

Comparative Analysis of Coronary Angiographic Patterns in Patients with and Without Diabetes: An Observational Hospital-Based Investigation

Roonak Hamid Wani*,^{ID} Abhishek Gupta,^{ID} and Mustafa Bashir^{ID}

Department of Cardiovascular Technology, University School of Allied Health Sciences, Rayat Bahra University, Mohali, Punjab, India

ABSTRACT

Background: Type 2 diabetes mellitus represents a key modifiable contributor to coronary artery disease (CAD), typically resulting in more widespread and intense atherosclerotic changes than in individuals without diabetes. Coronary angiography (CAG) serves as the primary method for determining the distribution, extent, and degree of CAD. The current investigation examined differences in CAG outcomes among diabetic and non-diabetic subjects.

Methods: Conducted as a single-centre observational study, this work enrolled 60 individuals (aged 35–70 years) referred for CAG due to suspected CAD. Participants were categorized into diabetic (n=30) and non-diabetic (n=30) cohorts. Evaluated features encompassed the count of affected vessels (normal, single-, double-, or triple-vessel disease), degree of narrowing (<50%, 50–70%, >70%), and complications arising after the procedure. Comparisons relied on chi-square and Fisher's exact tests, with significance set at $p < 0.05$.

Results: Subjects with diabetes displayed markedly greater multi-vessel involvement, notably triple-vessel disease, along with a higher frequency of critical narrowing (>70%) relative to the non-diabetic group ($p < 0.001$ for vessel distribution; $p < 0.05$ for stenosis grade). The left anterior descending artery emerged as the predominant site of involvement across both cohorts. Procedure-related issues, including pain and haemorrhage, occurred more often among

diabetics. Notable variations appeared in age, body weight, and BMI ($p < 0.05$).

Conclusion: The presence of diabetes correlates with increased CAD severity and breadth as revealed by angiography, reinforcing the value of prompt identification and intensive control of cardiovascular risks in affected individuals.

Keywords: Diabetes Mellitus, Coronary Artery Disease, Coronary Angiography, Multi-Vessel Involvement, Lesion Severity.

Introduction

Diabetes mellitus (DM), particularly type 2, is a major risk factor for coronary artery disease (CAD) and is often considered equivalent to established CAD in terms of cardiovascular risk [1,2]. Globally, diabetes prevalence continues to rise, with significant contributions from India [3-5]. CAD remains a leading cause of mortality, and diabetes accelerates its progression through endothelial dysfunction, chronic hyperglycemia, advanced glycation end products (AGEs), oxidative stress, and activation of pathways such as diacylglycerol-protein kinase C [6-14].

Endothelial dysfunction in DM impairs vasodilation due to reduced nitric oxide availability, promotes oxidative stress, and enhances plaque formation and vulnerability [11-21]. Diabetic patients develop more aggressive atherosclerosis, with higher rates of multi-vessel involvement, diffuse lesions, and poorer outcomes post-acute coronary events [22-28]. Despite overall declines in CAD mortality, improvements are less pronounced in diabetic populations [29].

Coronary angiography is the gold standard for assessing CAD severity and pattern [30]. Multiple studies have shown that diabetic patients exhibit more extensive disease, including higher prevalence of triple-vessel disease, severe stenosis, and diffuse involvement compared to non-diabetics [31-37].

This study aimed to compare coronary angiographic findings between diabetic and non-diabetic patients, focusing on vessel involvement, stenosis severity, and post-procedure adverse effects.

Materials and Methods

This observational study was conducted in the Department of Cardiology, North End Hospital, Tapper, Baramulla, following institutional ethics committee approval.

Study Population: Sixty consecutive patients aged 35–70 years undergoing CAG for suspected CAD were included. Group A: Diabetic patients (n=30, confirmed diagnosis or ongoing treatment); Group B: Non-diabetic patients (n=30, normal fasting glucose, HbA1c, and history).

Inclusion Criteria: Age 35–70 years, BMI <30 kg/m², SpO₂ >90% on room air, normal serum creatinine and potassium.

Exclusion Criteria: Age >70 years, BMI >30 kg/m², pregnancy, ejection fraction <35%, chronic kidney disease, deranged creatinine, permanent pacemaker, septal defect, significant coronary dissection.

Procedure: Detailed clinical history, baseline investigations (fasting blood sugar, HbA1c, lipid profile, ECG, echocardiography), and informed consent were obtained. CAG was performed via femoral or radial access using the Judkins technique under local anaesthesia, with images acquired in standard projections.

Assessment Parameters:

Demographics: Age, gender, height, weight, BMI.

Citation: Roonak Hamid Wani, Abhishek Gupta, and Mustafa Bashir (2026). Comparative Analysis of Coronary Angiographic Patterns in Patients with and Without Diabetes: An Observational Hospital-Based Investigation. *Journal of American Medical Science and Research*. DOI: <https://doi.org/10.51470/AMSR.2026.05.01.06>

Received 14 November 2025

Revised 10 December 2025

Accepted 03 January 2026

Corresponding Author: Roonak Hamid Wani

Email Address: ronaqwani000@gmail.com

Copyright: © The Author(s) 2026. This article is Open Access under a Creative Commons Attribution 4.0 International License, allowing use, sharing, adaptation, and distribution with appropriate credit. License details: <http://creativecommons.org/licenses/by/4.0/>. Data is under the CC0 Public Domain Dedication (<http://creativecommons.org/publicdomain/zero/1.0/>) unless otherwise stated.

Angiographic: Number of vessels involved (normal/SVD/DVD/TVD), specific vessels (LAD, LCX, RCA, LMCA), stenosis severity (<50%, 50–70%, >70%).

Post-CAG adverse effects: Pain, bleeding, allergic reaction.

Statistical Analysis: Categorical variables were analysed using chi-square or Fisher's exact tests; continuous variables with Student's t-test. A p-value <0.05 was considered statistically significant.

Results

Table 1: Comparison of Demographic Variables between Diabetic and Non-diabetic Patients

Variable	Diabetic (n=30)	Non-diabetic (n=30)	p-value
Age (years)	64.10 ± 7.16	59.40 ± 9.26	0.032
Male	21 (70.0%)	22 (73.3%)	0.77
Female	9 (30.0%)	8 (26.7%)	0.77
Height (cm)	164.47 ± 5.43	165.40 ± 6.12	0.535
Weight (kg)	70.67 ± 5.02	60.37 ± 5.05	<0.001
BMI (kg/m ²)	26.31 ± 2.45	22.15 ± 2.48	<0.001

Student's t-test, Chi-square test

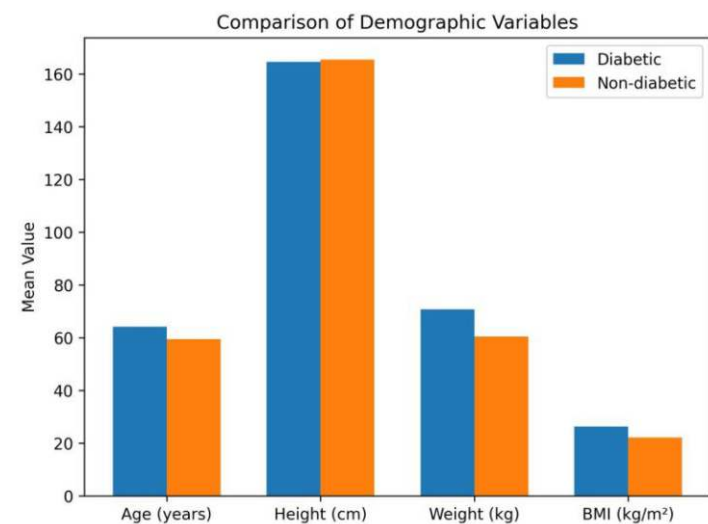


Figure 1: Comparison of demographic variables between diabetic and non-diabetic patients

This table compares basic patient information like age, height, weight, BMI, and gender between the two groups. Diabetic patients were significantly older (higher mean age), had greater body weight, and higher BMI than non-diabetic patients ($p < 0.05$ using Student's t-test). There was no significant difference in height or gender distribution. These findings suggest that the diabetic group had slightly older and heavier patients, which could contribute to higher cardiovascular risk [Table 1].

Table 2: Comparison of Angiographic Findings Based on Number of Vessels Involved

Number of vessels involved	Diabetic n (%)	Non-diabetic n (%)	p-value
Normal coronary arteries	0 (0.00)	6 (20.0%)	0.024
Single vessel disease (SVD)	3 (10.0%)	14 (46.7%)	0.003
Double vessel disease (DVD)	9 (30.0%)	7 (23.3%)	0.771
Tripple vessel disease (TVD)	18 (60.0%)	3 (10.0%)	<0.001

Chi-square test, Fisher's exact test

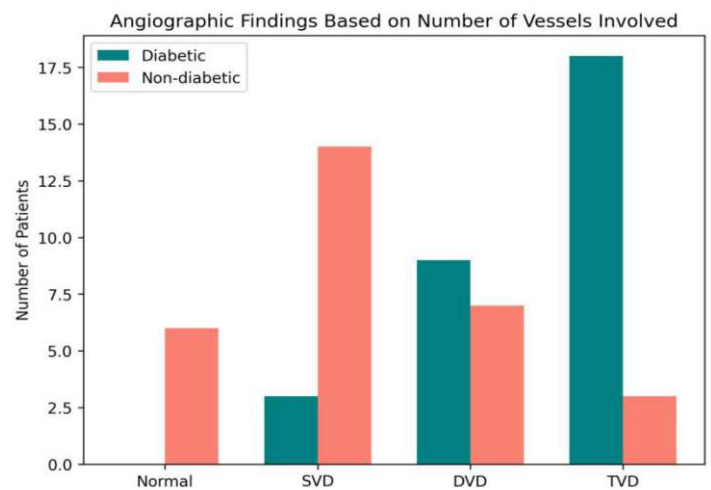


Figure 2: Angiographic findings based on the number of vessels involved

The table shows the pattern of coronary artery involvement: normal CAG, single vessel disease (SVD), double vessel disease (DVD), and triple vessel disease (TVD). In diabetic patients, multi-vessel disease (especially DVD and TVD) was much more common. In non-diabetic patients, single-vessel disease or completely normal angiograms were seen more often. The overall difference between the groups was highly significant (Pearson's Chi-square test, $p < 0.001$), and individual comparisons were also significant (Fisher's exact test, $p < 0.05$). This clearly indicates that diabetes is linked to more widespread coronary artery blockages [Table 2].

Table 3: Comparison of Stenosis Severity and Post-CAG Adverse Effects between Diabetic and Non-diabetic Patients

% stenosis	Diabetic n (%)	Non-diabetic n (%)	p-value
<50%	2 (6.7%)	12 (40.0%)	0.005
50-70%	5 (16.7%)	10 (33.3%)	0.233
>70%	23 (76.7%)	8 (26.7%)	0.022

Chi-square test

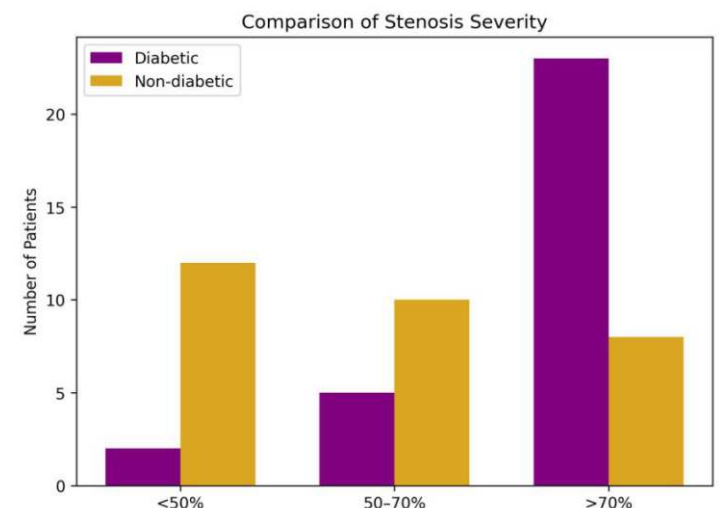


Figure 3: Stenosis severity in diabetic and non-diabetic patients

This table compares the degree of narrowing (stenosis) in the coronary arteries: mild (<50%), moderate (50–70%), and severe (>70%). Severe stenosis (>70%) was significantly more frequent in the diabetic group, while mild stenosis was more common in non-diabetic patients (Chi-square test, $p < 0.05$). This means blockages in diabetic patients were generally tighter and more dangerous [Table 3].

Table 4: Post-CAG adverse effects

Adverse effect	Diabetic n (%)	Non-diabetic n (%)
Pain	7 (23.3%)	2 (6.7%)
Bleeding	6 (20.0%)	2 (6.7%)
Allergic reaction	2 (6.7%)	1 (3.3%)

Chi-square test

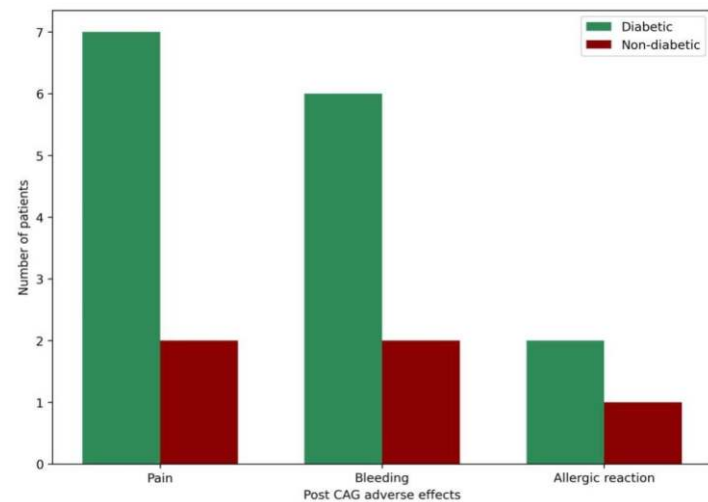


Figure 4: Post-CAG adverse effects in diabetic and non-diabetic patients

This table looks at complications right after the coronary angiography procedure: pain at the puncture site, bleeding/hematoma, and allergic reaction to contrast. Pain and bleeding occurred more frequently in diabetic patients compared to non-diabetics (Chi-square test, $p < 0.05$). Allergic reactions were very rare in both groups. The higher rate of local complications in diabetics may be due to weaker blood vessels or other diabetes-related factors [Table 4].

Discussion

The present study demonstrates that diabetic patients have more extensive and severe coronary artery disease on angiography compared to non-diabetic patients. Multi-vessel involvement, particularly triple-vessel disease, and severe stenosis (>70%) were significantly higher in diabetics, aligning with previous findings [31-37].

Similar patterns were reported by Siddiqui et al. (2023), who observed higher triple-vessel disease in diabetic females with acute coronary syndrome [32]; Girdhar et al. (2018), who noted increased multi-vessel disease, diffuse lesions, and greater stenosis in diabetics [31]; and Al Baker et al. (2023), who found higher positive lesions, multi-vessel involvement, and severe stenosis in diabetics [33]. Parvin et al. (2014), Sharma et al. (2018), Bettamer et al. (2021), Moosavi et al. (2006), and Çakır & Gören (2023) consistently reported more diffuse, extensive, and vulnerable plaque characteristics in diabetic patients [34-37].

Mechanisms such as endothelial dysfunction, chronic hyperglycemia, and AGEs contribute to accelerated atherosclerosis and plaque instability in DM [6-21]. Higher post-procedure adverse effects in diabetics may relate to vascular fragility or comorbidities.

Conclusion

Diabetic patients exhibit more severe and widespread coronary artery disease on CAG compared to non-diabetics, with increased multi-vessel involvement and severe stenosis. These findings highlight the need for early screening, strict glycemic control, and comprehensive risk-factor management to improve outcomes in this high-risk population.

References

- Ahmad E, Lim S, Lamptey R, Webb DR, Davies MJ. (2022) Type 2 diabetes. *The Lancet*;400(10365):1803-1820.
- Grundy S, Becker D, Clark LT, Cooper RS, Denke MA, Howard J, Hunninghake DB, Illingworth DR, Luepker RV, McBride P, McKenney JM (2002). Detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *Circulation*, 106(25):3143.
- International Diabetes Federation (2017). IDF Diabetes Atlas. International Diabetes Federation.
- World Health Organization (2016). Global report on diabetes. Geneva: World Health Organization.
- Institute for Health Metrics and Evaluation (n.d.). GBD compare data visualization.
- Goligorsky MS (2005). Endothelial cell dysfunction: can't live with it, how to live without it. *American Journal of Physiology- Renal Physiology*, 288(5):F871-F880.
- Deanfield J, Donald A, Ferri C, Giannattasio C, Halcox J, Halligan S, Lerman A, Mancia G, Oliver JJ, Pessina AC, Rizzoni D, Rossi GP, Salvetti A, Schiffrin EL, Taddei S, Webb DJ (2005). Endothelial function and dysfunction. Part I: methodological issues for assessment in the different vascular beds. *Journal of Hypertension*, 23(1):7-17.
- Roberts AC, Porter KE (2013). Cellular and molecular mechanisms of endothelial dysfunction in diabetes. *Diabetes & Vascular Disease Research*, 10(6):472-482.
- Geraldes P, King GL (2010). Activation of protein kinase C isoforms and its impact on diabetic complications. *Circulation Research*, 106(8):1319-1331.
- Hadi HAR, Al Suwaidi JA (2007). Endothelial dysfunction in diabetes mellitus. *Vascular Health and Risk Management*, 3(6):853-876.
- Madonna R, De Caterina R (2011). Cellular and molecular mechanisms of vascular injury in diabetes—part II: cellular mechanisms and therapeutic targets. *Vascular Pharmacology*, 54(3-6):75-79.
- Hartge MM, Unger T, Kintscher U (2007). The endothelium and vascular inflammation in diabetes. *Diabetes and Vascular Disease Research*, 4(2):84-88.
- Manrique C, Lastra G, Sowers JR (2014). New insights into insulin action and resistance in the vasculature. *Annals of the New York Academy of Sciences*, 1311(1):138-150.
- Liu XF, Yu JQ, Dalan R, Liu AQ, Luo KQ (2014). Biological factors in plasma from diabetes mellitus patients enhance hyperglycaemia and pulsatile shear stress-induced endothelial cell apoptosis. *Integrative Biology*, 6(5):511-522.
- Piconi L, Quagliaro L, Assaloni R, Da Ros R, Maier A, Zuodar G, Ceriello A (2006). Constant and intermittent high glucose enhances endothelial cell apoptosis through mitochondrial superoxide overproduction. *Diabetes/Metabolism Research and Reviews*, 22(3):198-203.

16. Avogaro A, Albiero M, Menegazzo L, de Kreutzenberg S, Fadini GP (2011). Endothelial dysfunction in diabetes: the role of reparatory mechanisms. *Diabetes Care*, 34(Suppl 2):S285-S290.
17. McClung JA, Naseer N, Saleem M, Rossi GP, Weiss MB, Abraham NG, Kappas A (2005). Circulating endothelial cells are elevated in patients with type 2 diabetes mellitus independently of HbA1c. *Diabetologia*, 48(2):345-350.
18. Nomura S (2009). Dynamic role of microparticles in type 2 diabetes mellitus. *Current Diabetes Reviews*, 5(4):245-251.
19. Alsheikh-Ali AA, Kitsios GD, Balk EM, Lau J, Ip S (2010). The vulnerable atherosclerotic plaque: scope of the literature. *Annals of Internal Medicine*, 153(6):387-395.
20. Aso Y et al. (2021). Pathophysiological Association between Diabetes Mellitus and Endothelial Dysfunction. *Antioxidants*, 10(8):1306.
21. Aronson D, Rayfield EJ, Chesebro JH (1997). Mechanisms determining course and outcome of diabetic patients who have had acute myocardial infarction. *Annals of Internal Medicine*, 126(4):296-306.
22. McGuire DK (2008). Diabetes and the Cardiovascular System. In: Libby P, Bonow RO, Mann DL, Zipes DP, editors. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 8th ed. Philadelphia: Saunders; p. 1392-1410.
23. Gu K, Cowie CC, Harris MI (1999). Diabetes and decline in heart disease mortality in US adults. *JAMA*, 281(13):1291-1297.
24. Bryfogle JW, Bradley RF (1957). The vascular complications of diabetes mellitus. *Diabetes*, 6(2):159-167.
25. Folsom AR, Szklo M, Stevens J, Liao F, Smith R, Eckfeldt JH (1997). A prospective study of coronary heart disease in relation to fasting insulin, glucose, and diabetes. *Diabetes Care*, 20(6):935-942.
26. Lundberg V, Stegmayr B, Asplund K, Eliasson M, Huhtasaari F (1997). Diabetes as a risk factor for MI: population and gender perspectives. *Journal of Internal Medicine*, 241(6):485-492.
27. Panzram G (1987). Mortality and survival in Type II (non-insulin-dependent) diabetes mellitus. *Diabetologia*, 30(3):123-131.
28. Stamler J, Vaccaro O, Neaton JD, Wentworth D (1993). Diabetes, other risk factors, and 12-year cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. *Diabetes Care*, 16(2):434-444.
29. Gu K, Cowie CC, Harris MI (1999). Diabetes and decline in heart disease mortality in US adults. *JAMA*, 281(13):1291-1297.
30. Girdhar R, Kothari Y, Kamat AS, Raj RA, Koithara BJ (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):753-759.
31. Siddiqui MF, Khan H, Khalid MR, Korejo AA, Faizan F, Yousif A (2023). Comparison of Angiographic Findings in Diabetic and Non-Diabetic Female patients presenting with Acute Coronary Syndrome at a Tertiary Cardiac Care Center in Karachi, Pakistan. *Pakistan Journal of Medical & Health Sciences*, 17(05):219.
32. Al Baker SME, Showdagor MNH, Rahman M, Mahmood M, Habib A, Rahman F, Ahsan SA (2023). Coronary Angiographic Findings between Diabetic and non-diabetic Patients in Coronary Artery Disease: A Comparative Study. *University Heart Journal*, 19(1):5-9.
33. Parvin T, Haque KS, Siddique MA, Habib SA, Rahman M, Rahman M, Hoque MH (2015). Angiographic Severity of Coronary Artery Disease in Diabetic and Non-Diabetic Patients in a Tertiary Care Centre. *University Heart Journal*, 10(1):13-17.
34. Sharma MK, Kurmi P, Ameta D, Chandan CB (2019). Comparative Study of Coronary Angiographic Findings Between Diabetic and Nondiabetic Patients. *International Journal of Medical and Biomedical Studies*, 3(5):204-209.
35. Bettamer Z, Elkadiki AH, Alsaeiti KD (2021). Coronary Angiographic Characteristics of Type 2 DM Compared with Nondiabetic Patients in Benghazi-Libya. A Cross-Sectional Study. *Libyan Journal of Medical Sciences*, 5(3):125-127.
36. Moosavi M, Nematipour E, Mehrpouya M (2006). Comparison of Extent of Coronary Artery Disease in Angiography of Diabetics and Non-Diabetics. *Iranian Heart Journal*, 7:37-42.
37. Çakır M, Gören M (2023). Comparison of Atherosclerotic Plaque Compositions in Diabetic and Non-diabetic Patients. *Cureus*, 15(9):e45721.