e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(8); 1200-1209

Original Research Article

Comparative Coronary Angiographic Profile in Diabetic and Non-Diabetic Patients with Coronary Artery Disease: A Cross-Sectional Study from Central India

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Received: 01-05-2026 / Revised: 15-06-2026 / Accepted: 21-07-2026

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Conflict of interest: Nil

Abstract

Background: Diabetes mellitus is an independent determinant of coronary artery disease severity, yet comparative angiographic data from central India remain limited.

Objective: To compare the extent and severity of coronary artery disease between diabetic and non-diabetic patients undergoing coronary angiography.

Methods: This observational cross-sectional study enrolled 226 consecutive patients with angiographically confirmed coronary artery disease at a tertiary care hospital in Bhopal over 18 months, allocated equally to diabetic and non-diabetic groups. Clinical, biochemical, electrocardiographic, echocardiographic and angiographic parameters were compared using the chi-square or Fisher's exact test and the unpaired t test.

Results: Diabetic patients were older (58.4 ± 9.2 versus 54.6 ± 10.8 years, $p = 0.005$) with higher body mass index, hypertension (67.3% versus 42.5%, $p < 0.001$) and dyslipidaemia (72.6% versus 51.3%, $p = 0.001$). Symptoms, electrocardiographic diagnosis and biomarker elevation did not differ. Triple vessel disease was far more frequent in diabetics (41.6% versus 16.8%; odds ratio 3.52, 95% confidence interval 1.90–6.54; $p < 0.001$), whereas single vessel disease predominated in non-diabetics (46.0% versus 24.8%, $p < 0.001$). Diabetics had more severe lesions (55.8% versus 36.3%, $p = 0.003$), greater lesion burden (2.8 ± 1.2 versus 1.9 ± 0.8, $p < 0.001$), more complex lesion morphology, and lower ejection fraction (44.6 ± 11.8% versus 48.4 ± 10.6%, $p = 0.012$).

Conclusion: Diabetes was associated with substantially more extensive, severe and complex coronary artery disease despite a clinical presentation indistinguishable from that of non-diabetic patients, supporting early angiographic evaluation in this group.

Keywords: Coronary Artery Disease; Diabetes Mellitus; Coronary Angiography; Triple Vessel Disease; Lesion Complexity; India.

DOI: 10.25258/ijcpr.18.8.180

Introduction

Coronary artery disease remains the single largest cause of death worldwide and accounts for a disproportionate share of premature mortality in low- and middle-income countries (Ralapanawa & Sivakanesan, 2021; World Health Organization, 2025). India carries one of the heaviest burdens globally, with coronary disease presenting five to ten years earlier and in a more extensive form than in Western populations (Sreeniwas Kumar & Sinha, 2020). Rapid urbanisation, dietary transition and a rising prevalence of metabolic risk factors have accelerated this trend. Among the recognised determinants of coronary disease, diabetes mellitus occupies a distinctive position. Landmark epidemiological work established a two- to fourfold excess risk of coronary events in diabetic individuals (Qazi & Malik, 2013), and a graded relationship between glycaemic exposure and vascular complications has since been demonstrated repeatedly (King et al., 1999; Zoungas et al., 2012). The magnitude and consistency of this association led to the characterisation of diabetes as a coronary heart disease risk equivalent (Hajar, 2017). India is now

home to approximately 90 million adults with diabetes, a figure projected to exceed 150 million by 2050 (International Diabetes Federation, 2025), so the intersection of these two epidemics is of immediate clinical relevance. Beyond simply increasing risk, diabetes appears to alter the phenotype of coronary atherosclerosis. Chronic hyperglycaemia produces endothelial dysfunction, oxidative stress and a pro-inflammatory, prothrombotic milieu; diabetic dyslipidaemia contributes elevated triglycerides, reduced high-density lipoprotein cholesterol and small dense low-density lipoprotein particles; and insulin resistance amplifies each of these processes (Fazio et al., 2024; Juricic et al., 2025; Li et al., 2023; Wu & Parhofer, 2014). The anatomical consequence is disease that is more diffuse, more distal, more heavily calcified and more often multivessel in distribution (Morgan et al., 2004; Scarsini et al., 2022; Shrivastav et al., 2023).

Coronary angiography, introduced by Sones in 1958, remains the reference standard for defining this anatomy (Mohyeldin et al., 2024). It permits assessment of the number of vessels involved, the location and severity of stenoses and the morphological complexity of individual lesions — the parameters that most directly determine whether percutaneous coronary intervention or bypass surgery is the more appropriate strategy (Farkouh et al., 2012; Liang et al., 2021). Characterising these differences therefore has consequences that extend well beyond description.

Indian data comparing angiographic profiles in diabetic and non-diabetic patients are available but remain fragmented. Most studies are small, single-centre and methodologically heterogeneous, and few originate from central India. The present study was designed to address this gap by systematically comparing the extent and severity of coronary artery disease between diabetic and non-diabetic patients undergoing coronary angiography at a tertiary care hospital in Bhopal, Madhya Pradesh.

Materials and Methods

This single-centre, hospital-based, observational cross-sectional study was conducted over 18 months in the Department of General Medicine, L.N. Medical College and Research Centre and the associated J.K. Hospital, Bhopal, Madhya Pradesh, India.

Patients aged 18 years or above of either sex with coronary artery disease confirmed on coronary angiography were enrolled; those with incomplete or poor-quality angiographic images and those unwilling to consent were excluded. Diabetes was defined as glycated haemoglobin of 6.5% or above, or an established diagnosis of diabetes on treatment, giving a diabetic group (n = 113) and a non-diabetic group (n = 113). Sample size was calculated using the formula for a single proportion, taking prevalence as 9.5%, margin of error as 4% and Z as 1.96, and was inflated by 10% to 226. Age, sex, body mass index by Asian criteria, presenting symptoms and the risk factors of smoking, hypertension, dyslipidaemia and alcohol consumption were recorded. Blood glucose, glycated haemoglobin, lipid profile, renal function, haemoglobin, creatine kinase-MB and troponin I were measured. Electrocardiography classified patients as ST-elevation myocardial infarction, non-ST-elevation myocardial infarction, unstable angina or stable angina, and two-dimensional echocardiography categorised left ventricular ejection fraction as below 40%, 40–50% or above 50%. Angiograms were interpreted by experienced interventional cardiologists, recording the number of vessels involved, the specific vessels involved, the total number of lesions, lesion morphology (diffuse, calcified, longer than 20 mm, bifurcation), and severity graded for each patient according to the most severe lesion as Grade 1 (below 25%), Grade 2 (25–49%), Grade 3 (50–69%), Grade 4 (70–99%) or Grade 5 (total occlusion). Analysis used R version 4.5.2. Significance was set at a two-sided p value below 0.05. Secondary comparisons were numerous and should be regarded as exploratory.

Results

A total of 226 patients with angiographically confirmed coronary artery disease were enrolled, comprising 113 diabetic and 113 non-diabetic patients. All participants satisfied the inclusion criteria and provided written informed consent.

Baseline Characteristics and Risk Factor Profile: Diabetic patients were significantly older than non-diabetic patients, with a mean age of 58.4 ± 9.2 years compared with 54.6 ± 10.8 years ($p = 0.005$), a difference of 3.8 years. The distribution across age bands did not itself differ significantly ($p = 0.238$), with the sixth decade accounting for the largest proportion in both groups. Male predominance was evident throughout, giving an overall male to female ratio of 2.42 to 1, and gender distribution did not differ between groups (69.0% versus 72.6% male, $p = 0.558$).

Body mass index was markedly higher among diabetic patients (26.8 ± 3.4 versus 24.2 ± 3.1 kg/m², $p < 0.001$), and the distribution across body mass index categories differed significantly ($p < 0.001$). Among cardiovascular risk factors, hypertension (67.3% versus 42.5%, $p < 0.001$) and dyslipidaemia (72.6% versus 51.3%, $p = 0.001$) were significantly more prevalent in the diabetic group. Smoking and alcohol consumption were numerically more common among non-diabetic patients but neither difference reached statistical significance (Table 1).

Table 1: Baseline demographic, anthropometric and cardiovascular risk profile of the study population (n = 226)

Variable	Diabetic (n=113)	Non-diabetic (n=113)	Total (n=226)	p value
Age				
≤ 40 years	6 (5.3)	14 (12.4)	20 (8.8)	
41–50 years	22 (19.5)	28 (24.8)	50 (22.1)	
51–60 years	42 (37.2)	38 (33.6)	80 (35.4)	0.238[a]
61–70 years	35 (31.0)	26 (23.0)	61 (27.0)	
> 70 years	8 (7.1)	7 (6.2)	15 (6.6)	
Mean age (years)	58.4 ± 9.2	54.6 ± 10.8	56.5 ± 10.2	0.005*
Gender				
Male	78 (69.0)	82 (72.6)	160 (70.8)	0.558
Female	35 (31.0)	31 (27.4)	66 (29.2)	
Body mass index (Asian criteria)				
Normal (< 23 kg/m²)	22 (19.5)	42 (37.2)	64 (28.3)	
Overweight (23–24.9)	6 (5.3)	28 (24.8)	34 (15.0)	< 0.001*[b]
Obese I (25–29.9)	56 (49.6)	39 (34.5)	95 (42.0)	
Obese II (≥ 30)	29 (25.7)	4 (3.5)	33 (14.6)	
Mean BMI (kg/m²)	26.8 ± 3.4	24.2 ± 3.1	25.5 ± 3.5	< 0.001*
Cardiovascular risk factors				
Smoking	54 (47.8)	62 (54.9)	116 (51.3)	0.287
Hypertension	76 (67.3)	48 (42.5)	124 (54.9)	< 0.001*
Dyslipidaemia	82 (72.6)	58 (51.3)	140 (61.9)	0.001*
Alcohol consumption	32 (28.3)	38 (33.6)	70 (31.0)	0.388

Values are expressed as n (%) or mean ± standard deviation. *Statistically significant ($p < 0.05$). [a] Chi-square for the overall age-group distribution: $\chi^2 = 5.515$, df = 4, $p = 0.238$. [b] Chi-square for the overall body mass index category distribution: $\chi^2 = 42.467$, df = 3, $p < 0.001$.

Clinical presentation, electrocardiographic diagnosis and cardiac biomarkers: Chest pain was the dominant presenting symptom in both groups, reported by 78.8% of diabetic and 86.7% of non-diabetic patients, a difference that did not reach significance (p = 0.113). No individual symptom differed significantly between the groups. Shortness of breath, dizziness, syncope, vomiting or nausea and epigastric pain were each numerically more frequent among diabetic patients, but all p values exceeded 0.2. The distribution of electrocardiographic diagnoses was almost identical between groups ($\chi^2 = 0.313$, df = 3, p = 0.958). ST-elevation myocardial infarction was the commonest presentation in both groups (40.7% versus 44.2%), followed by non-ST-elevation myocardial infarction and unstable angina; stable angina accounted for only 3.5% of presentations in each group, reflecting the predominantly acute case-mix of a tertiary referral centre. Elevation of creatine kinase-MB and troponin I was likewise comparable between the two groups (Table 2).

Table 2: Clinical presentation, electrocardiographic diagnosis and cardiac biomarker status (n = 226)

Variable	Diabetic (n=113)	Non-diabetic (n=113)	Total (n=226)	p value
Presenting symptoms[a]				
Chest pain	89 (78.8)	98 (86.7)	187 (82.7)	0.113
Shortness of breath	67 (59.3)	58 (51.3)	125 (55.3)	0.229
Palpitation	42 (37.2)	38 (33.6)	80 (35.4)	0.578
Dizziness	28 (24.8)	22 (19.5)	50 (22.1)	0.336
Syncope	11 (9.7)	8 (7.1)	19 (8.4)	0.472
Vomiting / nausea	16 (14.2)	11 (9.7)	27 (11.9)	0.305
Epigastric pain	18 (15.9)	12 (10.6)	30 (13.3)	0.239
Electrocardiographic diagnosis				
STEMI	46 (40.7)	50 (44.2)	96 (42.5)	0.590
NSTEMI	37 (32.7)	34 (30.1)	71 (31.4)	0.667
Unstable angina	26 (23.0)	25 (22.1)	51 (22.6)	0.874
Stable angina	4 (3.5)	4 (3.5)	8 (3.5)	1.000
Cardiac biomarkers				
CK-MB elevated	72 (63.7)	68 (60.2)	140 (61.9)	0.584
Troponin I elevated	78 (69.0)	74 (65.5)	152 (67.3)	0.571

Values are expressed as n (%). No comparison in this table reached statistical significance. [a] Symptom categories are not mutually exclusive; each symptom was compared independently between groups. Chi-square for the overall distribution of electrocardiographic diagnoses: $\chi^2 = 0.313$, df = 3, p = 0.958.

Biochemical parameters and left ventricular function: As expected, all measures of glycaemia were substantially higher in the diabetic group, with a mean glycated haemoglobin of 8.2 ± 1.6% compared with 5.4 ± 0.6% (p < 0.001), indicating suboptimal glycaemic control in the diabetic cohort. The lipid profile showed the characteristic diabetic pattern: higher total cholesterol, triglycerides and low-density lipoprotein cholesterol with lower high-density lipoprotein cholesterol, each difference reaching statistical significance. Serum urea and creatinine were significantly higher and haemoglobin significantly lower among diabetic patients. Left ventricular systolic function was significantly poorer in the diabetic group, with a mean ejection fraction of 44.6 ± 11.8% compared with 48.4 ± 10.6% (p = 0.012). Severe systolic dysfunction was more frequent among diabetics (37.2% versus 24.8%, p = 0.044) while preserved function was correspondingly less frequent (29.2% versus 44.2%, p = 0.019); the overall distribution across ejection fraction categories differed significantly (p = 0.041) (Table 3).

Table 3: Biochemical parameters and left ventricular systolic function (n = 226)

Parameter	Diabetic (n=113)	Non-diabetic (n=113)	p value
Glycaemic profile			
Fasting blood sugar (mg/dL)	156.8 ± 42.6	94.2 ± 12.4	< 0.001*
Postprandial blood sugar (mg/dL)	224.6 ± 58.4	128.4 ± 18.6	< 0.001*
HbA1c (%)	8.2 ± 1.6	5.4 ± 0.6	< 0.001*
Lipid profile			
Total cholesterol (mg/dL)	198.4 ± 42.8	182.6 ± 38.4	0.004*
Triglycerides (mg/dL)	172.8 ± 54.6	148.2 ± 42.4	< 0.001*
HDL cholesterol (mg/dL)	38.6 ± 8.4	42.8 ± 9.2	< 0.001*
LDL cholesterol (mg/dL)	124.8 ± 36.4	110.4 ± 32.6	0.002*
Renal function and haemogram			
Serum urea (mg/dL)	38.4 ± 14.6	32.6 ± 11.2	< 0.001*
Serum creatinine (mg/dL)	1.18 ± 0.42	0.96 ± 0.28	< 0.001*
Haemoglobin (g/dL)	12.4 ± 1.8	13.2 ± 1.6	< 0.001*
Left ventricular systolic function			
EF < 40% (severe dysfunction)	42 (37.2)	28 (24.8)	0.044*
EF 40–50% (moderate dysfunction)	38 (33.6)	35 (31.0)	0.670
EF > 50% (preserved function)	33 (29.2)	50 (44.2)	0.019*
Mean LVEF (%)	44.6 ± 11.8	48.4 ± 10.6	0.012*

Values are expressed as mean ± standard deviation or n (%). *Statistically significant (p < 0.05). Chi-square for the overall distribution of left ventricular ejection fraction categories: $\chi^2 = 6.405$, df = 2, p = 0.041. EF, ejection fraction; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction.

Angiographic Profile: The extent of coronary involvement differed markedly between the two groups ($\chi^2 = 19.279$, df = 2, p < 0.001). Triple vessel disease was present in 41.6% of diabetic patients compared with 16.8% of non-diabetic patients, corresponding to more than a threefold increase in odds (odds ratio 3.52, 95% confidence interval 1.90–6.54; p < 0.001). Conversely, single vessel disease predominated among non-diabetic patients (46.0% versus 24.8%, p < 0.001). Double vessel disease occurred with comparable frequency in both groups. All four major coronary vessels were significantly more often involved in diabetic patients. The left anterior descending artery was the most frequently affected vessel in both groups (81.4% versus 69.0%, p = 0.031), followed by the right coronary artery (65.5% versus 46.0%, p = 0.003) and the left circumflex artery (60.2% versus 42.5%, p = 0.008). Left main coronary artery involvement, although least common overall, was more than twice as frequent among diabetics (15.9% versus 7.1%, p = 0.037). Lesion severity also differed significantly between groups (p = 0.041).

Total occlusion was 2.5 times more prevalent in diabetic patients (13.3% versus 5.3%, p = 0.039), and severe lesions of Grade 4 or 5 were present in 55.8% of diabetics compared with 36.3% of non-diabetics (odds ratio 2.21, 95% confidence interval 1.30–3.77; p = 0.003). The mean number of lesions per patient was substantially higher in the diabetic group (2.8 ± 1.2 versus 1.9 ± 0.8, p < 0.001). Every marker of morphological complexity examined favoured the same direction: diffuse disease (51.3% versus 28.3%, p < 0.001), calcified lesions (40.7% versus 21.2%, p = 0.002), long lesions exceeding 20 mm (46.0% versus 24.8%, p < 0.001) and

bifurcation lesions (33.6% versus 19.5%, p = 0.016) were all significantly more prevalent among diabetic patients (Table 4).

Table 4: Angiographic profile: extent, distribution, severity and morphological complexity of coronary lesions (n = 226)

Angiographic variable	Diabetic (n=113)	Non-diabetic (n=113)	p value	Odds ratio (95% CI)
Extent of coronary artery disease				
Single vessel disease	28 (24.8)	52 (46.0)	< 0.001*	0.39 (0.22–0.68)
Double vessel disease	38 (33.6)	42 (37.2)	0.578	0.86 (0.50–1.48)
Triple vessel disease	47 (41.6)	19 (16.8)	< 0.001*	3.52 (1.90–6.54)
Coronary vessel involved[a]				
Left main coronary artery	18 (15.9)	8 (7.1)	0.037*	2.49 (1.03–5.98)
Left anterior descending	92 (81.4)	78 (69.0)	0.031*	1.97 (1.06–3.65)
Left circumflex	68 (60.2)	48 (42.5)	0.008*	2.05 (1.20–3.48)
Right coronary artery	74 (65.5)	52 (46.0)	0.003*	2.23 (1.30–3.80)
Severity of stenosis				
Grade 1 (< 25%)	8 (7.1)	12 (10.6)	0.349	–
Grade 2 (25–49%)	14 (12.4)	22 (19.5)	0.146	–
Grade 3 (50–69%)	28 (24.8)	38 (33.6)	0.143	–
Grade 4 (70–99%)	48 (42.5)	35 (31.0)	0.073	–
Grade 5 (100% occlusion)	15 (13.3)	6 (5.3)	0.039*	2.73 (1.02–7.31)
Severe lesions (Grade 4 + 5)	63 (55.8)	41 (36.3)	0.003*	2.21 (1.30–3.77)
Mean lesions per patient	2.8 ± 1.2	1.9 ± 0.8	< 0.001*	–
Lesion morphology[a]				
Diffuse disease	58 (51.3)	32 (28.3)	< 0.001*	2.67 (1.54–4.63)
Calcified lesions	46 (40.7)	24 (21.2)	0.002*	2.55 (1.42–4.58)
Long lesions (> 20 mm)	52 (46.0)	28 (24.8)	< 0.001*	2.59 (1.47–4.55)
Bifurcation lesions	38 (33.6)	22 (19.5)	0.016*	2.10 (1.14–3.85)

Values are expressed as n (%) or mean ± standard deviation. *Statistically significant (p < 0.05). [a] Categories are not mutually exclusive; each category was compared independently between groups. Chi-square for the overall distribution of disease extent: $\chi^2 = 19.279$, df = 2, p < 0.001. Chi-square for the overall distribution of stenosis grade: $\chi^2 = 9.986$, df = 4, p = 0.041. CI, confidence interval.

Electrocardiographic presentation in relation to angiographic findings: When angiographic findings were examined within each electrocardiographic diagnostic stratum, diabetic patients consistently showed more extensive and more severe disease across every presentation. The strongest stratum-specific signal was seen among patients presenting with unstable angina, in whom triple vessel disease was present in 50.0% of diabetics but only 8.0% of non-diabetics (χ^2 = 11.975, df = 2, p = 0.003). Among patients presenting with non-ST-elevation myocardial infarction, severe lesions were significantly more frequent in diabetics (62.2% versus 32.4%, χ^2 = 5.171, df = 1, p = 0.023). After stratification by electrocardiographic presentation, the association between diabetes and angiographic burden persisted for triple vessel disease (common odds ratio 3.06, p < 0.001), for severe lesions (common odds ratio 2.38, p = 0.003) and, more marginally, for severe left ventricular dysfunction (common odds ratio 1.90, p = 0.046), indicating that the anatomical differences observed were not explained by differences in the mode of clinical presentation (Table 5).

Table 5: Electrocardiographic diagnosis correlated with angiographic extent, lesion severity and left ventricular function, stratified by diabetes status (n = 226)

ECG diagnosis	Group	n	Vessel extent, n (%)			Lesion severity, n (%)		LVEF category, n (%)		
			SVD	DVD	TVD	Grade 1–3	Grade 4–5	EF < 40%	EF 40–50%	EF > 50%
STEMI	Diabetic	46	14 (30.4)	17 (37.0)	15 (32.6)	17 (37.0)	29 (63.0)	23 (50.0)	15 (32.6)	8 (17.4)
	Non-diabetic	50	21 (42.0)	19 (38.0)	10 (20.0)	25 (50.0)	25 (50.0)	17 (34.0)	19 (38.0)	14 (28.0)
NSTEMI	Diabetic	37	8 (21.6)	12 (32.4)	17 (45.9)	14 (37.8)	23 (62.2)	12 (32.4)	13 (35.1)	12 (32.4)
	Non-diabetic	34	14 (41.2)	12 (35.3)	8 (23.5)	23 (67.6)	11 (32.4)	6 (17.6)	10 (29.4)	18 (52.9)
Unstable angina	Diabetic	26	6 (23.1)	7 (26.9)	13 (50.0)	17 (65.4)	9 (34.6)	6 (23.1)	8 (30.8)	12 (46.2)
	Non-diabetic	25	15 (60.0)	8 (32.0)	2 (8.0)	21 (84.0)	4 (16.0)	4 (16.0)	5 (20.0)	16 (64.0)
Stable angina	Diabetic	4	0 (0.0)	2 (50.0)	2 (50.0)	2 (50.0)	2 (50.0)	1 (25.0)	2 (50.0)	1 (25.0)
	Non-diabetic	4	2 (50.0)	1 (25.0)	1 (25.0)	3 (75.0)	1 (25.0)	1 (25.0)	1 (25.0)	2 (50.0)

Values are expressed as n (%) of the row total within each block.

Stratum-specific comparisons between diabetic and non-diabetic patients: Vessel extent — STEMI: $\chi^2 = 2.349$, df = 2, p = 0.309. NSTEMI: $\chi^2 = 4.758$, df = 2, p = 0.093. Unstable angina: $\chi^2 = 11.975$, df = 2, p = 0.003*. Stable angina: Fisher's exact p = 0.486.

Lesion severity — STEMI: $\chi^2 = 1.169$, df = 1, p = 0.280. NSTEMI: $\chi^2 = 5.171$, df = 1, p = 0.023*. Unstable angina: $\chi^2 = 1.449$, df = 1, p = 0.229. Stable angina: Fisher's exact p = 1.000.

LVEF category — STEMI: $\chi^2 = 2.845$, df = 2, p = 0.241. NSTEMI: $\chi^2 = 3.471$, df = 2, p = 0.176. Unstable angina: Fisher's exact p = 0.478. Stable angina: Fisher's exact p = 1.000.

Association between diabetes and angiographic outcome, adjusted for electrocardiographic presentation (Cochran–Mantel–Haenszel test): Triple vessel disease: common odds ratio 3.06, $\chi^2 = 12.704$, df = 1, p < 0.001*.

Severe lesions (Grade 4–5): common odds ratio 2.38, $\chi^2 = 8.640$, df = 1, p = 0.003*.

Left ventricular ejection fraction below 40%: common odds ratio 1.90, $\chi^2 = 3.995$, df = 1, p = 0.046*.

*Statistically significant (p < 0.05). Fisher's exact test was used in strata where any expected cell count was below five. DVD, double vessel disease; SVD, single vessel disease; TVD, triple vessel disease.

Discussion

This comparison of 226 patients produced a clinically important dissociation: diabetic and non-diabetic patients presented to hospital looking much the same, but their coronary anatomy differed substantially. Symptom profile, electrocardiographic diagnosis and cardiac biomarker elevation were statistically indistinguishable, yet diabetic patients carried more than three times the odds of triple vessel disease, more than double the odds of severe stenosis, a greater lesion burden, more complex lesion morphology on every measure examined, and poorer left ventricular systolic function.

Diabetic patients were 3.8 years older than their non-diabetic counterparts, which differs from Indian series reporting earlier presentation in diabetics (Hegde et al., 2014; Sharma et al., 2019) and from others finding no age difference (Kumar et al., 2025; Sareddy et al., 2021). Since every patient here already had established coronary disease, the difference reflects the point at which patients crossed the threshold for angiographic referral rather than the onset of atherosclerosis.

The male preponderance of 2.42 to 1 is consistent with Indian coronary epidemiology (Hasabi & Mudagall, 2020). Hypertension, dyslipidaemia and body mass index were all significantly higher among diabetics, reproducing the metabolic clustering described in comparable cohorts (Pandey et al., 2016; Sareddy et al., 2021); hypertension was one of only three findings here to survive a conservative correction for multiple comparisons. Smoking and alcohol were numerically higher in non-diabetics, suggesting the two groups reach

similar endpoints by partly different routes — metabolic in diabetics, predominantly tobacco-driven in non-diabetics.

No symptom differed significantly between groups and the distribution of electrocardiographic diagnoses was almost identical, matching Reddy et al. (2020) and Kumar et al. (2025). Chest pain was numerically less frequent among diabetics, directionally consistent with the reduced pain perception attributed to cardiac autonomic neuropathy (Shams et al., 2025) and with reports of atypical presentation in diabetic coronary disease (Sareddy et al., 2021), but the difference did not reach significance. Symptoms were recorded as reported at presentation, and the study was not designed to detect silent ischaemia, which requires systematic screening of asymptomatic individuals. The actionable point is the negative finding itself: if presentation, electrocardiographic category and biomarker release carry no discriminatory information about anatomical burden, clinical assessment alone will underestimate disease extent in precisely the group in whom it matters most.

Triple vessel disease in 41.6% of diabetics versus 16.8% of non-diabetics places this cohort within the published range, alongside 50% versus 18% (Gavli et al., 2024), 42.5% versus 23% (Dyrmishi et al., 2018), 39.6% versus 6.7% (Shaher et al., 2023), 38% versus 22.6% (Sridevi et al., 1995) and 35.2% versus 24.0% (Gui et al., 2009). Studies reporting multivessel rather than triple vessel disease describe the same gradient, with 47.9% versus 18.3% (Sareddy et al., 2021) and 52.8% versus 15.3% (Hasabi & Mudagall, 2020), and the mirror-image predominance of single vessel disease among non-diabetics reproduces published patterns (Gui et al., 2009; Shaher et al., 2023). All four major epicardial vessels were more frequently involved in diabetics, the left anterior descending being most often affected in both groups as reported by Shaher et al. (2023). Left main involvement was more than twice as frequent among diabetics, in keeping with earlier Indian data (Sharma et al., 2019), though this comparison was of borderline statistical robustness.

Diabetic patients had not only more diseased vessels but worse disease within them: total occlusion was two and a half times more common, severe stenosis affected more than half, and the mean lesion count was nearly one lesion per patient higher. Diffuse disease, calcification, long lesions and bifurcation lesions were all significantly more prevalent, aligning with Parvez et al. (2025), Sharma et al. (2019) and Girdhar (2018), who reported both more diffuse lesions and a greater average degree of stenosis in diabetics. Endothelial dysfunction, oxidative stress, inflammation and diabetic dyslipidaemia together produce a longitudinally continuous rather than focal atherosclerotic process, with smaller reference vessel calibre and heavier calcium burden (Morgan et al., 2004; Scarsini et al., 2022; Wu & Parhofer, 2014). Mean ejection fraction was correspondingly lower in diabetics, although some studies found no echocardiographic difference (Kumar et al., 2025). Two explanations are likely to operate together: the greater ischaemic burden documented here, and the independent contribution of diabetic cardiomyopathy, in which substrate shift, advanced glycation end-product accumulation, fibrosis and impaired calcium handling degrade myocardial performance independently of epicardial disease (Al Hroob et al., 2019; Li et al., 2023).

Stratified analysis produced the most clinically striking observation in this study. Among patients presenting with unstable angina — conventionally the least alarming acute coronary presentation — half of the diabetic patients had triple vessel disease compared with 8.0% of non-diabetics, consistent with the classic demonstration that diabetes remains the dominant risk determinant in unstable coronary disease even after angiographic extent is taken into account (Norhammar et al., 2004). A reassuring clinical label may therefore conceal a far more threatening anatomy, arguing for a low threshold for early angiography rather than conservative observation. This pattern maps directly onto the evidence governing revascularisation: the FREEDOM trial established the advantage of bypass surgery over percutaneous intervention in diabetics with multivessel disease (Farkouh et al., 2012), a large national cohort confirmed higher three-year mortality after percutaneous intervention in diabetics (Lehto et al., 2021), and meta-analytic data show lower all-cause mortality and markedly reduced repeat revascularisation with surgery, offset by increased stroke risk (Liang et al., 2021). Poorer ventricular function strengthens the case further (Nagendran et al., 2018). Alongside revascularisation planning, the clustering of hypertension, dyslipidaemia and obesity identifies modifiable targets, and the mean glycated haemoglobin of 8.2% indicates substantial scope for improved glycaemic control, which is itself graded in its relationship to angiographic severity (Sajjanar et al., 2024; Zoungas et al., 2012).

Several limitations qualify these findings. The study was single-centre, hospital-based and cross-sectional, so no outcomes were followed and no causal inference is possible; sampling from a tertiary referral centre introduces referral bias, and the predominantly acute case-mix limits generalisability. The sample size was calculated for prevalence estimation rather than between-group comparison, so the study was not formally powered for the comparisons reported. Angiographic severity was assessed by vessel count and visual

percentage stenosis rather than quantitative angiography or composite indices such as the SYNTAX or Gensini scores, limiting comparability with studies using those instruments (Aksu & Ahmed, 2024); interpretation was not performed by blinded dual readers and inter-observer variability was not quantified. Duration of diabetes, glycaemic strata and medication history were not analysed against angiographic severity, so the dose–response relationship between glycaemic exposure and disease burden could not be examined (Dal Canto et al., 2019). Prediabetes was neither excluded from the comparison group nor analysed separately, and small-vessel and distal-bed disease were not quantified. Finally, the comparisons are unadjusted: no multivariable modelling was undertaken to account for the higher age, blood pressure, lipid and renal burden of the diabetic group, so findings other than the principal angiographic outcomes should be regarded as hypothesis-generating.

Conclusion

In this cross-sectional comparison of 226 patients with angiographically confirmed coronary artery disease, diabetes was associated with markedly more extensive, more severe and more morphologically complex coronary disease. Diabetic patients had over three times the odds of triple vessel disease, more than double the odds of severe stenosis, a higher lesion burden, more diffuse, calcified, long and bifurcation lesions, and significantly poorer left ventricular systolic function — despite a clinical presentation, electrocardiographic diagnosis and biomarker profile that were statistically indistinguishable from those of non-diabetic patients. The novelty of this work lies in demonstrating that this anatomical excess persists even within individual electrocardiographic presentation categories, and is most pronounced among patients presenting with unstable angina, where clinical reassurance is most likely to be misleading. Clinical assessment alone therefore underestimates coronary disease burden in diabetic patients. These findings support early and liberal angiographic evaluation in diabetic patients with suspected coronary disease, careful anatomical risk assessment before selecting a revascularisation strategy, and aggressive management of the hypertension, dyslipidaemia, obesity and hyperglycaemia that cluster within this group.

References

1. Aksu, F., & Ahmed, S. A. (2024). Gensini score's severity and its relationship with risk factors for coronary artery disease among patients who underwent angiography in Somalia's largest PCI centre. International Journal of General Medicine, 17, 187–192. https://doi.org/10.2147/IJGM.S384626
2. Al Hroob, A. M., Abukhalil, M. H., Hussein, O. E., & Mahmoud, A. M. (2019). Pathophysiological mechanisms of diabetic cardiomyopathy and the therapeutic potential of epigallocatechin-3-gallate. Biomedicine & Pharmacotherapy, 109, 2155–2172. https://doi.org/10.1016/j.biopha.2018.11.086
3. Dal Canto, E., Ceriello, A., Rydén, L., Ferrini, M., Hansen, T. B., Schnell, O., Standl, E., & Beulens, J. W. (2019). Diabetes as a cardiovascular risk factor: An overview of global trends of macro and micro vascular complications. European Journal of Preventive Cardiology, 26(2_suppl), 25–32. https://doi.org/10.1177/2047487319878371
4. Dyrmishi, B., Olldashi, T., Lumi, E., Puca, E., & Ylli, D. (2018). Comparative angiographic profile in diabetic and non-diabetic patients (retrospective study). Endocrine Abstracts, 56, P416. https://doi.org/10.1530/endoabs.56.P416
5. Farkouh, M. E., Domanski, M., Sleeper, L. A., Siami, F. S., Dangas, G., Mack, M., Yang, M., Cohen, D. J., Rosenberg, Y., Solomon, S. D., Desai, A. S., Gersh, B. J., Magnuson, E. A., Lansky, A., Boineau, R., Weinberger, J., Ramanathan, K., Sousa, J. E., Rankin, J., … Fuster, V. (2012). Strategies for multivessel revascularization in patients with diabetes. New England Journal of Medicine, 367(25), 2375–2384. https://doi.org/10.1056/NEJMoa1211585
6. Fazio, S., Mercurio, V., Tibullo, L., Fazio, V., & Affuso, F. (2024). Insulin resistance/hyperinsulinemia: An important cardiovascular risk factor that has long been underestimated. Frontiers in Cardiovascular Medicine, 11, 1380506. https://doi.org/10.3389/fcvm.2024.1380506
7. Gavli, J., Rai, J. K., Mehrotra, N., & Arya, S. P. D. V. (2024). A clinical study of comparative angiographic profile among diabetic and non-diabetic patients with acute coronary syndrome. Journal of Cardiovascular Disease Research, 15(1), 1517–1525.
8. Girdhar, R. (2018). Coronary angiographic (CAG) findings between diabetic and non-diabetic patients in coronary artery disease: A comparative study. Journal of Medical Science and Clinical Research, 6(8). https://doi.org/10.18535/jmscr/v6i8.126
9. Gui, M. H., Qin, G. Y., Ning, G., Hong, J., Li, X. Y., Lu, A. K., Chen, X., Zhou, W. W., & Chen, M. D. (2009). The comparison of coronary angiographic profiles between diabetic and nondiabetic patients with coronary artery disease in a Chinese population. Diabetes Research and Clinical Practice, 85(2), 213–219. https://doi.org/10.1016/j.diabres.2009.05.010

10. Hajar, R. (2017). Diabetes as "coronary artery disease risk equivalent": A historical perspective. Heart Views, 18(1), 34–37. https://doi.org/10.4103/HEARTVIEWS.HEARTVIEWS_37_17
11. Hasabi, I. S., & Mudagall, G. S. (2020). A comparative study of angiographic severity of coronary artery disease in diabetic and non-diabetic patients with acute coronary syndrome by Gensini scoring system. Journal of Evidence Based Medicine and Healthcare, 7(21), 1009–1013. https://doi.org/10.18410/jebmh/2020/219
12. Hegde, S. S., Mallesh, P., Yeli, S. M., Gadad, V. M., & Punith Kumar, M. G. (2014). Comparative angiographic profile in diabetic and non-diabetic patients with acute coronary syndrome. Journal of Clinical and Diagnostic Research, 8(9), MC07–MC10. https://doi.org/10.7860/JCDR/2014/9072.4851
13. International Diabetes Federation. (2025). Diabetes facts and figures. https://idf.org/about-diabetes/diabetes-facts-figures/
14. Juricic, S., Klac, J., Stojkovic, S., Zivkovic, M., Kostic, J., Beleslin, B., & Tesic, M. (2025). Molecular and pathophysiological mechanisms leading to ischemic heart disease in patients with diabetes mellitus. International Journal of Molecular Sciences, 26(9), 3924. https://doi.org/10.3390/ijms26093924
15. King, P., Peacock, I., & Donnelly, R. (1999). The UK Prospective Diabetes Study (UKPDS): Clinical and therapeutic implications for type 2 diabetes. British Journal of Clinical Pharmacology, 48(5), 643–648. https://doi.org/10.1046/j.1365-2125.1999.00092.x
16. Kumar, S. S., Karthikeyan, S., Kumar, S. S., & Munusamy, T. (2025). Clinical and angiographic profiles of diabetic and non-diabetic patients in a tertiary care centre. [Journal name, volume and page numbers to be completed by the authors.]
17. Lehto, H. R., Winell, K., Pietilä, A., Niiranen, T. J., Lommi, J., & Salomaa, V. (2021). Outcomes after coronary artery bypass grafting and percutaneous coronary intervention in diabetic and non-diabetic patients. European Heart Journal – Quality of Care and Clinical Outcomes, 8(6), 692–700. https://doi.org/10.1093/ehjqcco/qcab065
18. Li, Y., Liu, Y., Liu, S., Gao, M., Wang, W., Chen, K., Huang, L., & Liu, Y. (2023). Diabetic vascular diseases: Molecular mechanisms and therapeutic strategies. Signal Transduction and Targeted Therapy, 8(1), 152. https://doi.org/10.1038/s41392-023-01400-z
19. Liang, B., He, X., & Gu, N. (2021). Reassessing revascularization strategies in coronary artery disease and type 2 diabetes mellitus. Frontiers in Cardiovascular Medicine, 8, 738620. https://doi.org/10.3389/fcvm.2021.738620
20. Mohyeldin, M., Mohamed, F. O., Mustafa, A., Allu, S., & Shrivastava, S. (2024). F. Mason Sones Jr.: The serendipitous discovery of coronary angiography and its lasting impact on cardiology. Cureus, 16(5), e61080. https://doi.org/10.7759/cureus.61080
21. Morgan, K. P., Kapur, A., & Beatt, K. J. (2004). Anatomy of coronary disease in diabetic patients: An explanation for poorer outcomes after percutaneous coronary intervention and potential target for intervention. Heart, 90(7), 732–738. https://doi.org/10.1136/hrt.2003.021014
22. Nagendran, J., Bozso, S. J., Norris, C. M., McAlister, F. A., Appoo, J. J., Moon, M. C., & Freed, D. H. (2018). Coronary artery bypass surgery improves outcomes in patients with diabetes and left ventricular dysfunction. Journal of the American College of Cardiology, 71(8), 819–827. https://doi.org/10.1016/j.jacc.2017.12.024
23. Norhammar, A., Malmberg, K., Diderholm, E., Lagerqvist, B., Lindahl, B., Rydén, L., & Wallentin, L. (2004). Diabetes mellitus: The major risk factor in unstable coronary artery disease even after consideration of the extent of coronary artery disease and benefits of revascularization. Journal of the American College of Cardiology, 43(4), 585–591. https://doi.org/10.1016/j.jacc.2003.08.050
24. Pandey, P., Chandrakar, R. K., Dobariya, P., Namewar, P. K., & Pandey, P. (2016). A comparative study of coronary artery disease in diabetics and non-diabetics. International Journal of Research in Medical Sciences, 4(6), 2252–2258. https://doi.org/10.18203/2320-6012.ijrms20161795
25. Parvez, M., Habib, M., & Ali, A. (2025). Impact of diabetes on coronary angiographic findings in ST-elevation myocardial infarction patients: A comparative study. Cureus, 17. https://doi.org/10.7759/cureus.91675
26. Qazi, M. U., & Malik, S. (2013). Diabetes and cardiovascular disease: Insights from the Framingham Heart Study. Global Heart, 8(1), 43–48. https://doi.org/10.1016/j.gheart.2012.12.008
27. Ralapanawa, U., & Sivakanesan, R. (2021). Epidemiology and the magnitude of coronary artery disease and acute coronary syndrome: A narrative review. Journal of Epidemiology and Global Health, 11(2), 169–177. https://doi.org/10.2991/jegh.k.201217.001
28. Reddy, R., Sharath, C. M., & Manjunath, M. E. (2020). Coronary artery involvement in diabetic and non-diabetic patients with acute coronary syndrome: A case control study. Journal of Cardiovascular Medicine and

Surgery, 6(1), 24–28. https://doi.org/10.21088/jcms.2454.7123.6120.4
29. Sajjanar, S. L., Math, M. S., & Mishra, J. (2024). Angiographic severity of coronary artery disease in patients with acute coronary syndrome in correlation to their glycemic status. European Journal of Cardiovascular Medicine, 14, 83–88.
30. Sareddy, P., Pandya, H. B., Sumple, R. S., & Lakhani, J. D. (2021). Diabetic CAD versus non-diabetic CAD: A comparative study of clinical features, risk factors and angiographic profile. International Journal of Advances in Medicine, 8(7), 927–933. https://doi.org/10.18203/2349-3933.ijam20212403
31. Scarsini, R., Fezzi, S., Leone, A. M., De Maria, G. L., Pighi, M., Marcoli, M., Tavella, D., Pesarini, G., Banning, A. P., Barbato, E., Escaned, J., & Ribichini, F. (2022). Functional patterns of coronary disease: Diffuse, focal, and serial lesions. JACC: Cardiovascular Interventions, 15(21), 2174–2191. https://doi.org/10.1016/j.jcin.2022.07.015
32. Shaher, A., Marof, B., & Boubou, A. (2023). Comparison of coronary diseases by coronary angiography between diabetic and non-diabetic patients. International Journal of Medical Science, 10(2). https://doi.org/10.14445/23939117/IJMS-V10I2P101
33. Shams, P., Gul, Z., & Makaryus, A. N. (2025). Silent myocardial ischemia. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK536915/
34. Sharma, M. K., Kurmi, P., Ameta, D., & Chandan, C. B. (2019). Comparative study of coronary angiographic findings between diabetic and non-diabetic patients. International Journal of Medical and Biomedical Studies, 3(5), 204–209. https://doi.org/10.32553/ijmbs.v3i5.267
35. Shrivastav, D., Dabla, P. K., Singh, D. D., & Mehta, V. (2023). Type 2 diabetes mellitus and coronary artery stenosis: A risk pattern association study. Exploration of Medicine, 4(3), 336–342. https://doi.org/10.37349/emed.2023.00145
36. Sreeniwas Kumar, A., & Sinha, N. (2020). Cardiovascular disease in India: A 360 degree overview. Medical Journal Armed Forces India, 76(1), 1–3. https://doi.org/10.1016/j.mjafi.2019.12.005
37. Sridevi, C., Krishna, L. S. R., Padmanabhan, T. N. C., & Jaishankar, S. (1995). A comparative study of coronary angiographic data between diabetics and non-diabetics. [Journal name, volume and page numbers to be completed by the authors.]
38. World Health Organization. (2025). Cardiovascular diseases (CVDs). https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)
39. Wu, L., & Parhofer, K. G. (2014). Diabetic dyslipidemia. Metabolism, 63(12), 1469–1479. https://doi.org/10.1016/j.metabol.2014.08.010
40. Zoungas, S., Chalmers, J., Ninomiya, T., Li, Q., Cooper, M. E., Colagiuri, S., Fulcher, G., de Galan, B. E., Harrap, S., Hamet, P., Heller, S., MacMahon, S., Marre, M., Poulter, N., Travert, F., Patel, A., Neal, B., & Woodward, M. (2012). Association of HbA1c levels with vascular complications and death in patients with type 2 diabetes: Evidence of glycaemic thresholds. Diabetologia, 55(3), 636–643. https://doi.org/10.1007/s00125-011-2404-1.