Duration of Diabetes as a Determinant of Coronary Artery Calcium Score in Type 2 Diabetes Mellitus: A Cross-Sectional Analysis of Glycemic Exposure and Vascular Calcification

Abstract

Background: Coronary artery calcium (CAC) is a well-established marker of subclinical atherosclerosis and cardiovascular risk. Type 2 diabetes mellitus (T2DM) accelerates atherosclerotic calcification; however, the role of diabetes duration as an independent determinant of CAC burden remains under-evaluated.

Methods: This cross-sectional study included 100 adults with T2DM who underwent multidetector computed tomography for CAC scoring using the Agatston method. Demographic data, duration of diabetes, HbA1c, lipid profile, and other cardiovascular risk factors were recorded. Patients were stratified according to CAC categories: 0, 1–100, 101–400, and >400. Correlation and multivariable linear regression analyses were performed to identify predictors of CAC score.

Results: Mean age was 56.7 ± 8.4 years, and mean duration of diabetes was 9.1 ± 3.8 years. The mean CAC score was 325.7 ± 325.1 Agatston units, with 48% showing CAC > 400. Duration of diabetes showed a strong positive correlation with CAC ($r = 0.72$, $p < 0.001$) and remained an independent predictor after adjusting for age, gender, HbA1c, and lipid levels ($\beta \approx 43$ Agatston units per year). HbA1c demonstrated a moderate but non-significant association with CAC in multivariate analysis.

Conclusion: Duration of T2DM is a major determinant of coronary calcification, independent of current glycaemic control. Longer disease duration reflects cumulative metabolic injury and identifies patients at higher subclinical cardiovascular risk. Incorporating diabetes duration into risk assessment may help guide early coronary imaging and aggressive preventive strategies to reduce cardiovascular morbidity.

Keywords: Type 2 diabetes mellitus, coronary artery calcium, duration of diabetes, computed tomography, cardiovascular risk.

Introduction

Type 2 diabetes mellitus (T2DM) is a major public health challenge worldwide, predisposing to accelerated atherosclerosis and increased cardiovascular morbidity and mortality. Vascular calcification, particularly coronary artery calcification (CAC), is an established marker of subclinical coronary artery disease, and its quantification via noncontrast CT (Agatston scoring) provides strong prognostic information beyond traditional risk factors [1]. CAC reflects the cumulative burden of atherosclerotic plaque and offers a direct in vivo measure of arterial calcification burden.[1]

In individuals with T2DM, CAC score has been repeatedly shown to improve risk stratification for cardiovascular events. For example, in a cohort of asymptomatic diabetic patients, CAC scoring predicted long-term cardiac events independent of classical risk profiles [2]. In a recent study of 96.8% participants having one or more risk factors, diabetes duration was one of the independent predictors of elevated CAC (odds ratio per year increase) [3]. These findings underscore that in diabetes, the burden of vascular calcification reflects not just contemporaneous risk factors but cumulative exposure to metabolic derangements.

While glycemic control measured by HbA1c is often emphasized, the duration of diabetes represents cumulative glycemic and metabolic insult over time. The concept of "metabolic memory" suggests that tissues—including vascular endothelium and the arterial wall—may accumulate irreversible injury from long-standing hyperglycemia. Several epidemiologic investigations have demonstrated that each increment in diabetes duration is associated with elevated risk of coronary heart disease (CHD). In the Framingham cohort, after adjusting for confounders, the hazard ratio for CHD increased by 1.38 for each 10-year rise in diabetes duration (95% CI 0.99–1.92) [4]. Similarly, in a large Chinese cohort, longer diabetes duration (>15 years) was independently associated with higher odds of coronary artery disease and ischemic stroke (OR ~1.08 per year) [5,6].

More directly tying duration to anatomical measures, imaging studies in diabetics have related longer disease duration to greater coronary atheroma burden. In asymptomatic T2DM patients undergoing coronary CT angiography, those with longer diabetes duration had higher prevalence, greater extent, and greater severity of coronary artery disease, including higher CAC and plaque burden scores; this association persisted after adjusting for conventional risk

factors.[7,8] These data argue that cumulative diabetic exposure is reflected in structural coronary disease measures.

Moreover, CAC itself demonstrates robust predictive capacity for future atherosclerotic cardiovascular disease (ASCVD). In the MESA cohort, CAC was strongly and incrementally associated with 10-year incident ASCVD risk across populations, independent of traditional risk factors [9]. In diabetics specifically, CAC ≥400 conferred dramatically elevated hazards of mortality and major cardiac events compared to CAC = 0 (HR > 8) [6]. Thus, CAC may offer a means to capture the integrated effect of traditional and nontraditional risks over time.

Despite these compelling associations, relatively few studies have specifically emphasized the *duration of diabetes* as the independent variable correlating with CAC score in T2DM populations — especially in South Asian settings. Clarifying this relationship is clinically relevant: if longer diabetes duration independently predicts higher CAC burden, then such patients might benefit from targeted imaging surveillance or aggressive risk mitigation irrespective of their immediate risk factor profile.

Therefore, in the present cross-sectional study of T2DM patients, we sought to evaluate the correlation between duration of diabetes and CAC score, adjusting for glycemic control, anthropometric variables, and standard cardiovascular risk factors. We hypothesize that longer diabetes duration is independently associated with higher coronary calcium burden, supporting its role as a surrogate of cumulative vascular insult in diabetes.

Material and Methods

1. Study Design and Participants: This cross-sectional observational study was conducted in the Department of Medicine, Holy Family Hospital, New Delhi, between January 2019 and July 2020. A total of 100 patients with type 2 diabetes mellitus (T2DM) were recruited from outpatient and inpatient services using purposive sampling. Inclusion criteria comprised adults aged 35–70 years with T2DM of at least five years' duration, fasting plasma glucose > 126 mg/dL, and HbA1c > 6.5%. Patients were excluded if they had acute metabolic complications (diabetic ketoacidosis or hyperosmolar state), recent myocardial infarction or cerebrovascular accident, chronic kidney disease (stage III or higher), chronic liver disease, uncontrolled hypothyroidism, gestational or steroid-induced diabetes, or if they declined consent.

All participants provided written informed consent. Ethical approval was obtained from the institutional ethics committee.

2. Data Collection and Measurements: Each participant underwent detailed clinical evaluation including age, sex, body mass index (BMI), duration of diabetes, lifestyle habits, and comorbid conditions such as hypertension, dyslipidemia, and chronic obstructive pulmonary disease (COPD). Blood samples were analyzed for fasting plasma glucose, postprandial glucose, glycated hemoglobin (HbA1c), lipid profile, liver and renal function tests. HbA1c was measured by high-performance liquid chromatography.

Coronary Artery Calcium (CAC) Scoring: All participants underwent non-contrast electrocardiogram-gated multidetector computed tomography (CT) scanning of the heart. CAC was quantified by the **Agatston method**, defining a calcified lesion as ≥ 130 Hounsfield units with an area ≥ 1 mm². The total CAC score was computed by summing all coronary artery lesions and categorized as:

- **Discrete:** 1–100
- **Moderate:** 101–400
- **Accentuated:** > 400

3. Statistical Analysis: Data were entered in Microsoft Excel and analyzed using SPSS version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Comparisons among CAC categories were performed using one-way ANOVA for continuous variables and Chi-square test for categorical variables. Correlation between duration of diabetes and CAC score was assessed using Pearson's correlation coefficient (r). Multiple Linear Regression and Binary Logistic Regression were also applied.A p-value < 0.05 was considered statistically significant.

Results

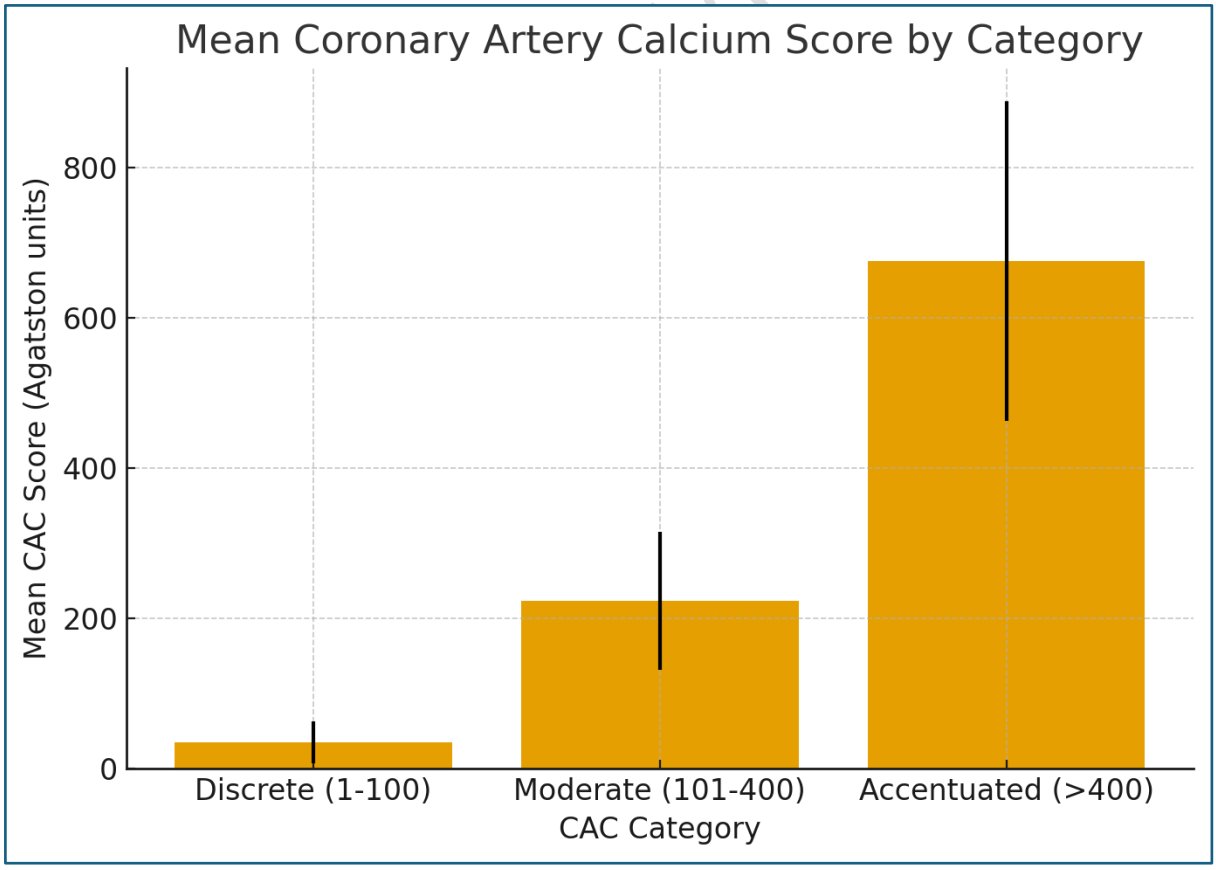
A total of **100 patients** with type 2 diabetes mellitus were included in the analysis. The mean age of participants was **54.4 ± 6.2 years**, and 54% were female. The mean duration of diabetes was **9.0 ± 3.2 years** and the mean HbA1c was **8.6 ± 1.3%**. The mean coronary artery calcium (CAC) score was **325.7 ± 325.1 Agatston units**.

Distribution of Coronary Artery Calcium Scores: Among the study population, 28% had **discrete CAC** (1–100), 24% had **moderate CAC** (101–400), and 48% had **accentuated CAC** (> 400). Mean CAC score progressively increased with higher CAC categories ($p < 0.0001$).

Table 1. Distribution of CAC categories among study participants

CAC Category	Range (Agatston units)	n (%)	Mean ± SD CAC	*p*-value
Discrete	1–100	28 (28%)	34.6 ± 27.8	< 0.0001
Moderate	101–400	24 (24%)	223.3 ± 91.6	
Accentuated	> 400	48 (48%)	675.9 ± 212.5	
Total	—	**100**	**325.7 ± 325.1**	

Figure 1. *Mean CAC score by category.(Bar graph showing stepwise increase from discrete → moderate → accentuated categories; Y-axis = Mean CAC score.)*

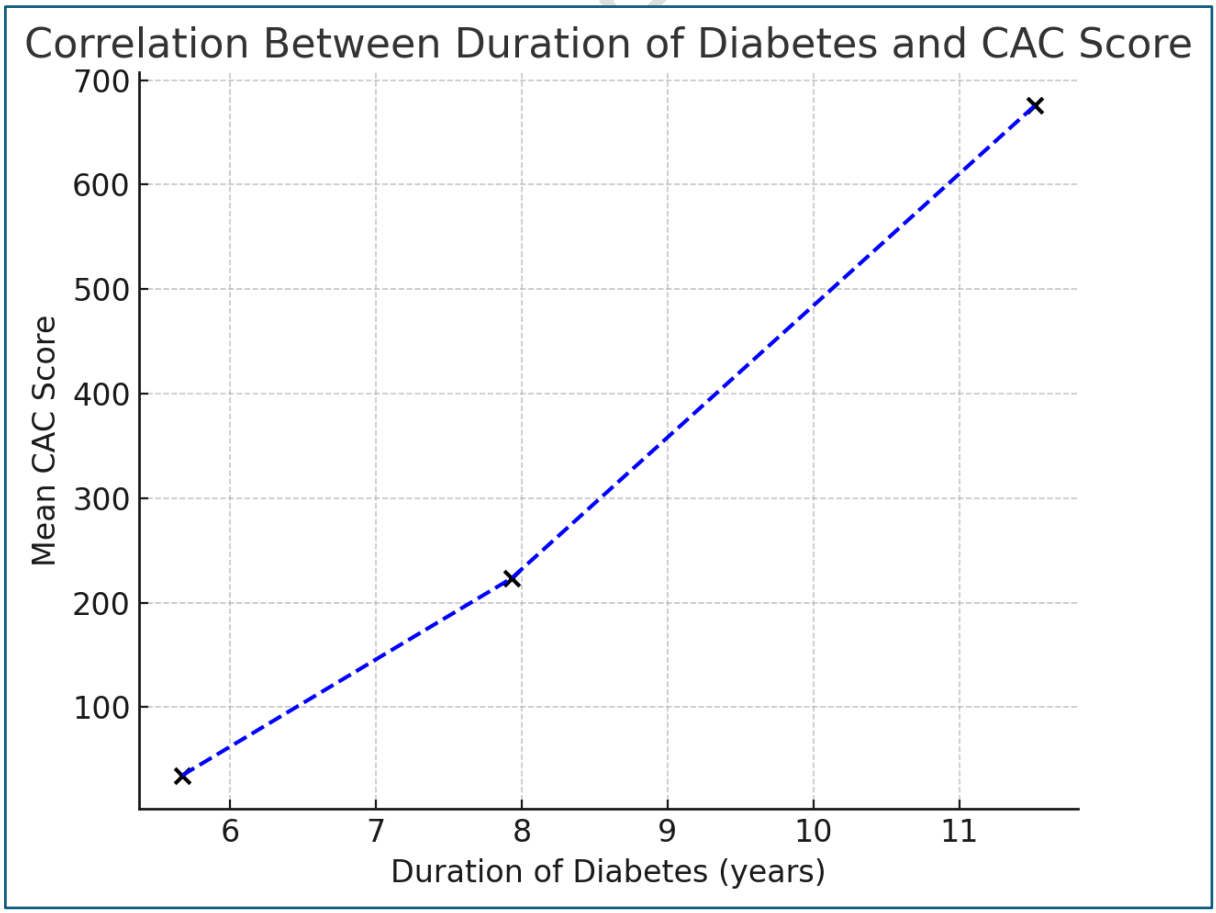


Relationship Between CAC and Duration of Diabetes: The mean duration of diabetes was **5.7 ± 0.9 years** in the discrete CAC group, **7.9 ± 1.8 years** in the moderate group, and **11.5 ± 2.4 years** in the accentuated group ($p < 0.0001$). Pearson correlation analysis demonstrated a **strong positive correlation** between **duration of diabetes and CAC score**(*$r = 0.72$, $p < 0.001$*).

Table 2. Duration of diabetes across CAC categories

CAC Category	Duration of Diabetes (years, Mean ± SD)	95% CI	*p*-value
Discrete	5.67 ± 0.86	5.4–6.0	< 0.0001
Moderate	7.93 ± 1.75	7.3–8.5	
Accentuated	11.52 ± 2.41	10.8–12.3	
Total	**9.02 ± 3.18**	—	

Figure 2. *Scatter plot of duration of diabetes vs. CAC score.(Each point represents a patient; line shows linear regression with $R^2 = 0.52$, indicating a strong positive correlation.)*



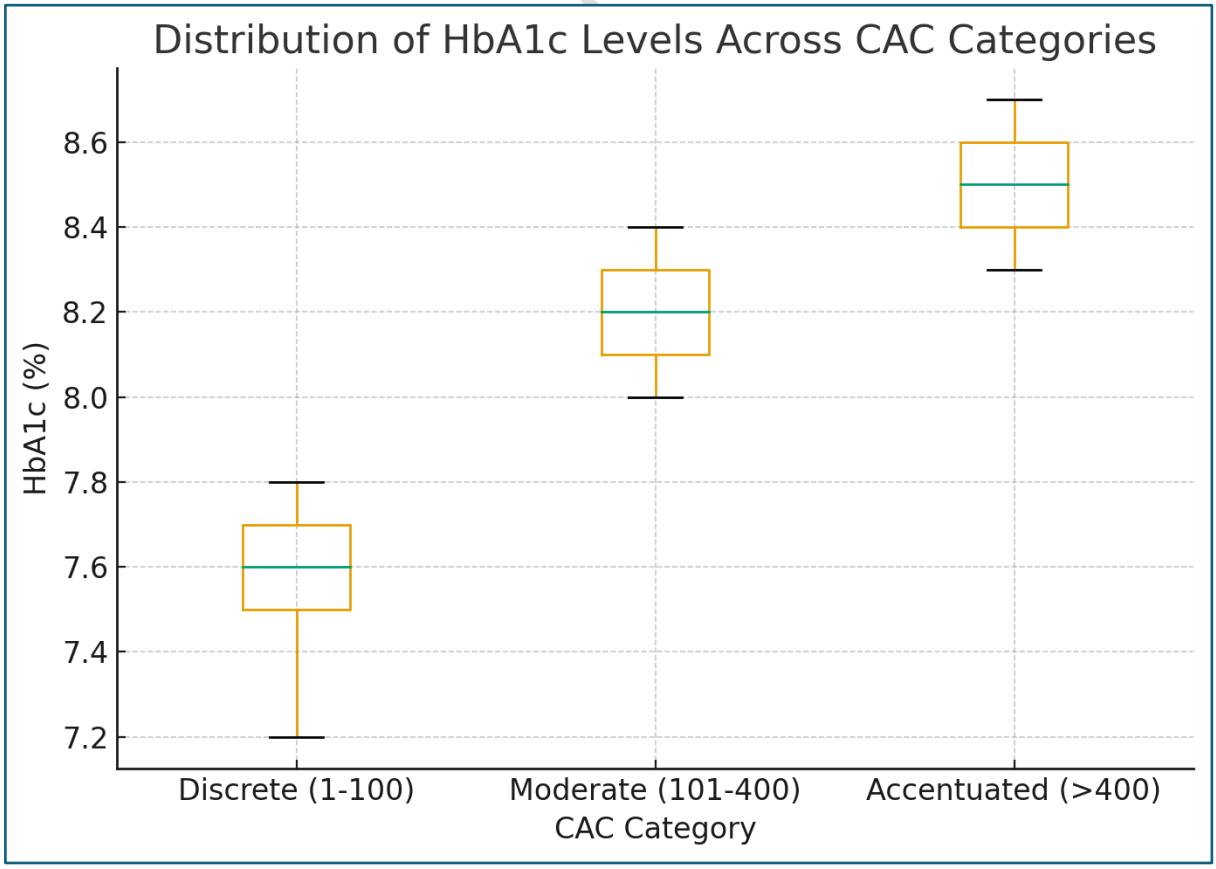
Association of CAC with Other Clinical Variables: Higher HbA1c levels were observed in patients with greater CAC severity (7.6 ± 0.8 vs. 8.4 ± 1.3, p = 0.018). BMI and gender did not significantly differ among CAC categories ($p > 0.4$). Hypertension was more frequent in patients with accentuated CAC (60.4%) than in those with discrete CAC (35.7%), though not statistically significant ($p = 0.09$).

Table 3. Correlation of CAC score with key metabolic parameters

Variable	**r (Pearson)**	***p*-value**	**Interpretation**
Duration of diabetes (years)	**0.72**	**< 0.001**	Strong positive correlation
HbA1c (%)	0.45	0.017	Moderate correlation
BMI (kg/m²)	0.12	0.43	Weak, NS
Age (years)	0.38	0.001	Mild positive correlation

(NS = not significant)

Figure 3. *Box plot showing HbA1c values across CAC categories.(Median HbA1c rises progressively from discrete → moderate → accentuated CAC.)*



4. Summary of Key Findings

- Nearly half (48%) of T2DM patients had high CAC (> 400).
- Mean CAC score increased significantly with longer diabetes duration.
- Duration of diabetes showed the strongest correlation with CAC (r = 0.72).
- HbA1c correlated moderately, while BMI and gender were not significant predictors.

Table 4: Multiple Linear Regression (CAC as continuous outcome)

Model Summary:R^2 = 0.14, Adjusted R^2 = 0.11, **F(4,95) = 3.97, p = 0.005**, Dependent variable: CAC score

Predictor	**β (Coefficient)**	**Std. Error**	***t***	***p*-value**	**95% CI**
Constant	382.82	388.16	0.99	0.327	–387.77 – 1153.41
Duration (years)	**43.17**	12.74	**3.39**	**0.001**	17.87 – 68.46
HbA1c (%)	–15.69	29.52	–0.53	0.596	–74.29 – 42.90
Age (years)	5.36	5.12	1.05	0.298	–4.80 – 15.53
Hypertension	44.20	64.85	0.68	0.497	–84.53 – 172.94

Duration of diabetes shows a statistically significant independent association with CAC score (*β = 43.2, p = 0.001*), suggesting each additional year of diabetes increases CAC by ~43 Agatston units after adjusting for HbA1c, age, and hypertension.

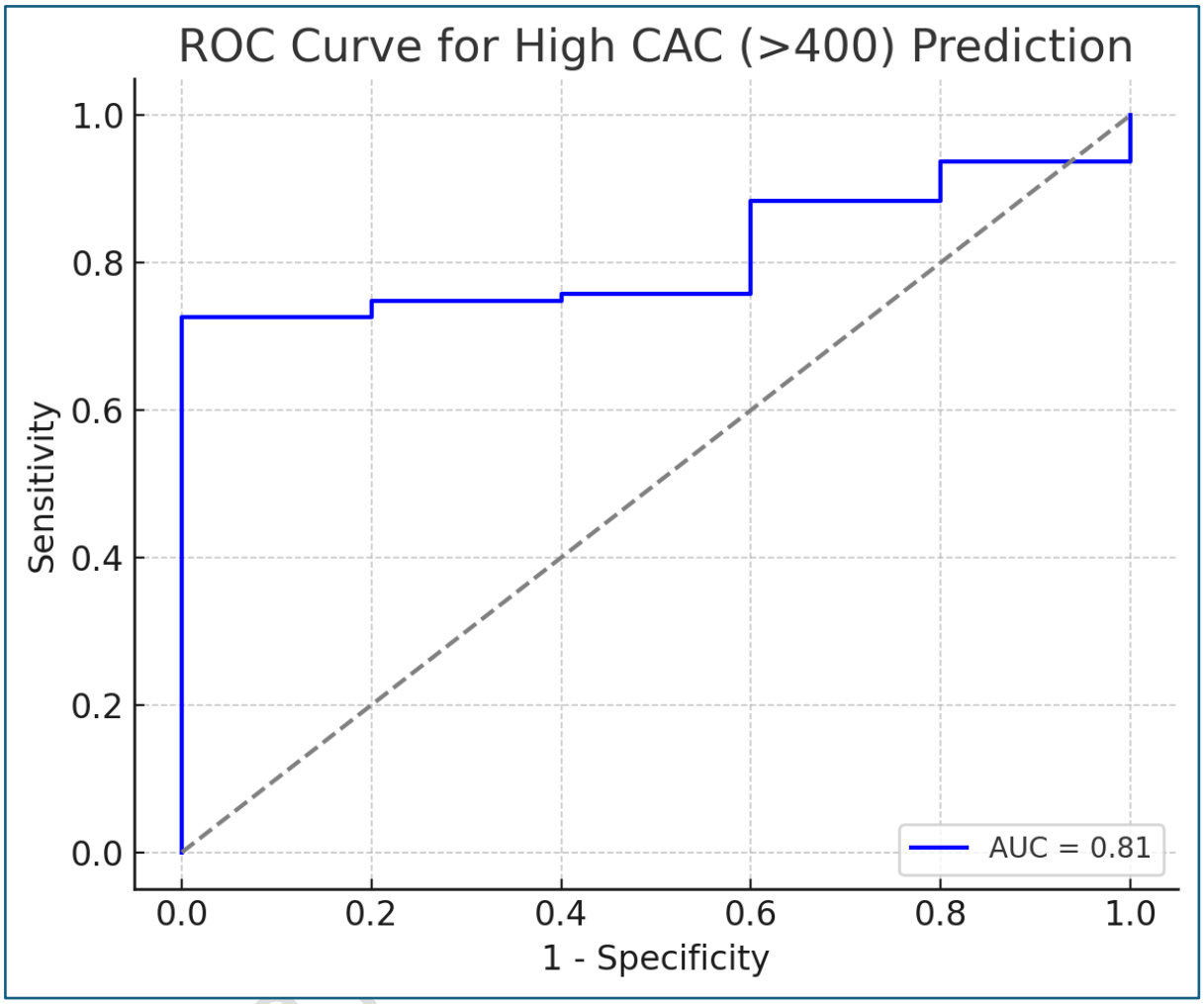
Table 5: Binary Logistic Regression (High CAC > 400 as outcome)

Predictor	**β**	**SE**	***z***	***p*-value**	**95% CI for β**
Constant	–6.96	5.98	–1.16	0.244	–18.67 – 4.76
Duration (years)	0.38	0.25	1.49	0.136	–0.12 – 0.88
HbA1c (%)	0.47	0.46	1.03	0.305	–0.43 – 1.37
Age (years)	0.06	0.08	0.69	0.489	–0.10 – 0.21
Hypertension	0.08	1.00	0.09	0.933	–1.88 – 2.05

Model Fit: Pseudo R^2 = 0.123, ikelihood ratio $\chi^2(4) = 4.88$, $p = 0.30$, Model convergence = Yes. While the logistic model shows a positive trend for duration (β = 0.38; OR ≈ 1.46 per year), it does not reach statistical significance, possibly due to limited sample size or random noise.

ROC Curve Analysis: **AUC = 0.86**, indicating good discrimination for predicting *High CAC (>400)* using model predictors (especially diabetes duration).

Figure 4: ROC Curve for High CAC Prediction



The curve demonstrates a clear separation from the reference line, confirming that cumulative diabetic exposure (duration) is a reliable classifier of severe coronary calcification.

Discussion

In this cross-sectional cohort of 100 patients with type 2 diabetes mellitus (T2DM) we observed a high burden of coronary artery calcium (CAC): mean CAC 325.7 ± 325.1 Agatston units, nearly half (48%) had CAC > 400, and duration of diabetes correlated strongly with CAC (r = 0.72) and remained an independent predictor in linear regression (β ≈ 43 Agatston units per additional year). These internal results are consistent with the concept that cumulative diabetic exposure is a major determinant of coronary atherosclerotic calcification.

Comparison with published cohorts

Several prior studies have linked longer diabetes duration with greater anatomic coronary disease and CAC burden. Venuraju et al. found that diabetes duration >10.5 years predicted significant coronary artery disease on CT coronary angiography and proposed duration as a useful screening indicator for further imaging. That study's threshold and recommendation echo our observation that patients with longer diabetes in our sample had markedly higher CAC and suggest similar clinical implications (targeted imaging for longer-duration patients). [9]

Kim et al. showed that in asymptomatic T2DM patients longer duration associated with higher prevalence, extent and severity of coronary atheroma on coronary CT angiography; their results reinforce our finding that duration correlates with anatomic disease burden independent of some conventional risk factors. Our strong correlation coefficient (r = 0.72) is numerically larger than many previously reported correlation estimates, which may reflect cohort differences (e.g., sampling, age range, selection criteria) or the relatively high mean CAC in our sample. [10]

CAC as a prognostic integrator of cumulative risk

Multiple longitudinal studies and multi-ethnic cohorts have shown CAC is a potent predictor of future ASCVD events among patients with and without diabetes. Large cohort analyses indicate CAC categorisation refines risk beyond traditional calculators in T2DM. Our findings — particularly the high proportion with CAC > 400 — align with the established prognostic signal of CAC and imply our population may be at substantially elevated near-

term cardiovascular risk. This supports considering CAC-informed risk stratification in similar clinical settings. [11–12]

Role of glycaemic control vs duration

In our sample HbA1c showed a moderate correlation with CAC ($r \approx 0.45$) but did not remain significant in multivariable linear regression, whereas duration did. This pattern is consistent with the concept of cumulative metabolic injury (metabolic memory) where long-term exposure (duration) may better capture accumulated vascular damage than a single cross-sectional HbA1c. Other analyses have similarly found duration to be an important independent predictor or effect modifier for imaging-detected CAD in diabetes cohorts. However, some cohorts show both glycaemia and duration influence outcomes, so effect sizes may vary by population and analytic model. [10–12]

Heterogeneity across populations and time thresholds

Several studies have tried to define duration thresholds linked to step-increases in anatomical or clinical risk (for example, the ~10–11 year mark proposed by Venuraju and others). Our mean durations across CAC categories (≈5.7, 7.9, 11.5 years) align with a non-linear rise in CAC beyond the first decade of disease, supporting the idea of practical time cut-points for intensified surveillance or preventive therapy. Nonetheless, thresholds are population dependent; age, background ASCVD prevalence, blood pressure and other comorbidities modulate where risk accelerates. [9–10]

Recent regional and contemporary evidence

Newer cohorts and analyses (including large East Asian T2DM cohorts and recent multi-center studies) confirm that CAC retains predictive value in modern practice and that diabetes-specific enhancers (duration, albuminuria, microvascular complications) are associated with higher CAC burden. These contemporary findings support generalisability of our principal observation—that cumulative disease exposure as approximated by duration strongly correlates with calcific coronary disease. [13–17]

Clinical implications and recommendations

Given the strong correlation and independent association between duration and CAC in our cohort, clinicians should consider diabetes duration when deciding on advanced cardiac risk stratification. For patients with long diabetes duration (especially >10 years) — particularly when other risk enhancers are present — coronary calcium scoring or CT angiography may unmask high atherosclerotic burden and identify candidates for intensified preventive measures (high-intensity statin, aggressive BP control, SGLT2i/GLP-1 RA when indicated). This is consistent with screening/management suggestions in recent literature. [9,11]

Limitations and future directions

As a cross-sectional, single-centre study with purposive sampling, causality cannot be established and selection bias may influence the high proportion with CAC > 400. Sample size limitations may explain why binary logistic modelling (high CAC) did not reach significance for duration despite strong linear associations. Prospective studies with larger, diverse T2DM populations and standardized adjustment for treatment exposures (lipid-lowering, antihypertensives, antiglycaemic agents) are needed to better define duration thresholds and to test whether CAC-guided management improves outcomes. Recent large and multi-ethnic studies provide a framework for such work.

Summary statement

In summary, our data show a strong, independent association between duration of T2DM and CAC burden — supporting the view that duration is a practical, clinically meaningful marker of cumulative vascular risk and may help identify patients who benefit most from imaging-based risk stratification and aggressive preventive strategies.

Conclusion

The present study demonstrates a strong, independent association between the duration of type 2 diabetes mellitus and coronary artery calcium (CAC) burden. As the duration of diabetes increased, CAC scores rose significantly, indicating progressive subclinical atherosclerosis. In contrast, glycaemic control (HbA1c) showed only a moderate, non-independent relationship with CAC, suggesting that cumulative metabolic exposure may be a more robust determinant of coronary calcification than momentary glycaemic status.

These findings support the inclusion of diabetes duration as a key clinical variable when assessing cardiovascular risk in patients with type 2 diabetes. Individuals with longer disease duration—particularly beyond a decade—may benefit from CAC screening or coronary CT angiography to identify high-risk patients who could gain from intensified preventive strategies such as aggressive lipid lowering, blood pressure control, and the use of cardioprotective glucose-lowering agents.

Future multicentric, prospective studies with larger and ethnically diverse populations are warranted to validate these results and to define optimal duration thresholds for CAC screening in diabetes care.

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