

Research Article

# Risk assessment analysis of type II Diabetes Mellitus and dyslipidemia with Coronary Artery Stenosis scoring: intervention strategies

Pradeep Kumar Dabla<sup>1\*</sup>, Dharmasheel Shrivastav<sup>1</sup>, Vimal Mehta<sup>2</sup>, Swati Singh<sup>1</sup>, Rashid Mir<sup>3</sup>, Shyamalendu Kandar<sup>4</sup>

<sup>1</sup>Department of Biochemistry, Govind Ballabh Pant Institute of Postgraduate Medical Education & Research, Associated Maulana Azad Medical College, Jawaharlal, Nehru Marg, New Delhi

<sup>2</sup>Department of Cardiology, Govind Ballabh Pant Institute of Postgraduate Medical Education & Research, Associated Maulana Azad Medical College, Jawaharlal Nehru Marg, New Delhi, India

<sup>3</sup>Prince Fahd Bin Sultan Research chair; Department Of Medical Lab Technology, Faculty of Applied Medical Sciences, University of Tabuk, Tabuk, Kingdom of Saudi Arabia

<sup>4</sup>Department of Information Technology, Indian Institute of Engineering Science and Technology, Shibpur, Botanical Garden Area, Howrah, West Bengal, India

## Article Info

### \*Corresponding Author:

Pradeep Kumar Dabla  
Department of Biochemistry, Govind Ballabh Pant Institute of Postgraduate Medical Education & Research, Associated Maulana Azad Medical College, Jawaharlal Nehru Marg, New Delhi, 110002, India  
E-mail: [pradeep\\_dabla@yahoo.com](mailto:pradeep_dabla@yahoo.com)  
ORCID ID: 0000-0003-1409-6771

## Keywords

Coronary artery disease, Diabetes Mellitus, Gensini scoring system, HbA1c, Lipid profile

## Abstract

**Background:** Diabetes is an established risk factor for coronary artery disease (CAD), with substantial evidence linking it to a higher prevalence of multivessel disease and worse cardiovascular outcomes. This study aimed to explore the relationship between lipid profile and diabetes with severity of CAD by evaluating the degree of stenosis as assessed by Gensini Score (GS).

**Methods:** A total of 300 participants were included: 100 CAD patients with diabetes, 100 CAD patients without diabetes, and 100 healthy controls. Serum lipoproteins were quantified using standard colorimetric methods, while HbA1c levels were determined through HPLC. GS was assessed using angiographic data obtained from the catheterization laboratory.

**Results:** Significant differences in lipid profile, and HbA1c were observed between diabetic CAD patients, non-diabetic CAD patients, and healthy controls. The GS in patients with CAD was markedly elevated in the diabetic CAD cohort ( $32.97 \pm 22.47$ ) in comparison to the non-diabetic CAD cohort ( $28.70 \pm 20.60$ ). A positive correlation was found between HbA1c ( $r^2=0.061$ ), random blood sugar ( $r^2=0.23$ ), and TG ( $r^2=0.00$ ) with the GS. Conversely, HDL levels ( $r^2= -0.074$ ) exhibited a significant negative correlation with GS.

**Conclusion:** Our results suggest that lipoproteins and diabetic indicators have a considerable impact on the severity of CAD. This highlights the need for a more personalized approach in managing diabetes and CAD, incorporating regular monitoring of glucose and lipid levels to evaluate cardiovascular risk.

## Introduction

Cardiovascular diseases remain the primary cause of mortality worldwide, responsible for an estimated 17.5 million deaths annually [1]. Diabetes is a known independent risk factor for coronary artery disease (CAD), with evidence demonstrating a higher prevalence of adverse cardiovascular events in patients with diabetes [3-6]. An earlier study reported that diabetic patients with intermediate coronary artery stenosis had the worse outcome as compared with non-diabetic ones [4]. Diabetes with cardiac complications significantly increased the risk of death, a finding that emphasizes the importance of mitigating the risk of diabetes-related complications in CAD patients [5]. In an earlier investigation by Morgan KP et al., authors documented a higher prevalence of multivessel disease, along with extensive distal involvement, and minimal collateral formation in CAD patients with type 2 diabetes mellitus (T2DM), ultimately leading to poorer outcomes [7].

Moreover, dysregulated lipid metabolism constitutes a crucial determinant in CAD and is considered a hallmark of dysfunctional angiogenesis. Numerous clinical observations have revealed reduced collateral circulation in diabetic CAD patients [8,9]. However, the impact of dyslipidemia and hyperglycemia on the relationship between impaired endothelial function and coronary artery stenosis severity in T2DM patients remains unclear. In this study, we investigated the relationship between lipoproteins, diabetic biomarkers (HbA1c and blood glucose), and the severity of coronary artery stenosis, and highlighted their potential clinical implications in T2DM patients with CAD.

## Methodology

### Participant enrollment

This tertiary care-hospital based study was conducted in the Department of Biochemistry, G.B. Pant Institute of Postgraduate Medical Education & Research, New Delhi, India. After obtaining ethical approval, we enrolled 300 participants, including 100 diabetic CAD patients (Group I), 100 non-diabetic CAD patients (Group II), and 100 age- and sex-matched healthy controls (Group III). Written informed consent was taken from all participants. The study included patients over the age of 18, who had been diagnosed with CAD through resting electrocardiography and invasive coronary angiography, showing more than 50% stenosis in at least one coronary artery. Exclusion criteria were patients below the age of 18 years, patients with renal and hepatic impairment, patients who had undergone previous procedures such as coronary artery bypass grafting, percutaneous transluminal coronary angioplasty, and stenting.

### Specimen collection and laboratory protocol

Venous blood was withdrawn from each participant. 3 mL blood was collected in EDTA vial for HbA1c, 2 mL blood in a plain

vial for lipid analysis and 2 mL blood in sodium citrate vial for blood glucose analysis. HbA1c measurement was performed using HPLC. Patients exhibiting HbA1c levels >6.5% were classified as diabetics and those with <6.5% were classified as non-diabetics [10]. Serum lipoproteins and blood glucose were measured on Cobas c501 (Roche) fully automated analyser. Serum total cholesterol (TC) was measured using Cholesterol Oxidase-Peroxidase method, triglycerides (TG) using Glycerol Phosphate Oxidase-Peroxidase end-point enzymatic colorimetric method, high density lipoprotein (HDL), and low density lipoprotein (LDL) using direct enzymatic colorimetric methods. Very low-density lipoprotein (VLDL) level was calculated by dividing triglyceride level by five. For measurement of blood glucose, hexokinase method was used. Coronary angiography was performed on all recruited patients (group I and group II) using Judkin's approach [11] to examine angiographic patterns. The left coronary artery was divided into the left anterior descending, circumflex, and obtuse marginal branches, while the right coronary artery considered as a single vessel. Based on the highest level of obstruction in each projection, the arteries were classified as normal, 50%, 75%, 90%, or 100% occluded, determined through visual assessment [12]. The patients were categorized into three subgroups based on angiography results: single-vessel, double-vessel, and triple-vessel disease, (disease defined as obstruction of 50% or more) [13]. The severity of coronary artery stenosis was assessed using the Gensini score, calculated based on luminal narrowing: 1 point for 25%, 2 points for 50%, 4 points for 75%, 8 points for 90%, 16 points for 99%, and 32 points for total occlusion [14].

### Statistical analysis

Data was analyzed using the Statistical Package for the Social Sciences, version 22. For group comparisons, independent t-test and ANOVA was used for comparing two independent variables, and more than two variables, respectively. The Chi-square test assessed differences within the groups, while Pearson's correlation was employed to examine relationships between variables. All statistical analyses were performed with  $p < 0.05$ .

## Results

### Demographic characteristics of the study population

The mean age of group I (82 male and 11 female), group II (89 male and 18 female) and group III (54 male and 46 female) was  $54.86 \pm 9.80$ ,  $53.15 \pm 10.30$  and  $43.62 \pm 12.03$  years respectively. In group I, 39 smokers, 42 tobacco chewers and 19 are alcoholic; in group II, 61 smokers, 39 tobacco chewers and 25 are alcoholic. However, in Group III, none of the participants are smokers, tobacco chewers, or alcoholics. 34 individuals of group I and 15 individuals of group II were diagnosed with dyslipidaemia (Table 1).

**Table 1:** Demographic characteristics of the study population.

Parameter	Group I (n=100)	Group II (n=100)	Group III (n=100)
Age (Mean $\pm$ SD)	54.86 $\pm$ 9.80	53.15 $\pm$ 10.30	43.62 $\pm$ 12.03
Gender (n)			
Male	82	89	54
Females	11	18	46
Smoker (n)	39	61	0
Tobacco Chewer (n)	42	39	0
Alcoholic (n)	19	25	0
Dyslipidemia (n)	34	15	0

Age is represented as Mean  $\pm$  SD; Qualitative variables as frequencies (n)

### Comparison of biochemical parameters and Gensini score between groups I, II and III

Table 2 illustrates comparison of biochemical parameters (diabetic parameters HbA1c, random blood sugar and lipid profile) between group I, II and III. In group I, II and III, serum random blood sugar (RBS) was 210.15 $\pm$ 101.13, 120.61 $\pm$ 42.52 and 129.16 $\pm$ 9.13 respectively; and serum HbA1c was 8.84 $\pm$ 2.02, 5.66 $\pm$ 0.38 and 4.31 $\pm$ 0.75 respectively. Significant difference

was observed between group I, II and III, for both serum RBS and HbA1c levels. Among groups I, II and III, significant difference was observed for TC, HDL, LDL and VLDL ( $p < 0.001$ ), however, for serum TG, the difference observed was non-significant ( $p = 0.241$ ). The Gensini scores for Group I and Group II were 32.97 $\pm$ 22.47 and 28.70 $\pm$ 20.60, respectively, demonstrating a significant difference ( $p < 0.001$ ).

**Table 2:** Comparison of biochemical parameters and Gensini score between various groups.

Parameter	Group I (N=100)	Group II (N=100)	Group III (N=100)	P Value
RBS (mg/dL)	210.15 $\pm$ 101.13	120.61 $\pm$ 42.52	129.16 $\pm$ 9.13	0.001
HbA1c (%)	8.84 $\pm$ 2.02	5.66 $\pm$ 0.38	4.31 $\pm$ 0.75	0.001
TG (mg/dL)	149.05 $\pm$ 49.47	138.14 $\pm$ 56.34	139.16 $\pm$ 44.70	0.241
TC (mg/dL)	176.32 $\pm$ 130.55	128.47 $\pm$ 81.96	101.96 $\pm$ 36.95	0.001
HDL (mg/dL)	39.30 $\pm$ 21.30	34.25 $\pm$ 11.64	53.57 $\pm$ 15.46	0.001
LDL (mg/dL)	96.23 $\pm$ 22.25	80.37 $\pm$ 43.74	79.61 $\pm$ 36.00	0.001
VLDL (mg/dL)	34.95 $\pm$ 26.12	25.70 $\pm$ 16.38	26.74 $\pm$ 7.24	0.001
Gensini Score	32.97 $\pm$ 22.47	28.70 $\pm$ 20.60	- -	0.001

HDL: high density cholesterol level; LDL: low-density lipoprotein; VLDL: Very low-density lipoprotein; RBS: Random Blood Sugar; TC: Total cholesterol; TG: Triglyceride

### Logistic regression analysis between biochemical parameters and Gensini score

Table 3 and Figure 1 and 2 illustrate the logistic regression analysis between biochemical parameters and Gensini score. RBS ( $R^2 = 0.023$ ,  $p = 0.009$ ) and HbA1c ( $R^2 = 0.161$ ,  $P < 0.001$ ) showed significant positive association with Gensini score. TG

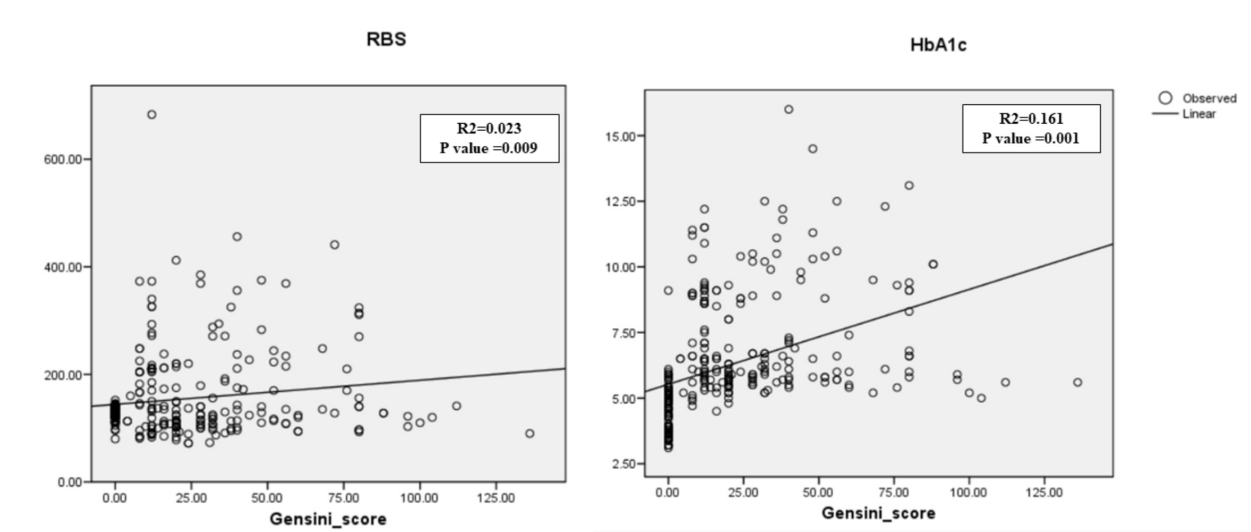
( $R^2 = 0.034$ ,  $p < 0.001$ ) and VLDL ( $R^2 = 0.009$ ,  $p < 0.001$ ) showed a significant positive correlation with the Gensini score, while HDL exhibited a negative correlation ( $R^2 = -0.007$ ,  $p < 0.001$ ). Serum TC and LDL showed no significant association with the Gensini score.

**Table 3:** Logistic regression analysis between Gensini score and biochemical parameters.

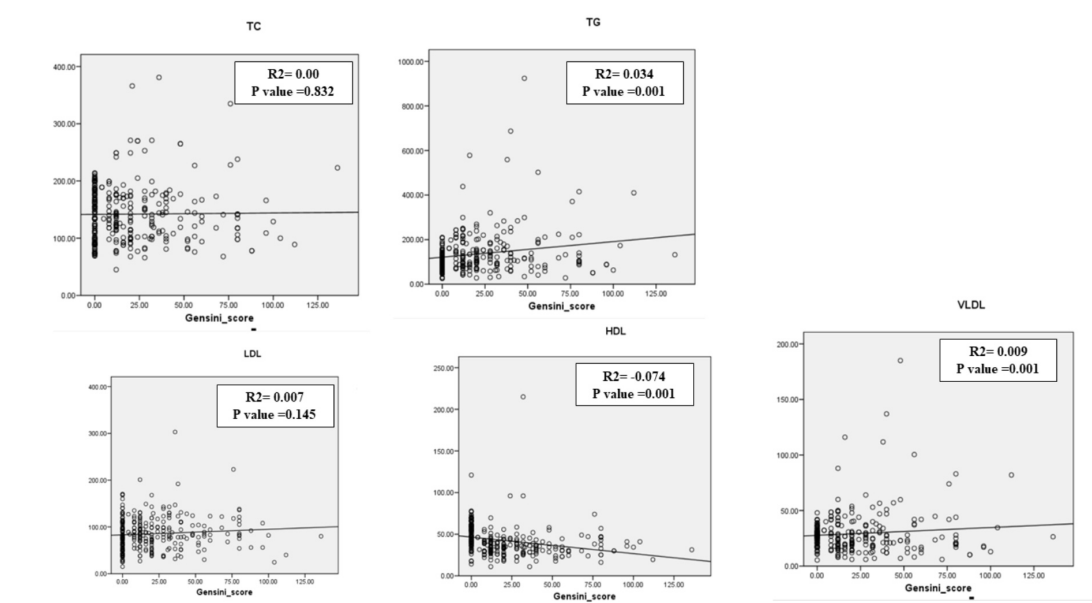
Parameter	R <sup>2</sup>	P Value
RBS	0.023	0.009
HbA1c	0.161	0.001
TC	0.00	0.832
TG	0.034	0.001
HDL	0.074	0.001
LDL	0.007	0.145
VLDL	0.009	0.09

HDL: high density cholesterol level; LDL: low-density lipoprotein; VLDL: Very low-density lipoprotein; RBS: Random Blood Sugar; TC: Total cholesterol; TG: Triglyceride; R<sup>2</sup>=coefficient of determination

**Figure 1:** Logistic regression analysis between Gensini score and Random Blood Sugar and HbA1c.



**Figure 2:** Logistic regression analysis between Gensini score and lipid profile parameters.



TC: Total cholesterol; TG: Triglyceride; HDL: high density cholesterol level; LDL: low-density lipoprotein; VLDL: Very low-density lipoprotein

## Discussion

In this cross-sectional study, we observed significant differences in diabetes-specific parameters and lipid profile between diabetic, nondiabetic CAD patients and healthy control individuals. These findings are in concordance with the observations of previous investigations. A study conducted by Ghazanfari Z. et al. identified a robust correlation between HbA1c and fasting blood sugar, particularly in individuals with diabetes [15]. Similarly, an earlier study revealed that HbA1c levels of >9.0 % are associated with higher mortality in clinically type II diabetic patients [16]. Furthermore, existing literature indicates that oxidative stress and endothelial dysfunction, commonly observed in diabetic individuals, may directly facilitate the pathogenesis of atherosclerosis and the progression of CAD [17-19]. These results imply that these parameters could assist healthcare providers in setting target plasma glucose levels with specific HbA1c values. Additionally, recent investigations have shown that while stable average glucose remains pivotal in preventing cardiovascular events, HbA1c fluctuations are an independent risk determinant for cardiovascular disease, even in patients meeting glycemic targets [20]. Yang et al. identified HbA1c variability as a predictor of in-stent restenosis in T2DM patients post-stent implantation [21]. These findings imply that HbA1c may also serve as a potential marker of glycemic control, aiding in the prevention of cardiovascular complications in CAD patients. In addition, in our study, significant differences were observed in lipid profile between diabetic, nondiabetic CAD patients and healthy controls. In agreement, Wang X et al. suggested that raised LDL and TG and decreased HDL are common in men and a significant contributor in late onset of coronary artery disease [22]. A study by Jia A et al. reported that elevated levels of serum VLDL can act as a biomarker for diabetic CAD patients [23]. The coronary artery distribution angiography investigation conducted by Hegde SS et al. posited that individuals with diabetes exhibit a significantly elevated susceptibility to multi-vessel coronary artery disease [24]. Taken together, these results highlight the clinical value of comprehensive lipid assessment and suggest that addressing dyslipidemia could potentially improve outcomes in CAD patients.

Logistic regression analysis was conducted to examine the relationship between the Gensini score (GS) and various biochemical parameters. The results revealed a positive association between RBS, HbA1c, and TG with the Gensini score, while HDL demonstrated a significant negative association. These observations are in line with the findings of earlier studies [25-27]. In a study conducted among 141 CAD patients, authors reported that GS was positively correlated with total cholesterol, LDL cholesterol and TG and a negative correlation between GS and HDL cholesterol was identified [25]. In an earlier study conducted among acute coronary syndrome patients, authors

uncovered a significant association between lipid parameters (total cholesterol, non-HDL cholesterol, and the Apo B/A ratio) and the degree of CAD, encompassing vessel involvement, specific arterial regions affected, and the distribution of disease across segments [29]. Another study compared the lipid ratios in STEMI patients and suggested that lipid ratios have significant correlation with coronary artery diseases with chest pain [29]. Overall, our findings substantiate the notion that fluctuation in HbA1c and lipid profile, may be related with the severity of CAD in individuals with T2DM, and is deserving of further attention in the context of glycemic control and managing lipid profile. However, the limited sample size may constrain the external validity of the findings, hindering their extrapolation to a more diverse population. Moreover, longitudinal studies with extended follow-up could yield stronger evidence regarding the prognostic significance of lipids and diabetic markers in CAD. By addressing these constraints, a more nuanced comprehension of the role of lipids and diabetic markers in CAD can be attained, ultimately enhancing risk stratification, and facilitating the development of tailored therapeutic strategies.

## Conclusion

In conclusion, this study underscores the critical role of Type II Diabetes Mellitus and dyslipidemia in the progression of Coronary Artery Disease. Elevated random blood sugar, HbA1c, and triglycerides levels, along with reduced HDL, were closely linked to CAD severity. Looking ahead, a more personalized approach in the management of T2DM and CAD is essential, integrating regular monitoring of glucose and lipid profiles to assess cardiovascular risk. Future research should explore novel biomarkers, including VLDL and lipid ratios, to improve risk stratification. Additionally, early intervention strategies focusing on stricter glycemic and lipid control, along with lifestyle modifications, may prevent or delay the onset of severe CAD in diabetic patients, ultimately improving long-term cardiovascular health outcomes.

## Abbreviations

CAD	Coronary Artery Disease
GS	Gensini Score
HDL	High Density Lipoprotein
LDL	Low Density Lipoprotein
RBS	Random Blood Sugar
TC	Total cholesterol
TG	Triglyceride
T2DM	Type II Diabetes Mellitus
VLDL	Very Low Density Lipoprotein

**Declarations****Acknowledgements**

None.

**Funding**

None to declare.

**Conflict of interest**

The authors declare that they have no conflicts of interest.

**Ethics approval and consent to participate**

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments. The study was approved by the Institutional Ethics Committee, Maulana Azad Medical College and associated hospitals, New Delhi, India (F1/IEC/MAMC/85/03/21/no.422; Dt-30.08.2021). Written informed consent was obtained from all participants included in the study.

**Author contributions**

P.K.D. designed and supervised the study, provided facilities for testing, contributed to data interpretation and preparation, revision and finalization of the manuscript; D.S. conducted experiments and contributed to data collection, performed data analysis; D.S. and S.S. drafted the manuscript; V.M. provided the facility for the enrolment of patients; V.M. and R.M. critically reviewed the manuscript and contributed in analysis and finalisation of the manuscript; S.K. contributed in data analysis. All authors have reviewed the entire content of this manuscript and approved it for submission.

**Data sharing statement**

Data is available from the corresponding author on reasonable request.

**Clinical trial number**

Not applicable.

**References**

1. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart Disease and Stroke Statistics-2020 Update: A Report From the American Heart Association. *Circulation*. 2020;141(9):e139-e596. doi:10.1161/CIR.0000000000000757
2. Rafieian-Kopaei M, Setorki M, Douidi M, Baradaran A, Nasri H. Atherosclerosis: process, indicators, risk factors and new hopes. *Int J Prev Med*. 2014;5(8):927-946.
3. Siscovick DS, Sotoodehnia N, Rea TD, Raghunathan TE, Jouven X, Lemaitre RN. Type 2 diabetes mellitus and the risk of sudden cardiac arrest in the community. *Rev Endocr Metab Disord*. 2010;11(1):53-59. doi:10.1007/s11154-010-9133-5
4. Zhang HW, Jin JL, Cao YX, Guo YL, Wu NQ, Zhu CG, et al. Association of diabetes mellitus with clinical outcomes in patients with different coronary artery stenosis. *Cardiovasc Diabetol*. 2021;20(1):214. doi:10.1186/s12933-021-01403-6
5. Tancredi M, Rosengren A, Svensson AM, Kosiborod M, Pivodic A, Gudbjornsdottir S, Wedel H, Clements M, Dahlqvist S, Lind M. Excess mortality among persons with type 2 diabetes. *N Engl J Med*. 2015;373(18):1720-1732. doi: 10.1056/NEJMoA1504347.
6. Whiteley L, Padmanabhan S, Hole D, Isles C. Should diabetes be considered a coronary heart disease risk equivalent?: Results from 25 years of follow-up in the Renfrew and Paisley survey. *Diabetes Care*. 2005;28(7):1588-1593. doi: 10.2337/diacare.28.7.1588.
7. Morgan KP, Kapur A, Beatt KJ. Anatomy of coronary disease in diabetic patients: an explanation for poorer outcomes after percutaneous coronary intervention and potential target for intervention. *Heart*. 2004;90(7):732-738. doi: 10.1136/hrt.2003.021014
8. Balakrishnan S, Senthil Kumar B. Factors causing variability in formation of coronary collaterals during coronary artery disease. *Folia Morphol (Warsz)*. (2021). doi: 10.5603/FM.a2021.0110
9. Liu T, Wu Z, Liu J, Lv Y, Li W. Metabolic syndrome and its components reduce coronary collateralization in chronic total occlusion: an observational study. *Cardiovasc Diabetol*. (2021) 20:104. doi: 10.1186/s12933-021-01297-4
10. Zemlin AE, Matsha TE, Hassan MS, Erasmus RT. HbA1c of 6.5% to diagnose diabetes mellitus--does it work for us?--the Bellville South Africa study. *PLoS One*. 2011;6(8):e22558. doi:10.1371/journal.pone.0022558
11. Harry L, Page Jr. The Judkins technique. *Cathet Cardiovasc Diagn*. 1979; 5(2):187-189. doi:10.1002/ccd.1810050214
12. Lipinski M, Do D, Morise A, Froelicher V. What percent luminal stenosis should be used to define angiographic coronary artery disease for noninvasive test evaluation? *Ann Noninvasive Electrocardiol*. 2002;7(2):98-105. doi:10.1111/j.1542-474X.2002.tb00149.x
13. Vyas P, Meghnathi H, Joshi H, Brahmabhatt J, Dake R, Satpute A, Patel K. Coexistent coronary artery disease in Indian patients undergoing permanent pacemaker implantation (PPI) for symptomatic bradyarrhythmia. *Indian Heart J*. 2021;73(5):577-581. doi:10.1016/j.ihj.2021.04.002
14. Avci A, Fidan S, Tabakçı MM, Toprak C, Alizade E, Acar E, et al. Association between the Gensini Score and Carotid Artery Stenosis. *Korean Circ J*. 2016;46(5):639-645. doi:10.4070/kcj.2016.46.5.639
15. Ghazanfari Z, Haghdoost AA, Alizadeh SM, Atapour J, Zolala F. A Comparison of HbA1c and Fasting Blood Sugar Tests in General Population. *Int J Prev Med*. 2010;1(3):187-194. [PMID: 21566790]
16. Nicholas J, Charlton J, Dregan A, Gulliford MC. Recent HbA1c values and mortality risk in type 2 diabetes.

- population-based case-control study. *PLoS One*. 2013;8(7):e68008. doi:10.1371/journal.pone.0068008
17. Martín-Timón I, Sevillano-Collantes C, Segura-Galindo A, Del Cañizo-Gómez FJ. Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength? *World J Diabetes*. 2014;5(4):444-470. doi:10.4239/wjd.v5.i4.444
18. Dave TH, Wasir HS, Prabhakaran D, Dev V, Das G, Rajani M, et al. Profile of coronary artery disease in Indian women: correlation of clinical, non invasive and coronary angiographic findings. *Indian Heart J*. 1991;43(1):25-29.
19. Ezhumalai B, Jayaraman B. Angiographic prevalence, and pattern of coronary artery disease in women. *Indian Heart J*. 2014;66(4):422-426. doi:10.1016/j.ihj.2014.05.009
20. Guo K, Zhao Q, Wang M, Lu Y, Wo M, Zhou X, Ying C. The Scope of HbA1c Variability and Risk of Vascular Complications Among Patients with Type 2 Diabetes: A Systematic Review and Meta-Analysis of Prospective Studies. *Horm Metab Res*. 2022;54(2):94-103. doi:10.1055/a-1730-4904
21. Yang CD, Shen Y, Lu L, Yang ZK, Hu J, Zhang RY, Shen WF, Ding FH, Wang XQ. Visit-to-visit HbA1c variability is associated with in-stent restenosis in patients with type 2 diabetes after percutaneous coronary intervention. *Cardiovasc Diabetol*. 2020;19(1):133. doi:10.1186/s12933-020-01111-7
22. Wang X, Yu D, Wang J, Huang J, Li W. Analysis of Coronary Artery Lesion Degree, and Related Risk Factors in Patients with Coronary Heart Disease Based on Computer-Aided Diagnosis of Coronary Angiography. *Comput Math Methods Med*. 2021;2021:2370496. doi:10.1155/2021/2370496
23. Jia A, Zeng W, Yu L, Zeng H, Lu Z, Song Y. Very low-density lipoprotein cholesterol is associated with extent and severity of coronary artery disease in patients with type 2 diabetes mellitus. *SAGE Open Med*. 2019;7:2050312119871786. doi:10.1177/2050312119871786
24. Hegde SS, Mallesh P, Yeli SM, Gadad VM, M GP. Comparative angiographic profile in diabetic and non-diabetic patients with acute coronary syndrome. *J Clin Diagn Res*. 2014;8(9):MC07-MC10. doi:10.7860/jcdr/2014/9072.4851
25. Gupta S, Priyadarshi A, Beg M, Rabbani MU. Decoding the Lipid-Angiogram Link: Can Serum Lipid Profile Help Predict Your Clogged Arteries? *Cureus*. 2024;16(11):e73454. doi:10.7860/jcdr/2014/9072.4851
26. Liu X, Yang X, Wu N. Relationship Between Glycosylated Hemoglobin Variability and the Severity of Coronary Artery Disease in Patients with Type 2 Diabetes Mellitus. *J Diabetes Res*. 2024 Aug 1;2024:9958586. doi:10.1155/2024/9958586
27. Shrivastav D, Dabla PK, Singh DD, Mehta V. Type 2 diabetes mellitus and coronary artery stenosis: a risk pattern association study. *Exploration of Medicine*. 2023: n. pag. doi:10.37349/emed.2023.00145
28. Tarchalski J, Guzik P, Wysocki H. Correlation between the extent of coronary atherosclerosis and lipid profile. *Mol Cell Biochem*. 2003;246(1-2):25-30. [PMID: 12841339]
29. Gao P, Wen X, Ou Q, Zhang J. Which one of LDL-C / HDL-C ratio and non-HDL-C can better predict the severity of coronary artery disease in STEMI patients. *BMC Cardiovasc Disord*. 2022;22(1):318. doi:10.1186/s12872-022-02760-0