



CORRELATION BETWEEN HBA1C LEVELS AND SEVERITY OF CORONARY ARTERY DISEASE

Dr. Wafa Hyder Maitlo^{1*}, Dr. Muhammad Nauman Khan², Dr. Farwa Anum³, Dr. Arif Mehmood Kamboh⁴, Dr. Ahsan Sikander⁵, Dr. Anum Zamir⁶

^{1*}Senior Registrar, Department of Medicine, RLKU Medical College, Lahore, Pakistan
Email: wafahyder@gmail.com

²MCPS Trainee, Department of Anesthesia, Fatima Memorial Hospital, Lahore, Pakistan
Email: misternaumankhan@gmail.com

³Post Graduate Resident, Department of Neurology, Sir Ganga Ram Hospital, Lahore, Pakistan.
Email: farwaanum32@gmail.com

⁴Senior Registrar, Department of Cardiology, Punjab Institute of Cardiology, Lahore, Pakistan.
Email: drfira001@gmail.com

⁵Medical Officer, Emergency Department, Farooq Hospital DHA, Lahore, Pakistan.
Email: ahs.sik666@gmail.com

⁶Medical Officer, Department of Medicine, Hameed Latif Hospital, Lahore, Pakistan.
Email: anum.zamir@hotmail.com

Abstract

Background: Chronic hyperglycemia is a risk factor for atherosclerotic progression, and coronary artery disease (CAD) is a major cause of morbidity and mortality in the world. One easy-to-obtain marker that could be used to assess coronary atherosclerotic burden is glycated hemoglobin (HbA1c).

Objective: To assess the relationship between HbA1c and the severity of coronary artery disease by angiography.

Methods: The study was cross-sectional analytical and was conducted at Hameed Latif Hospital, Lahore, from January, 2025 to June, 2025. Patients were recruited consecutively to the study for coronary angiography for suspected or established CAD (n = 200). HbA1C was determined by a standardized laboratory method, and the severity of the disease was determined by the number of diseased vessels. One-way ANOVA, correlation analysis, and receiver operating characteristic analysis (ROC) were used to assess the associations.

Results: The mean age was 57.8 ± 10.6 years, and 62.0% of patients were male. The mean HbA1c was significantly higher ($p < 0.001$) in patients with significant CAD ($7.9 \pm 1.3\%$) than in those without significant CAD ($6.1 \pm 0.7\%$). The number of diseased vessels was positively correlated with HbA1c ($\rho = 0.50$, $p < 0.001$). Multivessel disease was found in 72.0% of patients with $\text{HbA1c} \geq 7.0\%$ and 36.0% of patients with $\text{HbA1c} < 7.0\%$ ($p < 0.001$). The AUC for HbA1c was fair for the discrimination of multivessel disease (0.74).

Conclusion: An elevated HbA1c is highly correlated with angiographic severity of CAD and could be used to help cardiovascular risk stratification, but it is not a substitute for angiography. Larger multicenter studies are recommended.

Keywords: HbA1c; glycated hemoglobin; coronary artery disease; coronary angiography; multivessel disease; type 2 diabetes mellitus.

Introduction

Coronary artery disease (CAD) is a leading cause of morbidity and mortality in the world and is defined as a progressive narrowing of the coronary arteries due to atherosclerosis. Diabetes mellitus and glucose intolerance have been established as risk factors for atherosclerotic cardiovascular disease, and glycated hemoglobin (HbA1c) is a measure of average blood glucose levels over the past 2-3 months. Recent data have shown that higher levels of HbA1c are correlated with higher angiographic severity of CAD, which suggests that chronic glycemic exposure may be an important contributor to coronary atherosclerotic burden (1). In patients with type 2 diabetes mellitus (T2DM), chronic hyperglycemia contributes to endothelial dysfunction, oxidative stress, inflammation, and abnormalities in lipid metabolism, which is relevant to the relationship between HbA1c and coronary disease.

Higher HbA1c has been associated with significant coronary stenosis and with major adverse cardiovascular events in patients with both T2DM and coronary heart disease, suggesting a role for HbA1c in addition to its established function as a glycemic control monitor (2). It has been demonstrated that raised levels of TyG are linked to greater severity of coronary artery disease, and that insulin resistance and metabolic dysfunction may play a role in coronary atherosclerosis outside of the context of diagnosed diabetes (3). This evidence gives an important metabolic background to the study of HbA1c, as chronic hyperglycemia and insulin resistance often occur together and can have interrelated metabolic effects on vascular health. Acute disturbances of glucose metabolism can also affect the evaluation of coronary disease. Stress hyperglycemia ratio, representing the acute glucose rise in relation to the estimated chronic glycemic status, has been linked to the severity of CAD in various glucose metabolic states (4). This is significant because HbA1c is a measure of long-term glycaemic exposure, and not of acute hyperglycaemia during periods of acute illness.

New molecular markers are also supporting the biological link between hyperglycemia and coronary stenosis. Increased research interest is directed towards molecular markers of disease progression, including serum-derived exosomal hsa-let-7b-5p, which has been explored as a biomarker to predict the severity of coronary stenosis in patients with coronary heart disease and hyperglycemia (5). Likewise, angiographic disease burden has been linked to inflammatory and metabolic abnormalities. For instance, the neutrophil-to-high-density lipoprotein (HDL) cholesterol ratio is a ratio of an inflammatory cell marker to an atheroprotective lipid parameter and has been linked to the severity of CAD in stable patients (6). The results of this study show that coronary atherosclerosis is a complex process involving metabolic, inflammatory, and vascular pathways, not just hyperglycemia. Another link between diabetes and coronary atherosclerosis is oxidative modification of lipoproteins.

An increased oxidized LDL cholesterol-to-LDL cholesterol ratio has been shown to be correlated with the degree of coronary atherosclerosis in patients with T2DM, indicating that oxidative lipid injury plays a role in the development and progression of the plaque (7). In patients with CHD and T2DM, reviews of glycemic control with HbA1c highlight the need to interpret glycemic targets and cardiovascular outcomes on an individual basis (8). The TyG index has been shown to have associations

with the severity of coronary disease in patients with various glucose metabolic conditions, which indicates that insulin resistance may be a risk factor for atherosclerosis prior to the onset of clinical diabetes (9). Longitudinal studies have also investigated the association of HbA1c with the subsequent anatomical progression and clinical outcomes of CAD, supporting the notion that chronic glycemic exposure is associated not only with the presence of CAD, but also with its subsequent anatomical progression and clinical consequences (10).

These observations, combined, support the use of HbA1c as a readily available clinical marker in patients being evaluated for CAD. The TyG index has also been shown to be related to the severity of CAD in large-scale studies in various glucose metabolic conditions (11). These results imply that metabolic risk can be present on a continuum and not only in the usual diagnostic categories. This is especially important as young adults with high TyG have also been found to be associated with severe obstructive CAD and atherosclerotic target lesion failure (12). The relationship between glycemia and coronary disease could be modified by other components of the metabolic and inflammatory environment. Remnant cholesterol, or cholesterol contained in triglyceride-rich lipoproteins, and C-reactive protein, a marker of inflammation, have been shown to have a synergistic relationship with the severity of CAD (13). Moreover, the hemoglobin glycation index (HI), which represents the difference between actual HbA1C levels and predicted HbA1C levels based on glucose levels, has been studied in relation to cardiovascular outcomes in diabetes patients with CAD (14).

The results suggest that HbA1c can be clinically useful and suggest that glycemic exposure is associated with lipid abnormalities, inflammation, and inter-individual differences in hemoglobin glycation. Systemic inflammatory and oxidative pathways are also important to coronary atherosclerotic progression. In patients with coronary heart disease and diabetes, the systemic immune-inflammation index has been linked to the severity of coronary stenosis, suggesting a role of immune and inflammatory activation in coronary disease burden (15). Likewise, serum malondialdehyde, an indicator of lipid peroxidation and oxidative injury, has been studied as a predictor of severity of CAD and as a marker of cardiovascular outcomes, further supporting the importance of oxidative injury in atherosclerosis (16). Insulin resistance has been shown to be correlated with inflammatory activity in CAD patients, suggesting a link between metabolic dysfunction and inflammatory mechanisms involved in the pathogenesis of atherosclerosis (17). These findings together offer compelling evidence to investigate HbA1c as a readily available measure of chronic glycemic exposure in CAD patients. Measuring the relationship between long-term glycemic status and coronary atherosclerotic burden may be better understood if there is a correlation between the HbA1c level and angiographic severity and may also help identify patients with more extensive anatomic disease involvement.

Objective: To evaluate the relationship between HbA1c and the severity of coronary artery disease (CAD) as determined by angiography and whether there is an association between higher HbA1c and greater coronary arterial involvement.

Materials And Methods

Study Design: Cross-sectional analytical study.

Study Setting: The study was conducted at Hameed Latif Hospital, Lahore.

Study Duration: This study was carried out for a period of six months, from January, 2025 to June, 2025.

Sample Size: The study included 200 patients who were suspected to have or had been diagnosed with coronary artery disease and underwent coronary angiography.

Sample Technique: Patients were recruited using consecutive sampling, which involved recruiting patients who presented during the study period and were eligible for the study until the desired sample size was reached.

Inclusion criteria: Patients ≥ 18 years of age who had been referred for or had a coronary angiography for suspected or established coronary artery disease were included. Patients with HbA1c available at the time of evaluation for the study and good coronary angiographic visualization for the assessment of coronary severity were eligible to participate.

Exclusion criteria: Patients with acute or chronic conditions that significantly affect the interpretation of the HbA1c, such as hemoglobinopathies, significant anemia, recent blood transfusion, or advanced renal disease, were excluded. Patients with incomplete clinical, laboratory, or angiographic data were also excluded.

Methods

Demographic and clinical data were collected on a structured data collection proforma after obtaining informed consent. Age, sex, smoking, hypertension, diabetes mellitus, dyslipidaemia, and other cardiovascular risk factors were recorded. Venous blood samples were drawn for HbA1c measurement by a standardized laboratory technique. HbA1c levels were taken as percentages and used as the primary metabolic parameter. All the participants were subjected to coronary angiography as per standard institutional protocol. Angiographic results were analysed to identify the presence, location, number, and severity of coronary artery stenoses. The degree of luminal narrowing found on angiography was used to determine the presence of significant coronary artery disease, and the overall angiographic disease burden was classified based on the number and severity of involved coronary vessels. The association between HbA1c and severity of angiographic CAD was then evaluated. Correlations between HbA1c and severity of coronary disease were calculated by statistical analysis, using the appropriate test for continuous and categorical variables.

Results

There were 200 patients who had coronary angiography, 124 (62.0%) of whom were male, and the mean age was 57.8 ± 10.6 years. Of the 118 patients (59.0%) with diabetes mellitus, the mean HbA1c was 7.1%. Significant obstructive CAD was found in 170 patients (85.0%) including 62 with single vessel disease (31.0%), 58 with double vessel disease (29.0%) and 50 with triple vessel disease (25.0%). There were significant obstructive lesions in 170 patients (85.0%) of which 62 (31.0%) had single vessel disease, 58 (29.0%) had double vessel disease and 50 (25.0%) had triple vessel disease. The HbA1c levels were significantly different between the angiographic categories (Table 1). Mean HbA1c rose stepwise from $6.1 \pm 0.7\%$ in patients without significant CAD to $6.7 \pm 0.9\%$ in single-vessel, $7.2 \pm 1.1\%$ in double-vessel, and $7.9 \pm 1.3\%$ in triple-vessel disease (one-way ANOVA $F(3, 196) = 22.18$, $p < 0.001$; $\eta^2 = 0.25$, a large effect). The Kruskal-Wallis test was concordant ($H = 49.79$, $p < 0.001$). Patients with triple-vessel disease had significantly higher HbA1c levels than all other groups (all $p \leq 0.003$), with single- and double-vessel disease not significantly different (mean difference 0.49%, $p = 0.053$). This gradient is shown in Figure 1, as well as the overlap of individual distributions. The

difference between adjacent categories varied between 0.5 and 0.7 percentage points, with the greatest difference between double- and triple-vessel disease.

Table 1: Association of HbA1c Level With Angiographic Severity of Coronary Artery Disease

Angiographic category	n (%)	HbA1c, M $\pm$ SD	95% CI	Median (IQR)	Post hoc (Tukey)
No significant CAD	30 (15.0)	6.1 $\pm$ 0.7	5.84 to 6.35	6.1 (5.7 to 6.6)	Reference
Single-vessel disease	62 (31.0)	6.7 $\pm$ 0.9	6.49 to 6.93	6.8 (6.2 to 7.3)	p = 0.043 a
Double-vessel disease	58 (29.0)	7.2 $\pm$ 1.1	6.91 to 7.49	7.3 (6.4 to 8.1)	p < 0.001 a
Triple-vessel disease	50 (25.0)	7.9 $\pm$ 1.3	7.54 to 8.27	8.2 (6.8 to 8.6)	p < 0.001 a
Overall	200 (100)	7.1 $\pm$ 1.2	6.89 to 7.22	7.0 (6.2 to 7.9)	

Note. M = mean; SD = standard deviation; IQR = interquartile range. One-way ANOVA: $F(3, 196) = 22.18$, $p < 0.001$, $\eta^2 = 0.25$; Kruskal-Wallis $H = 49.79$, $p < 0.001$. a Versus no significant CAD; triple-vessel versus double-vessel $p = 0.003$; double-vessel versus single-vessel $p = 0.053$.

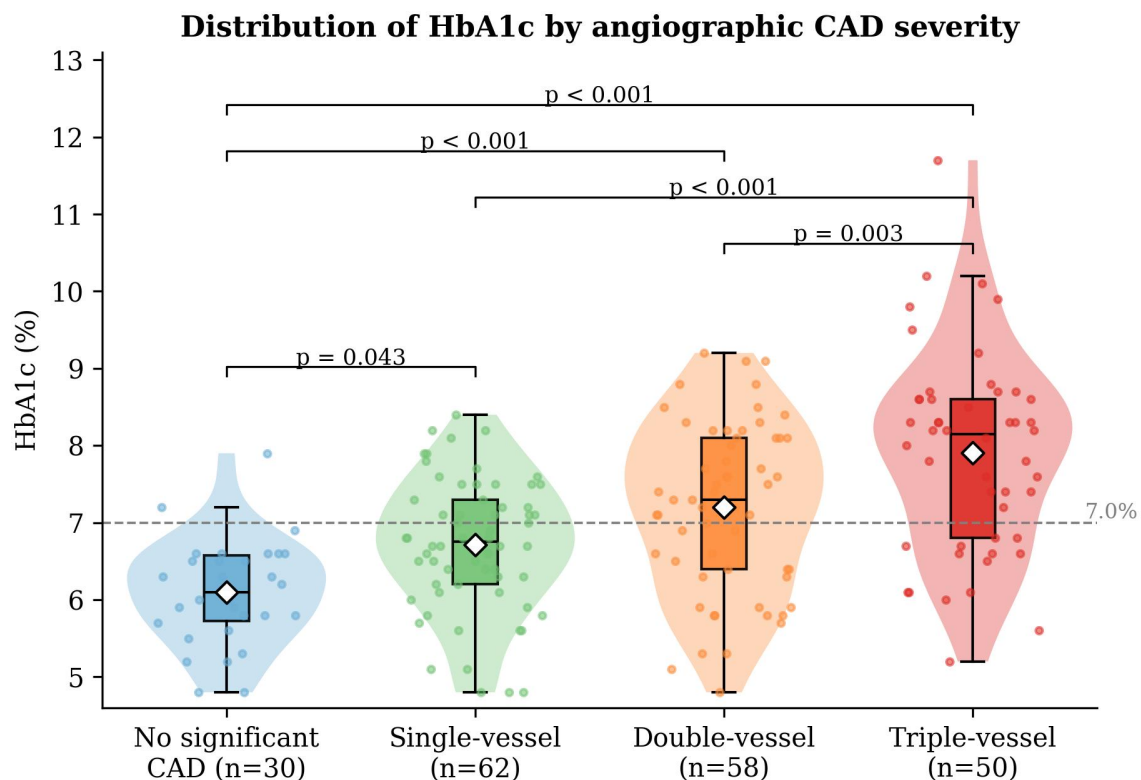


Figure 1 HbA1c Distribution Across Angiographic Categories of Coronary Artery Disease

Note. Violins show kernel density, boxes show median and interquartile range, white diamonds show means, and dots show individual patients. Brackets indicate Tukey HSD post hoc comparisons; the dashed line marks HbA1c 7.0%.

The number of diseased vessels was moderately associated with HbA1c (Spearman $\rho = 0.50$, $p < 0.001$). The proportion of multivessel disease increased across HbA1c strata (Figure 2), from 35.9% at HbA1c $< 6.5\%$ and 36.1% at 6.5% to 6.9%, to 52.9% at 7.0% to 7.9% and 91.8% at $\geq 8.0\%$ (linear-by-linear $\chi^2 = 45.34$, $p < 0.001$). Triple-vessel disease affected 55.1% (27/49) of patients with HbA1c $\geq 8.0\%$ but only 9.4% (6/64) of those with HbA1c $< 6.5\%$. When dichotomised at 7.0%, multivessel disease was present in 72.0% (72/100) of patients with HbA1c $\geq 7.0\%$ compared with 36.0% (36/100) of those below this level ($\chi^2 = 24.66$, $p < 0.001$). Overall, these results suggest a hierarchical association.

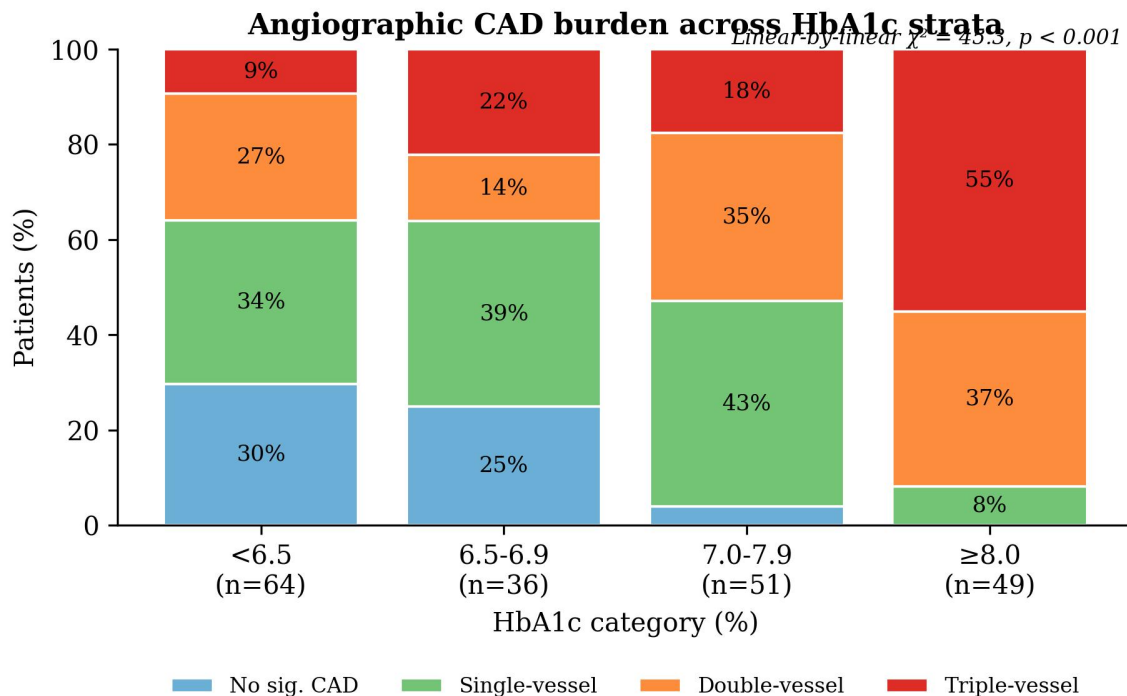


Figure 2 Distribution of Angiographic Severity Across HbA1c Categories

Note. Percentages are within each HbA1c category. Linear-by-linear association $\chi^2 = 45.34$, $p < 0.001$.

The discrimination of HbA1c was fair for the multivessel disease (AUC = 0.74, 95% CI 0.68 to 0.81; Figure 3). The diagnostic performance at selected thresholds is summarised in Table 2. The conventional 7.0% cut-off had a sensitivity of 66.7% and specificity of 69.6%, while the Youden-optimal threshold of 7.4% increased the specificity to 82.6% with a decrease in sensitivity (58.3%). The threshold cut point of 8.0% was very specific (95.7%) and had a positive predictive value of 91.8%, but sensitivity was low (41.7%) for the detection of multivessel disease. The negative predictive value was

relatively low for all thresholds (58.3–64.1%), suggesting that a lower HbA1c does not exclude extensive coronary disease and should not be used to obviate angiographic assessment.

Table 2: Diagnostic Performance of HbA1c Thresholds for Multivessel Coronary Artery Disease

HbA1c threshold	Sensitivity % (95% CI)	Specificity % (95% CI)	PPV %	NPV %	Youden J
≥ 6.5%	78.7 (70.1 to 85.4)	44.6 (34.8 to 54.7)	62.5	64.1	0.23
≥ 7.0%	66.7 (57.3 to 74.8)	69.6 (59.5 to 78.0)	72.0	64.0	0.36
≥ 7.4% (optimal)	58.3 (48.9 to 67.2)	82.6 (73.6 to 89.0)	79.7	62.8	0.41
≥ 8.0%	41.7 (32.8 to 51.1)	95.7 (89.3 to 98.3)	91.8	58.3	0.37

Note. Multivessel disease defined as double- or triple-vessel involvement (108/200). PPV = positive predictive value; NPV = negative predictive value; CI = Wilson 95% confidence interval.

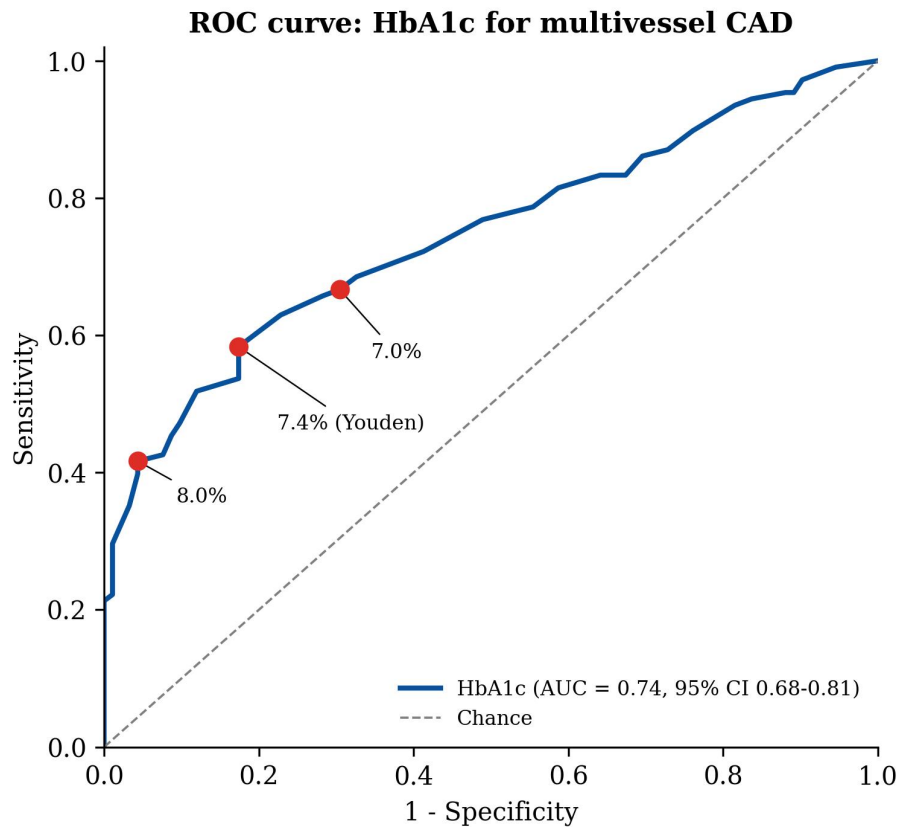


Figure 3 Receiver Operating Characteristic Curve of HbA1c for Predicting Multivessel Disease

Note. AUC = area under the curve (bootstrap 95% CI, 2,000 resamples). Red markers show selected thresholds.

Discussion

The study showed that the higher the extent of coronary involvement, the higher the HbA1c. The mean HbA1c was higher in the triple-vessel disease group compared with the double-vessel, single-vessel, and no significant obstructive disease groups. This is consistent with the notion that chronic glycemic exposure is linked to the coronary atherosclerotic burden. Yan et al. also found that there was a correlation between HbA1c and angiographic severity of CAD, further confirming its clinical significance in angiography (1). This observed relationship is consistent with that of patients with known type 2 diabetes and CHD. Jiao et al. found that HbA1c was an independent predictor of severe coronary stenosis and major adverse cardiovascular events, suggesting that worse glycemic control could be used to identify patients with higher cardiovascular anatomical and clinical risk (2).

There are a number of metabolic pathways that can account for this relationship. A surrogate marker for insulin resistance, the triglyceride-glucose index, has been linked to more severe CAD (3). Insulin resistance is often associated with abnormal glucose metabolism, and HbA1c may partly reflect a more general metabolic profile of glucose metabolism, dyslipidaemia, endothelial dysfunction and inflammation. Therefore, the observed association could be due to a combination of metabolic abnormalities and not just a single effect of chronic hyperglycemia. There is a difference between chronic and acute glycemic abnormalities. Stress hyperglycemia is an acute metabolic response, while HbA1c is an estimate of the longer-term glycemic exposure. In different glucose metabolic states, Zhang et al. found a correlation between the SHR and severity of CAD (4). Therefore, in the present study, HbA1c serves as a relatively stable indicator of previous glycemic exposure and is less influenced by the acute events of angiography.

Hyperglycemia is not the only factor that is associated with coronary stenosis by biological mechanisms. Hyperglycemia can lead to endothelial dysfunction, advanced glycation, inflammation, and vascular remodeling, as well as oxidative stress. Interest in molecular indicators of disease burden is clearly illustrated in Han et al.'s work, which examined serum-derived exosomal hsa-let-7b-5p as a biomarker of coronary stenosis severity in patients with hyperglycemia (5). Gao et al. also reported a correlation between the neutrophil-to-HDL cholesterol ratio and CAD severity, highlighting the interplay between inflammation and vascular protection related to lipids (6). The association between abnormal glycemia and coronary atherosclerosis may be accentuated by oxidative and lipid-related mechanisms. Xu et al. found a correlation between the levels of OxLDL/LDL-C and the severity of coronary atherosclerosis in type 2 diabetes, highlighting the importance of oxidative modification of atherogenic lipoproteins (7). However, these observations do not prove that reducing HbA1c is sufficient to reverse established disease. In a comprehensive cardiovascular management approach, intensified HbA1c-based glycemic control must be interpreted, as Chen et al. noted (8).

These results also corroborate other studies that have correlated insulin resistance with angiographic disease. Su et al. found that the triglyceride-glucose index was able to distinguish the severity of CAD in all glucose metabolic states, indicating that the adverse metabolic physiology may be important beyond the traditional diabetes categories (9). Dykun et al. reported that on-treatment HbA1c was independently associated with coronary atheroma progression and major adverse cardiovascular events based on serial intravascular ultrasound data even after accounting for a number of cardiovascular risk factors (10). Evidence from larger and younger populations further supports the importance of

metabolic abnormalities. In a large Asian population, Zhao et al. reported that there was a relationship between the triglyceride-glucose index and CAD severity in all glucose metabolic states (11). Higher TyG values were also related to severe obstructive CAD and atherosclerotic target lesion failure in young adults, as found by Shali et al. (12). While these studies measured insulin resistance instead of HbA1c, they do support a larger connection between the disturbance of glucose-lipid metabolism and coronary disease.

This relationship between glycemic exposure and coronary disease may be altered by inflammation and lipid abnormalities. Zhang et al. reported a synergistic relationship between remnant cholesterol and C-reactive protein for predicting CAD severity, which indicates a relationship between atherogenic lipoproteins and inflammation (13). Lin et al. also examined the relationship between HbGlycA and both diabetes and CAD and found that HbGlycA was relevant to cardiovascular outcomes, indicating that this glycation may be important for the prognosis of the patient in these studies (14). The present findings are put in perspective with additional inflammatory and oxidative markers. Wang et al. showed that the systemic immune-inflammation index is a predictor of the severity of coronary stenosis in coronary heart disease and diabetes mellitus patients (15). Hadi et al. studied malondialdehyde as an oxidative stress marker and found that it could be related to the severity of CAD and cardiovascular outcomes (16). These results are consistent with mechanisms proposed to be caused by chronic hyperglycemia, but they were not directly measured.

Last but not least, the relationship between abnormal glycemia and coronary disease could be mediated by insulin resistance and coronary inflammation. In patients with CAD, Yang et al. found an association between insulin resistance and coronary inflammatory processes, suggesting a combined metabolic-inflammatory model of atherosclerosis (17). In summary, the higher the HbA1c, the more severe the angiographic CAD was, and it may be a useful marker to identify patients with more extensive coronary involvement. However, the cross-sectional design does not allow for causal interpretation. Larger prospective studies with quantitative angiographic scoring, serial HbA1c measurements, and accounting for cardiovascular risk factors are required.

Conclusion

This study shows that there is a significant and progressive correlation between the HbA1C and the angiographic severity of coronary artery disease. The mean HbA1c increased gradually with progression of the severity of CAD, and the number of diseased vessels was moderately associated with the HbA1c. Multivessel disease occurred significantly more often in those with HbA1c levels of 7.0% or greater and was present in increasing proportions with higher levels of HbA1c. The discriminatory ability of HbA1c was fair for multivessel disease, with a threshold of approximately 7.4% providing the best balance of sensitivity and specificity. However, its relatively low negative predictive value (NPV) means that a low HbA1c does not rule out significant coronary disease. The results confirm that HbA1c is a convenient, readily available measure of chronic glycemic exposure that could help stratify cardiovascular risk in addition to angiography. More large multicenter studies with adjustment for confounding factors are recommended.

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