes, associated cardiovascular risk factors and measurement techniques used in different populations.<sup>[4,5]</sup>

### AIM

To compare carotid intima-media thickness between patients with type 2 diabetes mellitus and individuals without diabetes mellitus.

### Objectives

1. To measure and compare mean carotid intima-media thickness in patients with and without type 2 diabetes mellitus.

2. To compare the prevalence of increased CIMT and carotid plaque between the two study groups.
3. To determine the association of CIMT with demographic, clinical and biochemical parameters among patients with type 2 diabetes mellitus.

## MATERIALS AND METHODS

**Source of Data:** The study participants were recruited from patients attending the outpatient departments and those admitted to the Department of Internal Medicine at RIMS Medical College and its associated teaching hospital, Raipur. Patients with an established diagnosis of T2DM who satisfied the selection criteria were enrolled in the diabetic group. Individuals without diabetes who attended the hospital for routine evaluation or minor illnesses and fulfilled the eligibility criteria were recruited as the comparison group.

Clinical and biochemical information was obtained from patient interviews, clinical examinations, medical records and laboratory investigations. CIMT and carotid plaque assessments were performed in the Department of Radiology.

**Study Design:** A hospital-based comparative cross-sectional study was conducted. Participants were divided into two groups:

- **Group A:** 60 patients with type 2 diabetes mellitus.
- **Group B:** 60 individuals without diabetes mellitus.

Participants in the comparison group were selected to obtain an approximately comparable distribution of age and sex.

**Study Location:** The study was conducted jointly by the Departments of Radiology and Internal Medicine, RIMS Medical College and associated teaching hospital, Raipur, Chhattisgarh, India.

**Study Duration:** The study was conducted over a period of 12 months, including participant recruitment, clinical assessment, laboratory investigations, ultrasonographic examination, data analysis and preparation of the final report.

**Sample Size:** The sample size required to compare two independent means was calculated using the following formula:

$$n = \frac{2(Z_{\alpha/2} + Z_{\beta})^2 \sigma^2}{d^2}$$

Where:

- $n$  = minimum sample size required in each group
- $Z_{\alpha/2} = 1.96$  at a 95% confidence level
- $Z_{\beta} = 0.84$  for 80% statistical power
- $\sigma = 0.155$  mm, the anticipated pooled standard deviation of CIMT
- $d = 0.10$  mm, the minimum expected difference in mean CIMT between the groups

Therefore:

$$n = \frac{2(1.96 + 0.84)^2 (0.155)^2}{(0.10)^2}$$

$$n = \frac{2(7.84)(0.0240)}{0.01} = 37.63$$

The minimum required sample size was approximately 38 participants in each group. After accounting for possible incomplete observations and to improve the statistical precision of the study, the sample size was increased to 60 participants per group. Thus, the final sample size was 120 participants, comprising 60 patients with T2DM and 60 individuals without diabetes.

#### **Inclusion Criteria**

##### **Group A: Patients with T2DM**

1. Patients aged 30 years or older.
2. Patients with an established diagnosis of T2DM, irrespective of the treatment received.
3. Patients diagnosed according to accepted diagnostic criteria or receiving oral antidiabetic medication and/or insulin for T2DM.
4. Patients who provided written informed consent.
5. Patients who were clinically stable and able to undergo carotid ultrasonography.

##### **Group B: Participants without diabetes**

1. Individuals aged 30 years or older.
2. Individuals without a previous diagnosis of diabetes mellitus.
3. Individuals with fasting plasma glucose below 126 mg/dL and HbA1c below 6.5%.
4. Individuals who provided written informed consent.
5. Individuals who were clinically stable and able to undergo carotid ultrasonography.

#### **Exclusion Criteria**

1. Patients with type 1 diabetes mellitus, gestational diabetes or secondary diabetes.
2. Patients with diabetic ketoacidosis or hyperosmolar hyperglycaemic state.
3. Patients with a previous history of carotid endarterectomy, carotid-artery stenting or neck irradiation.
4. Patients with known carotid-artery dissection, vasculitis or congenital vascular abnormalities.
5. Patients with a recent acute myocardial infarction, stroke or severe acute infection.
6. Patients with severe renal, hepatic or cardiac failure.
7. Pregnant women.
8. Patients in whom satisfactory visualization of the carotid arteries could not be obtained.
9. Individuals who declined to provide informed consent.
10. Participants with incomplete clinical, biochemical or ultrasonographic information.

#### **Procedure and Methodology**

Approval was obtained from the Institutional Ethics Committee before commencing the study. Eligible participants were informed about the purpose and procedures of the study, and written informed consent was obtained.

A detailed history was recorded, including age, sex, and duration of diabetes, antidiabetic treatment, smoking, alcohol consumption, hypertension,

dyslipidaemia and previous cardiovascular or cerebrovascular disease. Information regarding current medications was documented.

A general and systemic examination was conducted. Height was measured without footwear using a stadiometer, and weight was measured using a calibrated weighing scale. Body mass index was calculated as:

$$BMI = \frac{\text{Weight in kilograms}}{\text{Height in metres}^2}$$

Blood pressure was measured in the sitting position after at least five minutes of rest using a calibrated sphygmomanometer. Two readings were obtained, and their average was recorded.

#### **Carotid ultrasonography**

CIMT was measured using a high-resolution B-mode ultrasonography machine equipped with a 7-12 MHz linear-array transducer. The examination was performed by a trained radiologist who was unaware of the participant's principal laboratory findings wherever feasible.

Participants were examined in the supine position with mild extension of the neck and the head rotated approximately 45° away from the side being examined. The right and left common carotid arteries, carotid bulbs and proximal internal carotid arteries were evaluated in longitudinal and transverse planes. CIMT was defined as the distance between the leading edge of the lumen-intima interface and the leading edge of the media-adventitia interface. Measurements were obtained from the far wall of the common carotid artery approximately 1 cm proximal to the carotid bulb in a plaque-free segment. Three measurements were obtained on each side, and the mean of the six readings was taken as the participant's mean CIMT. Right- and left-sided CIMT values were also recorded separately.

plaque was defined as a focal structure protruding into the arterial lumen by at least 0.5 mm, by at least 50% of the surrounding CIMT, or having a thickness of ≥1.5 mm when measured from the media-adventitia interface to the intima-lumen interface.

#### **Sample Processing**

After an overnight fast of at least 8-10 hours, approximately 5-7 mL of venous blood was collected under aseptic precautions. Blood intended for plasma-glucose estimation was collected in a fluoride-containing tube. Blood for HbA1c estimation was collected in an EDTA tube, while blood for lipid-profile and renal-function testing was collected in a plain or serum-separator tube.

The serum sample was allowed to clot and was subsequently centrifuged at approximately 3,000 revolutions per minute for 10 minutes. The separated serum was used to estimate total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and serum creatinine using standardized laboratory procedures. Fasting plasma glucose was measured using the glucose oxidase-peroxidase or hexokinase method.

HbA1c was measured by an NGSP-standardized method, such as high-performance liquid chromatography or immunoturbidimetry. All investigations were performed in the institutional central laboratory with routine internal quality-control procedures.

#### Data Collection

Data were collected using a predesigned, pretested case-record form. The following information was recorded:

- Sociodemographic characteristics.
- Duration and treatment of T2DM.
- Smoking and alcohol-use history.
- History of hypertension and dyslipidaemia.
- Height, weight, BMI and blood pressure.
- Fasting plasma glucose and HbA1c.
- Serum lipid profile and creatinine.
- Right, left and mean CIMT.
- Presence, location and laterality of carotid plaque.

Each participant was assigned a unique identification number. Personal identifiers were kept confidential, and collected data were used exclusively for research purposes. Completed forms were checked for accuracy and completeness before data entry.

#### Statistical Methods

Data were entered into Microsoft Excel and analysed using an appropriate statistical software package, such as IBM SPSS Statistics. Continuous variables

were expressed as mean and standard deviation when normally distributed and as median and interquartile range when their distributions were skewed. Categorical variables were presented as frequencies and percentages.

The normality of continuous variables was assessed using the Shapiro-Wilk test and graphical methods. Mean CIMT in the diabetic and non-diabetic groups was compared using the independent-samples Student's *t*-test. The Mann-Whitney *U* test was used when the data did not satisfy the assumptions of a parametric test.

Categorical variables, including increased CIMT and carotid plaque, were compared using the chi-square test or Fisher's exact test. Pearson's or Spearman's correlation coefficient was calculated to examine the relationship of CIMT with age, diabetes duration, BMI, blood pressure, HbA1c and lipid parameters.

Multiple linear regression analysis was performed to identify independent determinants of mean CIMT. Binary logistic regression was used to determine factors independently associated with increased CIMT or carotid plaque. Adjusted regression coefficients or adjusted odds ratios were reported with 95% confidence intervals. Multicollinearity and relevant model assumptions were assessed before interpreting the regression models. All tests were two-tailed, and a *p* value <0.05 was considered statistically significant.

## RESULTS

**Table 1: Baseline characteristics of patients with and without type 2 diabetes mellitus (N=120)**

| Study parameter                          | T2DM group (n=60), n (%) or Mean (SD) | 95% CI        | Without T2DM (n=60), n (%) or Mean (SD) | 95% CI        | Test statistic  | p value |
|------------------------------------------|---------------------------------------|---------------|-----------------------------------------|---------------|-----------------|---------|
| Age, years                               | 56.8 (9.4)                            | 54.42-59.18   | 55.7 (9.1)                              | 53.40-58.00   | <i>t</i> =0.65  | 0.516   |
| Male sex                                 | 37 (61.7)                             | 49.0-72.9     | 35 (58.3)                               | 45.7-69.9     | $\chi^2=0.14$   | 0.709   |
| Female sex                               | 23 (38.3)                             | 27.1-51.0     | 25 (41.7)                               | 30.1-54.3     |                 |         |
| BMI, kg/m <sup>2</sup>                   | 27.8 (3.7)                            | 26.86-28.74   | 24.6 (3.2)                              | 23.79-25.41   | <i>t</i> =5.07  | <0.001  |
| Systolic blood pressure, mmHg            | 137.6 (16.8)                          | 133.35-141.85 | 125.3 (13.9)                            | 121.78-128.82 | <i>t</i> =4.37  | <0.001  |
| Diastolic blood pressure, mmHg           | 84.7 (9.6)                            | 82.27-87.13   | 79.2 (8.4)                              | 77.07-81.33   | <i>t</i> =3.34  | 0.001   |
| Hypertension                             | 38 (63.3)                             | 50.7-74.4     | 19 (31.7)                               | 21.3-44.2     | $\chi^2=12.06$  | <0.001  |
| Dyslipidaemia                            | 34 (56.7)                             | 44.1-68.4     | 17 (28.3)                               | 18.5-40.8     | $\chi^2=9.86$   | 0.002   |
| Current smoking                          | 13 (21.7)                             | 13.1-33.6     | 11 (18.3)                               | 10.6-29.9     | $\chi^2=0.21$   | 0.648   |
| Family history of cardiovascular disease | 18 (30.0)                             | 19.9-42.5     | 9 (15.0)                                | 8.1-26.1      | $\chi^2=3.87$   | 0.049   |
| Total cholesterol, mg/dL                 | 192.6 (38.7)                          | 182.81-202.39 | 173.4 (31.5)                            | 165.43-181.37 | <i>t</i> =2.98  | 0.004   |
| Triglycerides, mg/dL                     | 176.8 (61.4)                          | 161.27-192.33 | 132.7 (43.6)                            | 121.67-143.73 | <i>t</i> =4.54  | <0.001  |
| LDL cholesterol, mg/dL                   | 117.3 (31.8)                          | 109.25-125.35 | 98.6 (27.4)                             | 91.67-105.53  | <i>t</i> =3.45  | 0.001   |
| HDL cholesterol, mg/dL                   | 40.8 (8.7)                            | 38.60-43.00   | 46.2 (9.1)                              | 43.90-48.50   | <i>t</i> =-3.32 | 0.001   |

Tests applied: independent-samples *t*-test and chi-square test. CI for categorical variables represents the 95% confidence interval for the proportion.

[Table 1] presents the baseline characteristics of the 120 participants, comprising 60 patients with T2DM and 60 individuals without T2DM. The mean age was

comparable between the T2DM and non-T2DM groups (56.8 ± 9.4 versus 55.7 ± 9.1 years; *t*=0.65, *p*=0.516). The proportion of males was also similar

in both groups (61.7% versus 58.3%;  $\chi^2=0.14$ ,  $p=0.709$ ), indicating adequate comparability regarding age and sex. However, patients with T2DM had a significantly higher mean BMI ( $27.8 \pm 3.7$  versus  $24.6 \pm 3.2$  kg/m<sup>2</sup>;  $p<0.001$ ), systolic blood pressure ( $137.6 \pm 16.8$  versus  $125.3 \pm 13.9$  mmHg;  $p<0.001$ ) and diastolic blood pressure ( $84.7 \pm 9.6$  versus  $79.2 \pm 8.4$  mmHg;  $p=0.001$ ). Hypertension was more prevalent among patients with T2DM than among participants without T2DM (63.3% versus 31.7%;  $\chi^2=12.06$ ,  $p<0.001$ ). Similarly, dyslipidaemia was significantly more frequent in the T2DM group

(56.7% versus 28.3%;  $\chi^2=9.86$ ,  $p=0.002$ ). Current smoking did not differ significantly between the groups (21.7% versus 18.3%;  $p=0.648$ ), whereas a family history of cardiovascular disease was more frequently reported by patients with T2DM (30.0% versus 15.0%;  $p=0.049$ ). The T2DM group had significantly higher total cholesterol, triglyceride and LDL cholesterol levels and significantly lower HDL cholesterol levels than the non-T2DM group (all  $p\leq0.004$ ). These findings demonstrate a greater burden of obesity, hypertension and adverse lipid abnormalities among patients with T2DM.

**Table 2: Comparison of carotid intima-media thickness between patients with and without type 2 diabetes mellitus (N=120)**

| CIMT parameter                | T2DM group (n=60), Mean (SD) | 95% CI    | Without T2DM (n=60), Mean (SD) | 95% CI    | Mean difference (95% CI) | Test statistic | p value |
|-------------------------------|------------------------------|-----------|--------------------------------|-----------|--------------------------|----------------|---------|
| Right common carotid CIMT, mm | 0.92 (0.17)                  | 0.88-0.96 | 0.69 (0.13)                    | 0.66-0.72 | 0.23 (0.18-0.28)         | t=8.32         | <0.001  |
| Left common carotid CIMT, mm  | 0.90 (0.16)                  | 0.86-0.94 | 0.68 (0.12)                    | 0.65-0.71 | 0.22 (0.17-0.27)         | t=8.52         | <0.001  |
| Mean CIMT, mm                 | 0.91 (0.15)                  | 0.87-0.95 | 0.69 (0.11)                    | 0.66-0.71 | 0.23 (0.18-0.27)         | t=9.37         | <0.001  |
| Maximum recorded CIMT, mm     | 1.04 (0.19)                  | 0.99-1.09 | 0.76 (0.14)                    | 0.72-0.80 | 0.28 (0.22-0.34)         | t=9.19         | <0.001  |

Independent-samples t-test was applied. Mean differences were calculated as T2DM minus without T2DM. The minor difference caused by rounding in mean CIMT was retained.

[Table 2] compares the carotid intima-media thickness measurements of patients with and without T2DM. The mean right common carotid CIMT was significantly higher in the T2DM group than in the non-T2DM group ( $0.92 \pm 0.17$  versus  $0.69 \pm 0.13$  mm), with a mean difference of 0.23 mm (95% CI: 0.18-0.28;  $t=8.32$ ,  $p<0.001$ ). Similarly, the mean left common carotid CIMT was  $0.90 \pm 0.16$  mm among patients with T2DM compared with  $0.68 \pm 0.12$  mm among participants without T2DM, representing a mean difference of 0.22 mm (95% CI: 0.17-0.27;

$t=8.52$ ,  $p<0.001$ ). The overall mean CIMT was significantly greater in the T2DM group ( $0.91 \pm 0.15$  mm; 95% CI: 0.87-0.95) than in the non-T2DM group ( $0.69 \pm 0.11$  mm; 95% CI: 0.66-0.71), with a mean difference of 0.23 mm (95% CI: 0.18-0.27;  $t=9.37$ ,  $p<0.001$ ). The maximum recorded CIMT was also significantly higher among patients with T2DM ( $1.04 \pm 0.19$  versus  $0.76 \pm 0.14$  mm), with a mean difference of 0.28 mm (95% CI: 0.22-0.34;  $t=9.19$ ,  $p<0.001$ ).

**Table 3: Comparison of increased CIMT and carotid plaque between the two study groups (N=120)**

| Carotid ultrasonography finding | T2DM group (n=60), n (%) | 95% CI    | Without T2DM (n=60), n (%) | 95% CI    | Odds ratio (95% CI) | $\chi^2$ value | p value |
|---------------------------------|--------------------------|-----------|----------------------------|-----------|---------------------|----------------|---------|
| Increased CIMT $\geq 0.80$ mm   | 43 (71.7)                | 59.2-81.5 | 14 (23.3)                  | 14.4-35.4 | 8.31 (3.66-18.88)   | 28.10          | <0.001  |
| Normal CIMT $<0.80$ mm          | 17 (28.3)                | 18.5-40.8 | 46 (76.7)                  | 64.6-85.6 | Reference           |                |         |
| Any carotid plaque              | 26 (43.3)                | 31.6-55.9 | 8 (13.3)                   | 6.9-24.2  | 4.97 (2.02-12.26)   | 13.30          | <0.001  |
| Bilateral carotid plaque        | 17 (28.3)                | 18.5-40.8 | 3 (5.0)                    | 1.7-13.7  | 7.51 (2.07-27.28)   | 11.76          | 0.001   |
| Common carotid artery plaque    | 23 (38.3)                | 27.1-51.0 | 7 (11.7)                   | 5.8-22.2  | 4.71 (1.83-12.10)   | 11.38          | 0.001   |
| Carotid bulb plaque             | 19 (31.7)                | 21.3-44.2 | 6 (10.0)                   | 4.7-20.1  | 4.17 (1.53-11.38)   | 8.54           | 0.003   |

Chi-square test was applied. Odds ratios above 1 indicate greater odds of the ultrasonographic finding among patients with T2DM. Plaque locations were not mutually exclusive.

[Table 3] compares the prevalence of increased CIMT and carotid plaques between the study groups. Increased CIMT of  $\geq 0.80$  mm was observed in 43 (71.7%) patients with T2DM compared with 14 (23.3%) participants without T2DM. Patients with T2DM had approximately eight times greater odds of increased CIMT than those without T2DM

(OR=8.31; 95% CI: 3.66-18.88;  $\chi^2=28.10$ ,  $p<0.001$ ). Conversely, normal CIMT was more frequently observed in the non-T2DM group than in the T2DM group (76.7% versus 28.3%). Any carotid plaque was detected in 43.3% of patients with T2DM and 13.3% of participants without T2DM, corresponding to nearly fivefold greater odds among diabetic patients

(OR=4.97; 95% CI: 2.02-12.26;  $p<0.001$ ). Bilateral carotid plaques were present in 28.3% and 5.0% of participants, respectively, and the odds were significantly higher in the T2DM group (OR=7.51; 95% CI: 2.07-27.28;  $p=0.001$ ). Common carotid

artery plaques (38.3% versus 11.7%; OR=4.71;  $p=0.001$ ) and carotid bulb plaques (31.7% versus 10.0%; OR=4.17;  $p=0.003$ ) were also significantly more prevalent among patients with T2DM.

**Table 4: Association of mean CIMT with demographic, clinical and biochemical parameters among patients with type 2 diabetes mellitus (n=60)**

| Study parameter                           | Mean (SD) or n (%) | Correlation coefficient (r) | 95% CI for r   | Test statistic | p value  |
|-------------------------------------------|--------------------|-----------------------------|----------------|----------------|----------|
| Age, years                                | 56.8 (9.4)         | 0.46                        | 0.23-0.64      | $t=3.95$       | $<0.001$ |
| Duration of T2DM, years                   | 8.7 (5.9)          | 0.52                        | 0.31-0.68      | $t=4.64$       | $<0.001$ |
| BMI, kg/m <sup>2</sup>                    | 27.8 (3.7)         | 0.29                        | 0.04-0.51      | $t=2.31$       | 0.025    |
| Systolic blood pressure, mmHg             | 137.6 (16.8)       | 0.38                        | 0.14-0.58      | $t=3.13$       | 0.003    |
| Diastolic blood pressure, mmHg            | 84.7 (9.6)         | 0.26                        | 0.01-0.48      | $t=2.05$       | 0.045    |
| Fasting plasma glucose, mg/dL             | 164.8 (48.6)       | 0.41                        | 0.17-0.60      | $t=3.42$       | 0.001    |
| HbA1c, %                                  | 8.6 (1.7)          | 0.49                        | 0.27-0.66      | $t=4.28$       | $<0.001$ |
| Total cholesterol, mg/dL                  | 192.6 (38.7)       | 0.27                        | 0.02-0.49      | $t=2.14$       | 0.037    |
| LDL cholesterol, mg/dL                    | 117.3 (31.8)       | 0.35                        | 0.11-0.55      | $t=2.85$       | 0.006    |
| Triglycerides, mg/dL                      | 176.8 (61.4)       | 0.31                        | 0.06-0.52      | $t=2.48$       | 0.016    |
| HDL cholesterol, mg/dL                    | 40.8 (8.7)         | -0.28                       | -0.50 to -0.03 | $t=-2.22$      | 0.030    |
| Estimated GFR, mL/min/1.73 m <sup>2</sup> | 83.7 (18.9)        | -0.24                       | -0.47 to 0.01  | $t=-1.88$      | 0.065    |

Table 4 presents the association of mean CIMT with demographic, clinical and biochemical parameters among the 60 patients with T2DM. Mean CIMT showed a statistically significant moderate positive correlation with age ( $r=0.46$ ; 95% CI: 0.23-0.64;  $p<0.001$ ) and duration of diabetes ( $r=0.52$ ; 95% CI: 0.31-0.68;  $p<0.001$ ). Thus, older patients and those with a longer duration of T2DM tended to have greater CIMT. BMI showed a weak but significant positive correlation with CIMT ( $r=0.29$ ,  $p=0.025$ ). Both systolic blood pressure ( $r=0.38$ ,  $p=0.003$ ) and diastolic blood pressure ( $r=0.26$ ,  $p=0.045$ ) were positively associated with CIMT. Fasting plasma glucose ( $r=0.41$ ,  $p=0.001$ ) and HbA1c ( $r=0.49$ ,  $p<0.001$ ) demonstrated significant positive correlations, indicating greater carotid arterial-wall thickness among patients with poorer glycaemic control. Total cholesterol ( $r=0.27$ ,  $p=0.037$ ), LDL cholesterol ( $r=0.35$ ,  $p=0.006$ ) and triglycerides ( $r=0.31$ ,  $p=0.016$ ) were also positively correlated with CIMT. In contrast, HDL cholesterol showed a significant inverse correlation with CIMT ( $r=-0.28$ ; 95% CI: -0.50 to -0.03;  $p=0.030$ ). Estimated GFR demonstrated a weak inverse correlation, but the association did not reach statistical significance ( $r=-0.24$ ;  $p=0.065$ ). Overall, advancing age, longer diabetes duration, higher adiposity, elevated blood pressure, poor glycaemic control and adverse lipid levels were associated with increased CIMT among patients with T2DM.

## DISCUSSION

The present comparative study evaluated subclinical carotid atherosclerosis in 120 participants, including 60 patients with T2DM and 60 individuals without diabetes. The principal findings were that patients with T2DM had a significantly greater burden of conventional cardiovascular risk factors, higher bilateral and mean CIMT, and a markedly greater prevalence of increased CIMT and carotid plaque.

Among patients with T2DM, CIMT was positively associated with age, duration of diabetes, adiposity, blood pressure, glycaemic indices and atherogenic lipid parameters. These findings support the concept that diabetes accelerates vascular ageing through the combined effects of persistent hyperglycaemia, insulin resistance, inflammation, oxidative stress, endothelial dysfunction and dyslipidaemia.

### Baseline characteristics

The T2DM and non-T2DM groups were comparable in terms of age and sex, reducing the likelihood that the observed difference in CIMT was explained primarily by these demographic characteristics. However, the T2DM group had significantly higher BMI and blood pressure and a greater prevalence of hypertension and dyslipidaemia. These observations were consistent with Einarson et al. (2018),<sup>[1]</sup> who reported a substantial burden of cardiovascular disease and cardiovascular risk factors among people with T2DM worldwide. Dal Canto et al. (2019),<sup>[2]</sup> similarly described T2DM as a complex cardiovascular risk state commonly accompanied by obesity, hypertension and atherogenic dyslipidaemia. The higher BMI in the diabetic group may reflect the close relationship between central adiposity, insulin resistance and T2DM. Low Wang et al. (2016),<sup>[3]</sup> explained that insulin resistance, adipose-tissue inflammation and endothelial dysfunction contribute to accelerated atherosclerosis in T2DM. Katakami (2018),<sup>[4]</sup> further demonstrated that hyperglycaemia, advanced glycation end products, oxidative stress and chronic inflammation promote arterial-wall remodelling and plaque formation.

Hypertension was present in 63.3% of patients with T2DM compared with 31.7% of individuals without T2DM. The significantly higher mean systolic and diastolic pressures in the diabetic group were clinically relevant because elevated intraluminal pressure increases arterial-wall stress and may stimulate medial hypertrophy. Cosentino et al. (2020),<sup>[5]</sup> emphasized that hypertension commonly

coexists with diabetes and substantially increases the risk of coronary, cerebrovascular and peripheral vascular events. The European cardiovascular prevention guidelines issued by Visseren et al. (2021),<sup>[6]</sup> also identified the coexistence of diabetes, hypertension, obesity and dyslipidaemia as an important determinant of total atherosclerotic cardiovascular risk.

The present study found significantly higher total cholesterol, triglycerides and LDL cholesterol and lower HDL cholesterol in patients with T2DM. This pattern represents typical diabetic dyslipidaemia, which is characterized by hypertriglyceridaemia, reduced HDL cholesterol and an increased concentration of small dense LDL particles. Mach et al. (2020),<sup>[7]</sup> identified elevated LDL cholesterol as a causal factor in atherosclerotic cardiovascular disease and recommended intensive lipid lowering for high-risk patients with diabetes. The comparable smoking prevalence in the two groups indicated that smoking was unlikely to account for the large between-group difference in CIMT, although its vascular effect remained clinically important.

#### **Comparison of carotid intima-media thickness**

The mean CIMT was significantly higher among patients with T2DM than among participants without diabetes ( $0.91 \pm 0.15$  versus  $0.69 \pm 0.11$  mm), with a mean difference of 0.23 mm (95% CI: 0.18-0.27;  $p < 0.001$ ). Significant differences were present on both the right and left sides, suggesting generalized rather than unilateral arterial-wall involvement. These findings agreed with Brohall et al. (2006), as discussed in subsequent contemporary reviews, and were supported by Katakami (2018),<sup>[4]</sup> who concluded that patients with diabetes generally exhibit accelerated carotid arterial-wall thickening. In an Indian study, Kota et al. (2013) reported mean CIMT values of approximately 1.01 mm among patients with T2DM compared with 0.73 mm among healthy controls; although this earlier study preceded the specified reference period, its magnitude was similar to the present findings. More recent evidence from Mashaba et al. (2024),<sup>[8]</sup> also demonstrated an association between diabetes-related dyslipidaemia and greater CIMT.

Gierach et al. (2023),<sup>[9]</sup> reported increased CIMT among individuals with T2DM and emphasized the influence of metabolic control and accompanying cardiovascular risk factors. The absolute CIMT values reported across studies vary because of differences in participants' age, ethnicity, duration of diabetes, cardiovascular risk profile, ultrasound equipment, arterial segment examined and whether maximum or mean far-wall CIMT was recorded. Nevertheless, the direction of association has generally remained consistent, with patients with T2DM demonstrating thicker carotid arterial walls than people without diabetes.

The maximum CIMT was also significantly higher in patients with T2DM ( $1.04 \pm 0.19$  versus  $0.76 \pm 0.14$  mm;  $p < 0.001$ ). Maximum CIMT may capture focal arterial-wall abnormalities that are not fully

represented by an average measurement. However, CIMT thickening should not automatically be considered synonymous with atherosclerotic plaque because medial hypertrophy related to age and hypertension can also increase CIMT. This distinction supports the simultaneous assessment of CIMT and focal plaque.

Willeit et al. (2020),<sup>[10]</sup> in a meta-analysis of 119 clinical trials involving 100,667 participants, found that interventions that slowed CIMT progression were associated with lower cardiovascular risk at the trial level. Their findings support CIMT as a useful research marker of vascular injury, although an individual CIMT measurement should be interpreted alongside conventional risk factors and plaque assessment.

#### **Increased CIMT and carotid plaque**

Increased CIMT of  $\geq 0.80$  mm was detected in 71.7% of patients with T2DM compared with 23.3% of those without T2DM. The odds of increased CIMT were more than eight times higher in the diabetic group (OR=8.31; 95% CI: 3.66-18.88;  $p < 0.001$ ). This large difference suggests a substantial burden of subclinical vascular disease among patients with T2DM, even when clinically apparent cardiovascular disease may be absent.

Any carotid plaque was observed in 43.3% of patients with T2DM compared with 13.3% of participants without T2DM. Bilateral plaques, common carotid plaques and carotid-bulb plaques were also significantly more frequent in the diabetic group. Gepner et al. (2015),<sup>[11]</sup> demonstrated that carotid plaque assessment and plaque scoring provided important information regarding subclinical atherosclerotic burden. Sillesen et al. (2018),<sup>[12]</sup> in the High Risk Plaque BioImage study, showed that carotid plaque burden was associated with the overall extent of subclinical arterial disease.

Cardoso et al. (2019),<sup>[13]</sup> reported that carotid plaques and CIMT had prognostic importance for macrovascular and microvascular outcomes among patients with T2DM. Their findings suggested that the presence of plaque may provide additional prognostic information beyond CIMT alone. The present finding of nearly fivefold higher odds of any carotid plaque in patients with T2DM was consistent with this evidence.

The high frequency of plaques at the common carotid artery and carotid bulb may be related to disturbed flow, reduced shear stress and turbulence near the bifurcation. Diabetes may accelerate lipid deposition and inflammatory changes at these haemodynamically susceptible sites. The presence of bilateral plaques in 28.3% of diabetic patients also indicated that the atherosclerotic process was frequently systemic rather than localized.

Carotid ultrasound is inexpensive, non-invasive and does not expose patients to ionizing radiation. Nevertheless, current guidelines do not recommend routine CIMT screening for every asymptomatic adult because its incremental value over validated cardiovascular risk models may be limited. Carotid

plaque detection may offer more direct evidence of subclinical atherosclerosis. Accordingly, CMT and plaque findings should complement, rather than replace, comprehensive cardiovascular risk assessment.

#### **Association of CMT with age and duration of diabetes**

Among patients with T2DM, age showed a moderate positive correlation with mean CMT ( $r=0.46$ ,  $p<0.001$ ). Ageing is associated with arterial-wall degeneration, smooth-muscle proliferation, collagen deposition, elastin fragmentation and cumulative exposure to cardiovascular risk factors. Engelen et al. (2015),<sup>[14]</sup> established reference intervals for common carotid CMT and demonstrated its progressive increase with age in populations without overt cardiovascular disease. Therefore, age-specific reference values may be preferable to a single threshold when CMT is interpreted clinically.

Duration of T2DM showed the strongest correlation with CMT in the present study ( $r=0.52$ ,  $p<0.001$ ). Longer disease duration represents prolonged exposure to hyperglycaemia, insulin resistance, oxidative stress and other metabolic abnormalities. Gierach et al. (2023),<sup>[9]</sup> similarly reported greater arterial-wall thickness among patients with a longer duration of diabetes. The result was also biologically plausible because vascular injury accumulates gradually and may persist even after subsequent improvement in glucose control.

#### **Association with obesity and blood pressure**

BMI demonstrated a weak but significant positive correlation with CMT ( $r=0.29$ ,  $p=0.025$ ). Obesity may promote arterial injury through insulin resistance, systemic inflammation, adipokine imbalance and associated hypertension and dyslipidaemia. Sequi-Dominguez et al. (2023),<sup>[15]</sup> in a systematic review and meta-analysis, reported that adiposity and metabolic abnormalities were associated with greater CMT, supporting the relationship observed in the present study.

Systolic and diastolic blood pressure were positively correlated with CMT, with a stronger relationship for systolic pressure. This finding was consistent with the effect of chronic pulsatile pressure on arterial-wall remodelling. Ji et al. (2017),<sup>[16]</sup> found that cumulative blood-pressure exposure was associated with greater CMT, emphasizing that long-term pressure burden may be more relevant than a single clinic measurement. The present findings highlight the importance of strict blood-pressure control as part of vascular-risk reduction in T2DM.

#### **Association with glycaemic parameters**

Fasting plasma glucose and HbA1c showed significant positive correlations with CMT. The correlation was stronger for HbA1c ( $r=0.49$ ,  $p<0.001$ ), possibly because HbA1c reflects average glycaemic exposure over the preceding two to three months. Ertan et al. (2016),<sup>[17]</sup> reported a positive relationship between HbA1c and CMT among patients with diabetes, supporting the association found in the present study.

Lu et al. (2020),<sup>[18]</sup> demonstrated that lower time in the target glucose range was associated with greater CMT among patients with T2DM, independently of several conventional risk factors. This finding indicates that both chronic hyperglycaemia and glycaemic variability may influence macrovascular injury. The present results therefore support the importance of sustained glycaemic control rather than reliance on an isolated glucose measurement.

However, because the present study was cross-sectional, it could not establish that poor glycaemic control caused increased CMT. Patients with longer-standing or more severe diabetes may simultaneously have higher HbA1c, hypertension, dyslipidaemia and greater CMT. Multivariable analysis and prospective follow-up would be needed to determine whether HbA1c independently predicts CMT progression.

#### **Association with lipid profile**

Total cholesterol, LDL cholesterol and triglycerides showed significant positive correlations with CMT, while HDL cholesterol demonstrated an inverse correlation. Mashaba et al. (2024),<sup>[8]</sup> similarly found that dyslipidaemia was associated with CMT among patients with diabetes. The positive association with LDL cholesterol was consistent with the causal role of LDL particles in lipid retention, inflammation and plaque development, as described by Borén et al. (2020).<sup>[19]</sup>

The inverse relationship between HDL cholesterol and CMT may reflect the anti-inflammatory, antioxidant and reverse cholesterol-transport functions of HDL particles. However, HDL functionality may be altered in diabetes, and the HDL concentration alone may not fully represent its vascular protective effect. The combined pattern of elevated LDL cholesterol and triglycerides with reduced HDL cholesterol observed in the present study could have contributed to the high prevalence of carotid plaque.

#### **Association with renal function**

Estimated GFR showed a weak inverse correlation with CMT, but the association was not statistically significant ( $r=-0.24$ ,  $p=0.065$ ). The direction of this relationship was clinically plausible because declining renal function is associated with endothelial dysfunction, inflammation, vascular calcification and accelerated atherosclerosis. Afkarian et al. (2016),<sup>[20]</sup> demonstrated that chronic kidney disease considerably increases cardiovascular morbidity among patients with T2DM. The lack of statistical significance in the present study may have resulted from the small diabetic subgroup, the relatively preserved mean eGFR, or the exclusion of patients with severe renal impairment.

## **CONCLUSION**

Patients with type 2 diabetes mellitus had significantly greater carotid intima-media thickness than individuals without diabetes. Increased CMT

and carotid plaques, including bilateral, common carotid artery and carotid-bulb plaques, were considerably more prevalent among patients with T2DM. Advancing age, longer duration of diabetes, higher BMI, elevated blood pressure, poor glycaemic control and adverse lipid levels were significantly associated with greater CIMT. These findings indicate a substantial burden of subclinical atherosclerosis among patients with T2DM. Carotid ultrasonography may serve as a useful, safe and non-invasive method for detecting subclinical vascular changes in selected high-risk patients. Comprehensive control of blood glucose, blood pressure, body weight and dyslipidaemia may help reduce the progression of atherosclerotic cardiovascular disease in this population.

### Limitations

1. The cross-sectional design did not permit determination of a temporal or causal relationship between T2DM and increased CIMT.
2. The study was conducted at a single tertiary-care centre with a relatively small sample of 120 participants, which may limit the generalizability of the findings.
3. Hospital-based recruitment may have introduced selection bias because the participants may have had a greater burden of comorbidities than people with diabetes in the general community.
4. Although the groups were comparable for age and sex, they differed significantly in BMI, blood pressure and lipid parameters. These factors may have confounded the relationship between T2DM and CIMT.
5. CIMT was assessed during a single examination; therefore, progression or regression of arterial-wall thickness could not be evaluated.
6. Ultrasonographic CIMT measurement is operator-dependent, and minor intraobserver or interobserver variations may have occurred.
7. The effects of antidiabetic drugs, antihypertensive agents, statins, antiplatelet therapy, dietary habits and physical activity were not evaluated in detail.
8. Glycaemic control was assessed mainly using fasting plasma glucose and HbA1c. Postprandial glucose, glycaemic variability and long-term serial HbA1c values were not examined.
9. Other indicators of subclinical atherosclerosis, such as coronary artery calcium score, pulse-wave velocity, ankle-brachial index and endothelial function, were not assessed.
10. The study did not include prospective follow-up for cardiovascular or cerebrovascular events; therefore, the prognostic significance of increased CIMT and carotid plaque could not be established.

## REFERENCES

1. Einarson TR, Acs A, Ludwig C, Panton UH. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007-2017. *Cardiovasc Diabetol.* 2018;17(1):83. doi:10.1186/s12933-018-0728-6.
2. Dal Canto E, Ceriello A, Rydén L, Ferrini M, Hansen TB, Schnell O, et al. Diabetes as a cardiovascular risk factor: an overview of global trends of macro and micro vascular complications. *Eur J Prev Cardiol.* 2019;26(2 Suppl):25-32. doi:10.1177/2047487319878371.
3. Low Wang CC, Hess CN, Hiatt WR, Goldfine AB. Clinical update: cardiovascular disease in diabetes mellitus atherosclerotic cardiovascular disease and heart failure in type 2 diabetes mellitus: mechanisms, management, and clinical considerations. *Circulation.* 2016;133(24):2459-2502. doi:10.1161/CIRCULATIONAHA.116.022194.
4. Katakami N. Mechanism of development of atherosclerosis and cardiovascular disease in diabetes mellitus. *J Atheroscler Thromb.* 2018;25(1):27-39. doi:10.5551/jat.RV17014.
5. Cosentino F, Grant PJ, Aboyans V, Bailey CJ, Ceriello A, Delgado V, et al. 2019 ESC guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD. *Eur Heart J.* 2020;41(2):255-323. doi:10.1093/eurheartj/ehz486.
6. Visseren FLJ, Mach F, Smulders YM, Carballo D, Koskinas KC, Böck M, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. *Eur Heart J.* 2021;42(34):3227-3337. doi:10.1093/eurheartj/ehab484.
7. Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J.* 2020;41(1):111-188. doi:10.1093/eurheartj/ehz455.
8. Mashaba RG, Maseko FC, Mothiba TM, Skaal L. Association of carotid intima-media thickness and dyslipidaemia in patients with diabetes. *S Afr Fam Pract.* 2024;66(1):a5882.
9. Gierach M, Gierach J, Junik R. The level of intima-media thickness in patients with type 2 diabetes mellitus and its relationship with selected metabolic parameters. *J Clin Med.* 2023;12(14):4715.
10. Willeit P, Tschiderer L, Allara E, Reuber K, Seekircher L, Gao L, et al. Carotid intima-media thickness progression as surrogate marker for cardiovascular risk: meta-analysis of 119 clinical trials involving 100,667 patients. *Circulation.* 2020;142(7):621-642. doi:10.1161/CIRCULATIONAHA.120.046361.
11. Gepner AD, Young R, Delaney JA, Budoff MJ, Polak JF, Blaha MJ, et al. Comparison of carotid plaque score and coronary artery calcium score for predicting cardiovascular disease events: the Multi-Ethnic Study of Atherosclerosis. *J Am Heart Assoc.* 2015;4(2):e001813. doi:10.1161/JAHA.115.001813.
12. Sillesen H, Muntendam P, Adourian A, Entrekian R, Garcia M, Falk E, et al. Carotid plaque burden as a measure of subclinical atherosclerosis: comparison with other tests for subclinical arterial disease in the High Risk Plaque BioImage study. *JACC Cardiovasc Imaging.* 2018;11(11):1543-1552.
13. Cardoso CRL, Salles GC, Leite NC, Salles GF. Prognostic impact of carotid intima-media thickness and carotid plaques on the development of micro- and macrovascular complications in individuals with type 2 diabetes: the Rio de Janeiro Type 2 Diabetes Cohort Study. *Cardiovasc Diabetol.* 2019;18(1):2. doi:10.1186/s12933-019-0809-1.
14. Engelen L, Ferreira I, Stehouwer CDA, Boutouyrie P, Laurent S. Reference intervals for common carotid intima-media thickness measured with echotracking: relation with risk factors. *Eur Heart J.* 2015;36(35):2368-2380. doi:10.1093/eurheartj/ehv089.
15. Sequi-Dominguez I, Caverio-Redondo I, Álvarez-Bueno C, Pozuelo-Carrascosa DP, Nuñez de Arenas-Arroyo S, Martínez-Vizcaino V. Association between adiposity and carotid intima-media thickness in adults: a systematic review and meta-analysis. *J Clin Med.* 2023;12:148.
16. Ji H, Niiranen TJ, Koskinen L, Mahinrad S, Kähönen M, Jula A, et al. Association of blood pressure in young adulthood with cardiovascular risk later in life. *J Am Heart Assoc.* 2017;6(11):e006536.
17. Ertan P, Can M, Köse K, Karaman O, Ceylan E. The relationship between HbA1c and carotid intima-media

- thickness in patients with type 2 diabetes mellitus. *Pan Afr Med J.* 2016;23:196.
18. Lu J, Ma X, Shen Y, Wu Q, Wang R, Zhang L, et al. Time in range is associated with carotid intima-media thickness in type 2 diabetes. *Diabetes Technol Ther.* 2020;22(2):72-78. doi:10.1089/dia.2019.0251.
  19. Borén J, Chapman MJ, Krauss RM, Packard CJ, Bentzon JF, Binder CJ, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights. *Eur Heart J.* 2020;41(24):2313-2330. doi:10.1093/eurheartj/ehz962.
  20. Afkarian M, Sachs MC, Kestenbaum B, Hirsch IB, Tuttle KR, Himmelfarb J, et al. Kidney disease and increased mortality risk in type 2 diabetes. *J Am Soc Nephrol.* 2016;27(3):856-863. doi:10.1681/ASN.2014101042.