



Subclinical Coronary Artery Disease in Asymptomatic Type 2 Diabetes Mellitus: A Cross-Sectional Coronary CT Angiography Study from the Bundelkhand Region, India

Aditya Singh¹, Kuldeep Chandel², Shaily Panwar³, Qutubuddin⁴

¹Junior Resident, Department of General Medicine, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh

²Professor, Department of General Medicine, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh

³Associate Professor, Department of Radiodiagnosis, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh

⁴Assistant Professor, Department of General Medicine, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh

Corresponding Author: Dr. Aditya Singh

DOI: <https://doi.org/10.5281/zenodo.23529392>

Article Received 18-09-2026 / Accepted 13-10-2026 / Published 21-10-2026

ABSTRACT:

Background: Type 2 diabetes mellitus (T2DM) accelerates coronary atherosclerosis, which often stays silent until an acute event. We determined the prevalence and severity of subclinical coronary artery disease (CAD) in asymptomatic diabetic patients using coronary CT angiography (CCTA), compared with non-diabetic controls.

Aims/Objectives: The aim of this study was to determine the prevalence and severity of subclinical coronary artery disease in asymptomatic patients with type 2 diabetes mellitus using coronary CT angiography, and to compare the findings with non-diabetic individuals in the Bundelkhand region.

Methodology: In this cross-sectional study, 172 asymptomatic adults (86 T2DM, 86 non-diabetic controls) underwent CCTA with coronary artery calcium (CAC) scoring and CAD-RADS 2.0 classification at a tertiary centre (March 2025–February 2026). Controls were a convenience sample of accompanying attendants or relatives and were not age- or sex-matched; between-group comparisons are therefore unadjusted and descriptive.

Results: Obstructive CAD (CAD-RADS ≥ 3) occurred in 36/86 diabetics (41.9%) versus 0/86 controls (0.0%; $p < 0.001$). Any coronary plaque was present in 86/86 (100%) versus 26/85 (30.6%). Calcified plaque (40/86, 46.5% vs 1/86, 1.2%), CAC ≥ 100 (36/86, 41.9% vs 2/86, 2.3%) and multivessel disease (21/86, 24.4% vs 0/86, 0.0%) were commoner in diabetics (all $p < 0.001$). Age was the strongest independent predictor (adjusted OR 1.28/year; $p < 0.001$).

Conclusions: Asymptomatic diabetics carried a high burden of subclinical, frequently obstructive CAD; CCTA enabled early detection and risk stratification. Because controls were substantially younger and unmatched, these between-group differences reflect the combined influence of diabetes and age rather than an age-independent effect of diabetes; the findings are hypothesis-generating and require confirmation in age-matched studies.

Key-words: type 2 diabetes mellitus; coronary CT angiography; subclinical coronary artery disease; CAD-RADS; coronary artery calcium score; cross-sectional studies; asymptomatic diseases.



This is an Open Access article distributed under the terms of the [Creative Commons Attribution 4.0 International License](https://creativecommons.org/licenses/by/4.0/).

© 2026 The Author(s). Published by [Scientific Publications](https://www.scientificpublications.in/).

How to Cite

Aditya Singh, Kuldeep Chandel, Shaily Panwar, & Qutubuddin. (2026). Subclinical Coronary Artery Disease in Asymptomatic Type 2 Diabetes Mellitus: A Cross-Sectional Coronary CT Angiography Study from the Bundelkhand Region, India. *International Journal of Medical Sciences and Academic Research*, 7(05), 261-269. <https://doi.org/10.5281/zenodo.23529392>

INTRODUCTION:

Coronary artery disease (CAD) is the leading cause of death among people with type 2 diabetes mellitus (T2DM). Diabetes accelerates atherosclerosis through chronic hyperglycaemia, endothelial dysfunction, oxidative stress, inflammation and dyslipidaemia, and it independently confers a roughly two-fold increase in atherosclerotic cardiovascular risk.¹ because diabetic autonomic neuropathy blunts the perception of ischaemic pain, a substantial proportion of coronary disease in these patients remains clinically silent and is discovered only after myocardial infarction or sudden cardiac death.²

Among asymptomatic patients with T2DM, non-invasive testing consistently uncovers a high burden of occult disease. Exercise-based screening detects silent ischaemia in more than one in five diabetic individuals,¹² and imaging reviews report silent myocardial ischaemia in approximately 6–35% of diabetic cohorts, with obstructive disease in a considerable minority.² Determinants of silent CAD include longer diabetes duration, poor glycaemic control, hypertension, dyslipidaemia and smoking.^{5,10,15}

Coronary CT angiography (CCTA) is a non-invasive, highly sensitive modality that visualises coronary stenosis, characterises plaque and quantifies coronary artery calcium (CAC). Standardised reporting with the Coronary Artery Disease–Reporting and Data System (CAD-RADS 2.0) links stenosis severity, plaque burden and modifiers to clinical management.⁹ Current guidelines favour selective, risk-based screening rather than universal testing of asymptomatic diabetic patients.¹

Regional data on subclinical CAD in asymptomatic diabetics remain limited, particularly for underserved parts of India such as the Bundelkhand region, where diabetes phenotype, diet, genetic susceptibility and healthcare access may differ from Western and East-Asian cohorts.³ We therefore used CCTA to determine the prevalence, plaque characteristics and severity of subclinical CAD in asymptomatic diabetic patients and to compare these with non-diabetic controls, so as to inform early risk stratification and preventive care.

Objectives:

The primary objective was to estimate the prevalence of subclinical coronary artery disease, defined as any coronary plaque (CAD-RADS ≥ 1) on CCTA, in asymptomatic diabetic patients. The secondary objectives were to assess disease severity using the CAD-RADS 2.0 classification, the coronary artery calcium score and the number of vessels involved; to characterise coronary plaque type (calcified, non-calcified or mixed) and plaque burden (P0–P4); to compare the prevalence, severity and plaque characteristics of subclinical

CAD between diabetic and non-diabetic participants; and to identify the clinical and biochemical factors—such as age, glycaemic control, diabetes duration, hypertension and dyslipidaemia—independently associated with obstructive CAD (CAD-RADS ≥ 3) among diabetic patients. Because the two groups were not age- or sex-matched, the diabetic-versus-control comparison was framed as a descriptive, unadjusted comparison and was not intended to isolate an age-independent effect of diabetes.

MATERIALS AND METHODS:

Study design and setting

This cross-sectional, observational study was conducted in the Department of General Medicine of a tertiary-care teaching hospital in the Bundelkhand region over one year (March 2025 to February 2026). Clinical, biochemical and imaging data were collected prospectively using a structured case-record form.

Participants

A total of 172 asymptomatic adults were enrolled: 86 patients with T2DM and 86 non-diabetic individuals (attendants or family members) without cardiac symptoms. Eligible diabetic patients were aged ≥ 40 years with T2DM of ≥ 5 years' duration, and had no history of angina, myocardial infarction or prior coronary revascularisation. Non-diabetic participants were similarly free of cardiac symptoms and prior coronary events. Patients were excluded if they had known symptomatic CAD, prior percutaneous coronary intervention or coronary artery bypass grafting, heart failure, stroke or peripheral vascular disease, an estimated glomerular filtration rate (eGFR) < 30 mL/min/1.73 m², known iodinated-contrast allergy, pregnancy or lactation, or severe comorbidity limiting life expectancy. Written informed consent was obtained from every participant. Participant flow was as follows: individuals attending the participating departments during the study period who met the eligibility criteria and gave consent were enrolled consecutively until the target of 86 participants per group was reached; 172 participants (86 diabetic and 86 non-diabetic) completed clinical and biochemical assessment and coronary CT angiography and were analysed, with no loss to follow-up in this cross-sectional design. Coronary CT angiography was fully reported in all 86 diabetic participants and in 85 of the 86 non-diabetic participants; in one non-diabetic participant the CAD-RADS category, plaque-burden category and calcium score were not recorded, and that participant is shown as “not recorded” in the corresponding distributions. Because this participant's plaque status is unknown, the prevalence of any coronary plaque in the non-diabetic group was reported as an available-case estimate (26/85) rather than being counted as plaque-negative. Isolated missing biochemical values (fasting glucose and HDL in one non-diabetic participant each, and total cholesterol in one

diabetic participant) were handled by available-case analysis. Numbers screened but found ineligible before consent were not separately recorded in a screening register, which is acknowledged under Limitations.

Potential sources of bias were considered when the study was planned. Non-diabetic participants were recruited as a convenience sample of attendants and family members accompanying patients to the hospital, and may therefore not represent the general non-diabetic population of the region; the absence of age and sex matching may exaggerate the apparent between-group differences; and recruitment at a tertiary-care centre may favour participants with a higher cardiovascular risk profile than the community average. These sources of bias were minimised by applying identical symptom-based inclusion and exclusion criteria, uniform case-record documentation, and a single standardised CCTA acquisition and CAD-RADS 2.0 reporting protocol in both groups, and they are acknowledged under Limitations.

Sample size

Assuming an expected prevalence of subclinical CAD of 30% ($p=0.30$), a precision (d) of 7% and a 95% confidence level ($Z=1.96$), the estimated sample size was $n = Z^2pq/d^2 \approx 165$; allowing for 5% non-response gave a final target of 172 participants, distributed equally between the two groups.

Clinical and biochemical assessment

Demographic details, diabetes duration, smoking, alcohol use, physical activity, blood pressure and family history were recorded. Fasting and postprandial blood glucose, glycated haemoglobin (HbA1c), fasting lipid profile (total cholesterol, LDL, HDL, triglycerides), serum creatinine and eGFR were measured using standardised, calibrated laboratory methods.

Coronary CT angiography and CAD-RADS classification

All participants underwent CCTA on a 128-slice multidetector CT scanner using a standard institutional protocol with a non-ionic iodinated contrast agent, after appropriate heart-rate control. Images were analysed with multiplanar reconstruction, curved planar reformation and maximum-intensity-projection techniques by experienced radiologists. Each participant was assigned a CAD-RADS 2.0 category based on the highest-grade luminal stenosis in any major epicardial artery (0, no plaque; 1, 1–24%; 2, 25–49%; 3, 50–69%; 4A, 70–99% in 1–2 vessels; 4B, left main $\geq 50\%$ or three-vessel disease; 5, total occlusion). For analysis, CAD-RADS 0–2 was classed as non-obstructive and CAD-RADS ≥ 3 ($\geq 50\%$ stenosis) as obstructive CAD.⁹ Plaque was characterised as calcified, non-calcified or mixed. The number of significantly diseased vessels ($\geq 50\%$ stenosis) defined single-, double- or triple-vessel disease. CAC was quantified by the Agatston method (0; 1–99, mild; 100–299, moderate;

≥ 300 , high). Plaque burden was summarised by the segment involvement score and its CAD-RADS P-category (P0–P4).

Statistical analysis

Data were analysed in Python (SciPy/statsmodels). Continuous variables are expressed as mean $\pm$ standard deviation and compared using the independent-samples (Welch) t-test; categorical variables are expressed as n (%) and compared using the Pearson chi-square test, with the Fisher exact test where expected cell counts were small. Independent predictors of obstructive CAD among diabetics were assessed by univariable and multivariable binary logistic regression, with results reported as odds ratios (OR) with 95% confidence intervals (CI). A two-sided $p < 0.05$ was considered statistically significant. A continuity (Yates') correction was applied to all 2×2 chi-square comparisons and was not applied to the larger multi-category contingency tables. Collinearity among the candidate predictors was assessed using the Pearson correlation between age and diabetes duration ($r=0.34$, $p=0.001$) and variance inflation factors (age 1.24; HbA1c 1.23; diabetes duration 1.22; LDL 1.18; hypertension 1.14), all well below the conventional threshold of 5, indicating no material multicollinearity; a sensitivity model omitting diabetes duration was also fitted. With 36 obstructive-CAD events and five candidate predictors, the events-per-variable ratio was approximately 7.2, below the conventional threshold of 10; the multivariable model was therefore treated as exploratory and its estimates are interpreted with caution. Participants with a missing value for a given variable were excluded from the analysis of that variable only.

RESULTS:

Baseline characteristics

The two groups differed markedly in age: diabetic patients were considerably older than controls (62.9 ± 12.2 vs 37.4 ± 9.8 years; $t=15.12$, $p < 0.001$), reflecting the ≥ 40 -year, ≥ 5 -year-duration entry criterion for the diabetic arm. Men predominated in both groups (56/86, 65.1% vs 66/86, 76.7%; $\chi^2=2.28$, $p=0.131$). Hypertension (44/86, 51.2% vs 6/86, 7.0%; $\chi^2=38.60$, $p < 0.001$), a family history of CAD (14/86, 16.3% vs 2/86, 2.3%; $\chi^2=8.34$, $p=0.004$) and a family history of diabetes (43/86, 50.0% vs 11/86, 12.8%; $\chi^2=25.94$, $p < 0.001$) were all significantly commoner among diabetics. Current smoking (26/86, 30.2% vs 17/86, 19.8%; $\chi^2=1.98$, $p=0.159$) and alcohol use (14/86, 16.3% vs 11/86, 12.8%; $\chi^2=0.19$, $p=0.665$) did not differ significantly. The mean diabetes duration was 13.9 ± 5.4 years (Table 1).

Biochemical profile

Diabetic patients showed the expected metabolic derangement. HbA1c (8.84 ± 1.28 vs $5.08 \pm 0.36\%$; $t=26.16$, $p < 0.001$), fasting glucose (170.7 ± 41.0 vs 145.4 ± 41.1

mg/dL; $t=4.04$, $p<0.001$) and postprandial glucose (272.8 ± 73.1 vs 229.2 ± 68.7 mg/dL; $t=4.04$, $p<0.001$) were all higher in diabetics. An atherogenic lipid pattern was evident, with higher total cholesterol (223.4 ± 48.0 vs 200.6 ± 33.6 mg/dL; $p<0.001$), LDL (154.0 ± 28.9 vs 134.9 ± 27.2 mg/dL; $p<0.001$) and triglycerides (171.1 ± 58.6 vs 149.1 ± 44.6 mg/dL; $p=0.006$), while HDL was similar (38.1 ± 11.2 vs 39.8 ± 8.5 mg/dL; $p=0.28$). eGFR was lower in diabetics (88.4 ± 19.2 vs 110.1 ± 20.7 mL/min/1.73 m²; $t=-7.10$, $p<0.001$), largely reflecting the age difference between groups (Table 2).

Prevalence and pattern of subclinical CAD

Subclinical CAD was far more prevalent and more severe in diabetics. Any coronary plaque (CAD-RADS ≥ 1) was detected in all diabetic patients (86/86, 100%) compared with 26/85 (30.6%) controls, and obstructive CAD (CAD-RADS ≥ 3 , $\geq 50\%$ stenosis) was present in 36/86 diabetics (41.9%) versus 0/86 controls (0.0%; Fisher $p<0.001$). Calcified plaque was substantially commoner in diabetics (40/86, 46.5% vs 1/86, 1.2%; $\chi^2=46.24$, $p<0.001$), whereas controls with plaque had almost exclusively non-calcified lesions. Multivessel disease (≥ 2 vessels) occurred in 21/86 diabetics (24.4%) and in no controls (Fisher $p<0.001$); single-vessel disease was the commonest pattern among diabetics (51/86, 59.3%) (Table 3).

CAD-RADS severity and plaque burden

The distribution of CAD-RADS categories differed markedly between groups ($\chi^2=119.59$, $p<0.001$). Plaque burden was correspondingly greater in diabetics, with severe-to-extensive categories (P3–P4) in 24/86 (27.9%) versus 1/86 (1.2%) of controls ($\chi^2=36.56$, $p<0.001$) (Table 4). These distributions are shown graphically in Figure 1.

Predictors of obstructive CAD in diabetics

In multivariable logistic regression among the 86 diabetic patients, age was the strongest independent predictor of obstructive CAD (adjusted OR 1.28 per year, 95% CI 1.15–1.42; $p<0.001$). HbA1c showed a positive but non-significant trend after adjustment (adjusted OR 1.66, 95% CI 0.93–2.97; $p=0.090$), while hypertension and LDL were not independent predictors, and diabetes duration was not independently associated with obstructive CAD once age was taken into account (Table 5). The model was highly significant overall (likelihood-ratio $p<0.001$; pseudo- $R^2=0.45$). The adjusted estimate for diabetes duration reflects collinearity with age and is not interpreted as an independent protective effect. Age and diabetes duration were only modestly correlated ($r=0.34$, $p=0.001$) and all variance inflation factors were ≤ 1.24 , so the attenuation of the duration estimate on adjustment reflects adjustment for age rather than multicollinearity. In a sensitivity model omitting diabetes duration, age remained the dominant predictor (adjusted OR 1.26 per year, 95% CI 1.14–1.38; $p<0.001$) and no other variable reached significance.

Table 1: Baseline demographic and clinical characteristics (diabetic vs non-diabetic; n=86 each).

Characteristic	Diabetic n (%)	Non-diabetic n (%)	Test; p
Age (years), mean $\pm$ SD	62.9 $\pm$ 12.2	37.4 $\pm$ 9.8	$t=15.12$; <0.001
Male sex	56 (65.1)	66 (76.7)	$\chi^2=2.28$; 0.131
Hypertension	44 (51.2)	6 (7.0)	$\chi^2=38.60$; <0.001
Family history of CAD	14 (16.3)	2 (2.3)	$\chi^2=8.34$; 0.004
Family history of diabetes	43 (50.0)	11 (12.8)	$\chi^2=25.94$; <0.001
Current smoker	26 (30.2)	17 (19.8)	$\chi^2=1.98$; 0.159
Alcohol use	14 (16.3)	11 (12.8)	$\chi^2=0.19$; 0.665
Diabetes duration (years), mean $\pm$ SD	13.9 $\pm$ 5.4	—	—

SD, standard deviation; CAD, coronary artery disease.

Table 2. Biochemical parameters (mean $\pm$ SD).

Parameter	Diabetic	Non-diabetic	t; p
HbA1c (%)	8.84 $\pm$ 1.28	5.08 $\pm$ 0.36	26.16; <0.001
Fasting glucose (mg/dL)	170.7 $\pm$ 41.0	145.4 $\pm$ 41.1	4.04; <0.001
Postprandial glucose (mg/dL)	272.8 $\pm$ 73.1	229.2 $\pm$ 68.7	4.04; <0.001
Total cholesterol (mg/dL)	223.4 $\pm$ 48.0	200.6 $\pm$ 33.6	3.59; <0.001
LDL (mg/dL)	154.0 $\pm$ 28.9	134.9 $\pm$ 27.2	4.47; <0.001
HDL (mg/dL)	38.1 $\pm$ 11.2	39.8 $\pm$ 8.5	-1.08; 0.28
Triglycerides (mg/dL)	171.1 $\pm$ 58.6	149.1 $\pm$ 44.6	2.77; 0.006
Serum creatinine (mg/dL)	0.86 $\pm$ 0.18	0.79 $\pm$ 0.14	2.54; 0.012
eGFR (mL/min/1.73 m ²)	88.4 $\pm$ 19.2	110.1 $\pm$ 20.7	-7.10; <0.001

Table 3. Coronary CT angiography findings and coronary calcium: diabetic vs non-diabetic (n=86 each).

CCTA finding	Diabetic n (%)	Non-diabetic n (%)	Test; p
Any coronary plaque (CAD-RADS $\geq$ 1)	86 (100)	26/85 (30.6)	Fisher; <0.001
Obstructive CAD (CAD-RADS $\geq$ 3)	36 (41.9)	0 (0.0)	Fisher; <0.001
Calcified plaque	40 (46.5)	1 (1.2)	$\chi^2=46.24$; <0.001
Multivessel disease ($\geq$ 2 vessels)	21 (24.4)	0 (0.0)	Fisher; <0.001
CAC 0	0 (0.0)	14 (16.3)	$\chi^2=47.46$; <0.001†
CAC 1–99	50 (58.1)	69 (80.2)	
CAC 100–299	16 (18.6)	1 (1.2)	
CAC $\geq$ 300	20 (23.3)	1 (1.2)	
CAC not recorded	0 (0.0)	1 (1.2)	

CCTA, coronary CT angiography; CAC, coronary artery calcium score (Agatston units). † The chi-square statistic shown against “CAC 0” is the omnibus test for the overall four-category CAC distribution (0, 1–99, 100–299, $\geq$ 300). Coronary CT angiography was not fully reported in one non-diabetic participant, who is shown as “not recorded”; percentages are of the whole group (n=86). For the primary outcome “any coronary plaque”, the non-diabetic prevalence is reported as an available-case estimate (26/85, 30.6%), excluding the participant whose CCTA was not recorded.

Table 4. CAD-RADS 2.0 severity and plaque-burden (P-category) distribution: diabetic vs non-diabetic (n=86 each).

Category	Diabetic n (%)	Non-diabetic n (%)	Test; p
CAD-RADS 0	0 (0.0)	59 (68.6)	$\chi^2=119.59$; <0.001
CAD-RADS 1	8 (9.3)	17 (19.8)	
CAD-RADS 2	42 (48.8)	9 (10.5)	
CAD-RADS 3	27 (31.4)	0 (0.0)	
CAD-RADS 4A	4 (4.7)	0 (0.0)	
CAD-RADS 4B	5 (5.8)	0 (0.0)	
CAD-RADS not recorded	0 (0.0)	1 (1.2)	
Plaque burden P0	0 (0.0)	14 (16.3)	$\chi^2=36.56$; <0.001
P1 (mild)	19 (22.1)	27 (31.4)	
P2 (moderate)	43 (50.0)	43 (50.0)	
P3 (severe)	22 (25.6)	1 (1.2)	
P4 (extensive)	2 (2.3)	0 (0.0)	
Plaque burden not recorded	0 (0.0)	1 (1.2)	

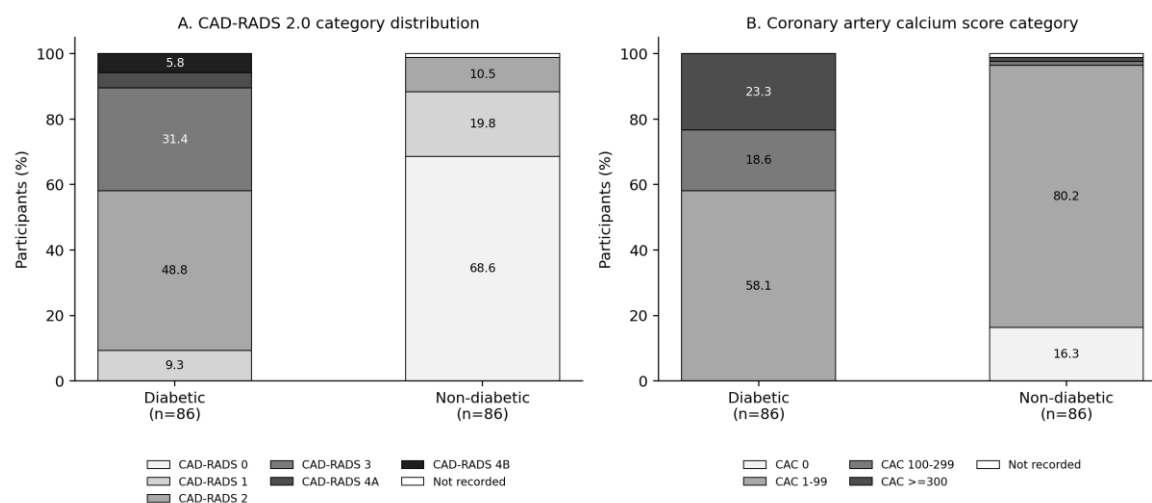


Figure 1. Distribution of CAD-RADS 2.0 categories (left) and coronary artery calcium score categories (right) in diabetic and non-diabetic participants (n=86 each).

Table 5. Univariable and multivariable logistic regression for obstructive CAD in diabetic patients (n=86).

Predictor	Univariable OR (95% CI)	Adjusted OR (95% CI)	Adj. p
Age (per year)	1.22 (1.12–1.33)	1.28 (1.15–1.42)	<0.001
HbA1c (per %)	1.20 (0.85–1.69)	1.66 (0.93–2.97)	0.090
Diabetes duration (per year)	1.03 (0.95–1.12)	0.87 (0.76–1.00)	0.043
LDL (per mg/dL)	0.99 (0.98–1.01)	0.98 (0.95–1.01)	0.146
Hypertension (yes)	1.57 (0.64–3.89)	0.59 (0.15–2.29)	0.447

OR, odds ratio; CI, confidence interval. Obstructive CAD defined as CAD-RADS ≥ 3 . Variance inflation factors for all predictors were ≤ 1.24 and the age–duration correlation was $r=0.34$, indicating no material multicollinearity; the adjusted estimate for diabetes duration reflects adjustment for age and is not interpreted as an independent protective effect.

DISCUSSION:

In this cross-sectional CCTA study of asymptomatic adults from the Bundelkhand region, subclinical CAD was highly prevalent among patients with T2DM: obstructive CAD (CAD-RADS ≥ 3) was present in 41.9% of diabetics and in none of the non-diabetic controls, and every diabetic patient had at least minimal coronary plaque. These findings indicate that, in a hitherto under-studied Indian population, clinically silent yet frequently obstructive coronary atherosclerosis is common in asymptomatic diabetes. Because the non-diabetic comparison group was substantially younger than the diabetic group (mean 37.4 vs 62.9 years) and was not age- or sex-matched, the between-group contrasts described below are unadjusted and reflect the combined influence of diabetes and age; they should not be read as isolating an age-independent effect of diabetes.

The prevalence we observed is consistent with, though at the higher end of, the published range. Yoo et al. reported obstructive stenosis in 13.3% of asymptomatic type 2 diabetics versus 8.5% of non-diabetics in a propensity-matched CCTA cohort, with a higher incidence of adverse cardiac events in the diabetic group.⁴ Takamura et al. found obstructive CAD in 26.7% of asymptomatic diabetics and high-risk plaque in 16.8%.¹⁰ A narrative review by Santilli et al. described silent myocardial ischaemia in 6–35% of diabetic individuals and obstructive disease in a substantial subset with positive ischaemic testing.² The comparatively high obstructive rate in our cohort is most plausibly explained by its older age (mean 62.9 years) and long diabetes duration (mean 13.9 years), both of which are established accelerants of coronary atherosclerosis.

Diabetic patients also showed a heavier and more calcified plaque burden. Calcified plaque was far commoner in

diabetics (46.5% vs 1.2%), and no diabetic patient had a zero calcium score, whereas 16.3% of controls did; moderate-to-high CAC (≥ 100) was present in 41.9% of diabetics. Jiang et al. similarly found that T2DM was independently associated with calcified plaque, obstructive CAD and multivessel disease on CCTA in hypertensive individuals.⁵ Heinsen et al. reported that CAC score and clinical risk factors identify high-risk plaque in asymptomatic type 2 diabetics,¹¹ and the systematic review by Dun et al. confirmed that asymptomatic CAD affects more than one in five diabetic patients.¹² A systematic review by Guaricci et al. likewise concluded that CCTA reliably detects a high prevalence of subclinical atherosclerosis in asymptomatic diabetics,¹⁷ and Halon et al. showed in a 7-year cohort that total plaque burden independently predicts coronary events in this population.¹⁸ The predominance of calcification in our older, long-duration cohort is in keeping with calcification being a marker of advanced, chronic atherosclerosis.

Multivessel disease was confined almost entirely to diabetics (24.4% vs 0%), and CAD-RADS severity was shifted strongly toward higher categories, mirroring the diffuse, multivessel pattern characteristic of diabetic coronary disease. Standardised CAD-RADS 2.0 reporting, combining stenosis grade with plaque-burden modifiers, provided a reproducible framework for this comparison and for downstream risk-based management.⁹

Among diabetics, age emerged as the dominant independent predictor of obstructive CAD (adjusted OR 1.28 per year), with a positive but non-significant trend for HbA1c after adjustment. The attenuation of the glycaemic and duration effects in the multivariable model likely reflects the relatively uniform, long-standing hyperglycaemia of the cohort. These observations are congruent with prognostic CCTA studies in asymptomatic diabetes: Hyun et al. showed that CCTA-defined obstructive CAD added incremental 10-year prognostic value beyond clinical risk scores,⁶ and Lee et al. demonstrated that longer diabetes duration is linked to worse outcomes even in non-obstructive disease.¹³ Valensi et al. found that a CAC-guided strategy efficiently selects diabetic patients for further ischaemia testing,⁸ supporting the role of calcium scoring in triage.

Clinically, these data support further evaluation of a selective, risk-based approach to screening rather than universal testing. Current ESC guidance recommends estimating cardiovascular risk in patients with diabetes using validated tools such as SCORE2-Diabetes and reserving anatomical or functional imaging for those at higher risk,^{1,7,14} and CAD-RADS-based CCTA offers a practical, non-invasive means of doing so while also guiding intensification of glycaemic, lipid-lowering and antihypertensive therapy. Early identification matters

because diabetic patients with established coronary disease face a high burden of major adverse cardiovascular events.¹⁶ In resource-limited regions such as Bundelkhand, targeting CCTA to older diabetics with long disease duration and elevated calcium scores may warrant evaluation as the strategy most likely to yield diagnostic and preventive value. Region-specific data such as ours from North India align with prior Indian work showing a high burden of silent CAD in T2DM.³ These observations are hypothesis-generating: as a single-centre, unmatched cross-sectional study without outcome follow-up, the present data cannot by themselves justify a screening-policy recommendation, and prospective, age-matched studies with clinical endpoints are needed before such a recommendation could be made.

CONCLUSION:

Asymptomatic patients with type-2 diabetes in the Bundelkhand region carried a high burden of subclinical coronary artery disease, with obstructive CAD in 41.9%, greater plaque and calcium burden and more multivessel involvement than non-diabetic controls. Age was the strongest independent predictor of obstructive disease. Coronary CT angiography with CAD-RADS 2.0 and calcium scoring appears to be a useful non-invasive tool for early detection and risk stratification. Because the comparison group was younger and unmatched, these hypothesis-generating findings may warrant further evaluation of a selective, risk-based screening strategy and of intensified preventive therapy in high-risk diabetic individuals, rather than supporting a screening recommendation at this stage.

DECLARATIONS:

Ethical Approval: The study was approved by the Institutional Ethics Committee (Ref. 719/IEC/PG-23-14/2026/SC-1, dated 02/05/2025), and was conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all participants.

Informed Consent: Informed consent was obtained from all participants prior to their inclusion in the study. Participants were informed that their data would be kept confidential and used solely for research purposes.

Funding Information: This research did not receive any specific grant from funding agencies in the public, commercial or not-for-profit sectors. The study was entirely self-funded by the authors.

Conflict of Interest: The authors declare that they have no conflicts of interest related to this study.

Data availability statement: The dataset presented in this study is available on request from the corresponding author.

AI Use Disclosure: No generative AI tools were used in the writing or data analysis. AI tools were limited to language assistance. The authors remain fully responsible for the content.

Acknowledgement: The authors gratefully acknowledge the technical, nursing and paramedical staff of the Departments of General Medicine and Radio-diagnosis for their assistance with patient recruitment, data collection and coronary CT angiography acquisition. We also thank the laboratory staff for the biochemical analyses and, above all, all the participants for their kind cooperation and consent. No external funding agency was involved in the conduct of this study.

REFERENCES:

- Marx N, Federici M, Schütt K, Müller-Wieland D, Ajjan RA, Antunes MJ, et al. 2023 ESC Guidelines for the management of cardiovascular disease in patients with diabetes. *Eur Heart J*. 2023;44(39):4043–4140. <https://doi.org/10.1093/eurheartj/ehad192>
- Santilli F, Blaha MJ, Ricci F, Simeone P. Hunting for a coronary artery disease diagnosis in asymptomatic patients with diabetes mellitus: if, how and when. *Cardiovasc Diabetol*. 2025;24:418. <https://doi.org/10.1186/s12933-025-02966-4>
- Kumar V, et al. Prevalence of coronary artery disease in asymptomatic type 2 diabetes mellitus patients with invasive correlation in North India. *Indian J Endocrinol Metab*. 2023;27(2):133–139. https://doi.org/10.4103/ijem.ijem_378_22
- Yoo JY, Kang SR, Bae YJ, Chun EJ. Comparison of cardiac events and survival between asymptomatic diabetic and nondiabetic patients: coronary CT study. *J Korean Med Sci*. 2025;40(40):e251. <https://doi.org/10.3346/jkms.2025.40.e251>
- Jiang Y, Yang Z, Wang J, Jiang L, Han P, Shi R, et al. Type 2 diabetes mellitus aggravates coronary atherosclerosis in hypertensive individuals based on coronary CT angiography: a retrospective propensity score-based study. *Front Cardiovasc Med*. 2024;11:1372519. <https://doi.org/10.3389/fcvm.2024.1372519>
- Hyun J, Lee PH, Lee J, Yang Y, Kim JH, Kim TO, et al. Ten-year prognostic value of coronary CT angiography in asymptomatic patients with type 2 diabetes. *Rev Esp Cardiol (Engl Ed)*. 2023;76(9):700–707. <https://doi.org/10.1016/j.rec.2023.01.004>
- Saraste A, Knuuti J, Bax J. Screening for coronary artery disease in patients with diabetes. *Curr Cardiol Rep*. 2023;25(12):1865–1871. <https://doi.org/10.1007/s11886-023-01999-z>
- Valensi P, Berkane N, Pinto S, Sellier N, Soussan M, Nguyen MT, et al. Performance of the 2019 ESC/EASD guideline strategy for the screening of silent coronary artery disease in patients with diabetes. *Cardiovasc Diabetol*. 2023;22:2. <https://doi.org/10.1186/s12933-023-01760-4>
- Cury RC, Leipsic J, Abbata S, Achenbach S, Berman D, Bittencourt M, et al. CAD-RADS™ 2.0 – 2022 Coronary Artery Disease-Reporting and Data System. *JACC Cardiovasc Imaging*. 2022;15(11):1974–2001. <https://doi.org/10.1016/j.jcmg.2022.07.002>
- Takamura K, Fujimoto S, Mita T, Kawaguchi YO, Kurita M, Kadowaki S, et al. Identification of risk factors for coronary artery disease in asymptomatic patients with type 2 diabetes mellitus. *J Clin Med*. 2022;11(5):1226. <https://doi.org/10.3390/jcm11051226>
- Heinsen LJ, Pararajasingam G, Andersen TR, Auscher S, Sheta HM, Precht H, et al. High-risk coronary artery plaque in asymptomatic patients with type 2 diabetes: clinical risk factors and coronary artery calcium score. *Cardiovasc Diabetol*. 2021;20:164. <https://doi.org/10.1186/s12933-021-01350-2>
- Dun Y, Wu S, Cui N, Thomas RJ, Olson TP, Zhou N, et al. Screening for asymptomatic coronary artery disease via exercise stress testing in patients with type 2 diabetes mellitus: a systematic review and meta-analysis. *Front Cardiovasc Med*. 2021;8:770648. <https://doi.org/10.3389/fcvm.2021.770648>
- Lee YA, Song SW, Kim SH, Jung J, Jang WY, Moon D, et al. Impact of diabetes duration on major adverse cardiac events in patients with non-obstructive coronary artery disease. *J Clin Med*. 2025;14(8):2797. <https://doi.org/10.3390/jcm14082797>
- SCORE2-Diabetes Working Group and the ESC Cardiovascular Risk Collaboration. SCORE2-Diabetes: 10-year cardiovascular risk estimation in type 2 diabetes in Europe. *Eur Heart J*. 2023;44(28):2544–2556. <https://doi.org/10.1093/eurheartj/ehad260>
- Klimontov VV, Koroleva EA, Khapaev RS, Korbut AI, Lykov AP. Carotid artery disease in subjects with type 2 diabetes: risk factors and biomarkers. *J Clin Med*. 2022;11(1):72. <https://doi.org/10.3390/jcm11010072>
- González-Juanatey C, et al. Major adverse cardiovascular events in coronary type 2 diabetic patients: identification of associated factors using electronic health records and natural language

- processing. J Clin Med. 2022;11(20):6004. <https://doi.org/10.3390/jcm11206004>
17. Guaricci AI, De Santis D, Carbone M, Muscogiuri G, Guglielmo M, Baggiano A, et al. Coronary atherosclerosis assessment by coronary CT angiography in asymptomatic diabetic population: a critical systematic review of the literature and future perspectives. Biomed Res Int. 2018;2018:8927281. <https://doi.org/10.1155/2018/8927281>
18. Halon DA, Azencot M, Rubinshtein R, Zafrir B, Flugelman MY, Lewis BS. Coronary computed tomography angiography as a predictor of cardiac and noncardiac vascular events in asymptomatic type 2 diabetics: a 7-year population-based cohort study. J Am Heart Assoc. 2016;5(6):e003226. <https://doi.org/10.1161/JAHA.116.003226>