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Original Article

The relationship between HbA1c and atherosclerotic risk: a clinical evaluation in a non-diabetic population

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Abstract

Background and Objective*:* To evaluate the relationship between hemoglobin A1c (HbA1c) levels and angiographic atherosclerotic burden in non-diabetic individuals.

Materials and methods*:* This retrospective study included adult patients who underwent coronary angiography (CAG) at İzmir Atatürk Training and Research Hospital between 2002 and 2006. All clinical, biochemical, and angiographic data were retrieved from archived hospital records. Individuals with a previous diagnosis of diabetes mellitus or with fasting plasma glucose ≥126 mg/dL were excluded. HbA1c levels, routine biochemical parameters, and classical cardiovascular risk factors were evaluated. The extent and severity of coronary artery disease (CAD) were quantified using the Gensini scoring system, which assigns stenosis-based severity points and multiplies them by segment-specific weighting factors to reflect anatomical importance. Patients were classified according to HbA1c categories and number of involved coronary vessels. Correlations between HbA1c, inflammatory markers, and angiographic severity were analyzed.

Results*:* Higher HbA1c levels were associated with increased Gensini scores and greater angiographic atherosclerotic burden. Individuals with HbA1c ≥6.0% showed significantly elevated fibrinogen and C-reactive protein levels, suggesting an accompanying low-grade inflammatory process. Although overall group comparisons indicated a significant difference in HbA1c levels, post-hoc analyses did not reveal differences between specific vessel-involvement subgroups. HbA1c demonstrated a modest but meaningful relationship with subclinical coronary atherosclerosis, independent of lipid parameters.

Conclusion*:* HbA1c may serve as an early, accessible marker of atherosclerotic risk even in individuals without diabetes. This study provides region-specific evidence supporting the integration of HbA1c into cardiovascular risk-stratification strategies for earlier detection and prevention.

Introduction

Cardiovascular diseases (CVDs) remain one of the leading causes of morbidity and mortality worldwide and represent a major public health burden. The underlying pathophysiological mechanism of these diseases is often ather

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osclerosis, and risk factors such as hyperlipidemia, hypertension, smoking, obesity, and diabetes mellitus (DM) play key roles in its progression [1]. Chronic hyperglycemia in diabetic individuals accelerates atherosclerotic processes through endothelial dysfunction, oxidative stress, and inflammation [2]. However, in recent years, strong evidence has emerged suggesting that similar pathophysiological mechanisms may also be present in individuals without a diagnosis of diabetes [3].

Hemoglobin A1c (HbA1c) is a stable glycoprotein formed by the covalent binding of glucose to hemoglobin during the lifespan of erythrocytes [4]. This biomarker reflects the average blood glucose level over the previous 2–3 months and is accepted as the gold-standard indicator for the diagnosis, treatment monitoring, and risk assessment of DM-related complications [4]. The major advantages of HbA1c are that it is not affected by fasting status or short-term glycemic fluctuations and has high reproducibility across laboratories. Recently, however, HbA1c has been proposed to serve not only as a marker of glycemic control but also as an independent predictor of cardiovascular disease risk [5].

Epidemiological studies in non-diabetic individuals have shown that increasing HbA1c levels are significantly associated with higher prevalence of coronary artery disease (CAD) and with increased Gensini and SYNTAX scores [6]. In the study by Kayali and Özder, even high-normal HbA1c values were correlated with the severity of coronary lesions [5]. Similarly, Abbaszadeh et al. [6] reported that HbA1c was an independent predictor of the presence and extent of CAD in non-diabetic individuals. These findings suggest that HbA1c reflects not only glycemic control but also subclinical atherosclerotic burden [2,3].

Another dimension of the prognostic value of HbA1c is glycemic variability. Even small fluctuations in HbA1c levels have been shown to adversely affect endothelial function, oxidative stress, and vascular inflammation, thereby increasing the risk of major adverse cardiovascular events (MACE) [7,8]. Pei et al. [8], analyzing data from the ACCORD study, demonstrated a significant association between HbA1c variability and MACE, while Shen et al. [8] reported that visit-to-visit changes in HbA1c predicted cardiovascular mortality. These findings indicate that glycemic stability is at least as important as the mean glycemic level. Moreover, elevated HbA1c can contribute to atherosclerotic plaque formation through protein glycosylation, structural damage, increased oxidative stress, and endothelial dysfunction [9].

In light of these findings, HbA1c may provide valuable prognostic information beyond its traditional role in diabetes monitoring. However, there remains a paucity of region-specific data evaluating the relationship between HbA1c and angiographic atherosclerotic burden in non-diabetic individuals within the Turkish population. The present study was therefore designed to explore the association between HbA1c levels and the extent and severity of coronary artery disease using the Gensini scoring system. By focusing specifically on non-diabetic individuals identified retrospectively from hospital records, this study aims to clarify whether HbA1c can serve as an early, accessible, and cost-effective biomarker for atherosclerotic risk assessment independent of classical cardiovascular risk factors.

Materials and Methods

This retrospective study was conducted at the 3rd Internal Medicine Department of İzmir Atatürk Training and Research Hospital between March 15 and June 30, 2006. The study was approved by the institutional ethics and review committee. Data confidentiality and patient privacy were maintained. Adult individuals who had previously undergone coronary angiography for any clinical indication, regardless of whether significant coronary artery disease was detected, and who hadfasting plasma glucose < 126 mg/dL, no history of oral antidiabetic or insulin therapy, and available HbA1c measurement results were included. Exclusion criteria included a history of acute myocardial infarction, chronic kidney disease, liver disease, hematological disorders, or systemic inflammatory conditions. Thus, the study evaluated the relationship between HbA1c levels and the severity of coronary artery disease (CAD) in non-diabetic individuals with normal glycemic profiles.

The study employed a simple random sampling approach from the hospital's angiography registry. The sample size (n=90) was determined based on available non-diabetic patient records meeting inclusion criteria during the study period, which provided a statistical power of approximately 80% to detect moderate correlations (r≥0.35) between HbA1c and Gensini scores at a 95% confidence level.

All laboratory data were obtained retrospectively from archived hospital records. The results of fasting plasma glucose, HbA1c, total cholesterol, HDL, LDL, triglycerides, uric acid, fibrinogen, and CRP had been measured previously as part of routine clinical care during the patients' original hospital admission between 2002 and 2006. HbA1c measurements had originally been performed using high-performance liquid chromatography (HPLC), and other biochemical parameters had been analyzed using enzymatic colorimetric methods according to the institutional Standard Operating Procedures (SOPs) of that period. No new blood samples or laboratory investigations were performed for this study.

All coronary angiography data were obtained retrospectively from archived angiographic reports and digital records. The angiographies had been performed previously by the cardiology department between 2002 and 2006 using the standard Judkins technique as part of routine clinical practice. For the purpose of this study, the existing angiographic images were re-evaluated by two experienced cardiologists who were blinded to the laboratory data.

Coronary artery disease was defined as ≥50% luminal narrowing in any major epicardial artery. The severity and extent of coronary lesions were quantified retrospectively using the Gensini scoring system, and patients were classified into mild, moderate, and severe categories based on their total scores.

Any discrepancies between reviewers were resolved through consensus. No new angiographic procedures were performed for this study. Participants were classified into four HbA1c categories: <5.0%, 5.0–5.49%, 5.50–5.99%, and ≥6.0%, in accordance with the analytical framework presented in Table-4.This stratification allowed a more detailed evaluation of the gradational relationship between HbA1c levels and metabolic or inflammatory parameters.

Additionally, patients were classified according to the number of affected coronary vessels (0–3) to examine the association between HbA1c and angiographic atherosclerotic burden.

All data were analyzed using SPSS (Statistical Package for the Social Sciences) version 26.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as percentages (%). Between-group comparisons were performed using the independent samples t-test and Chi-square test, depending on the data distribution.

Normality of the data was assessed using the Shapiro–Wilk test. For variables that did not meet normal distribution assumptions, the Kruskal–Wallis H test was applied to compare differences among multiple subgroups (e.g., HbA1c quartiles and vessel involvement groups). Post-hoc pairwise comparisons were performed using Dunn–Bonferroni correction to identify which specific groups differed significantly.

The relationship between HbA1c level and Gensini score was evaluated using Pearson's and Spearman's correlation analyses, depending on data distribution.

A p-value < 0.05 was considered statistically significant. Additionally, a graphical representation of the positive correlation between HbA1c and Gensini scores was generated to enhance visual interpretation of the relationship (Figure 1).All analyses were performed in accordance with the reporting standards for observational studies (STROBE guidelines).

Results

A total of 90 patients were included in the study. Of these, 60 were male (66.7%) and 30 were female (33.3%). The mean age was 54.36 ± 11.87 years (28–78), and the mean body mass index (BMI) was 26.25 ± 4.27 kg/m². There was no significant difference in age between male and female patients (male: 54 ± 12, female: 54 ± 11.8 years).

Patients' HbA1c levels were examined in four categories: HbA1c < 5.0% in 22 patients (24.4%), HbA1c between 5.0–5.49% in 24 patients (26.7%), HbA1c between 5.50–5.99% in 32 patients (35.6%), and HbA1c > 5.99% in 12 patients (13.3%).

According to coronary angiography findings, 38 patients (42.2%) had no coronary artery involvement, 27 patients (30%) had single-vessel disease, 13 patients (14.4%) had two-vessel disease, and 12 patients (13.3%) had three-vessel disease (Table-1).

Table-1: *The relationship between the number of affected vessels, mean age, and HbA1c levels of patients*

Vessel Involvement (n)	Number of Patients (n, %)	Mean Age (years)	Mean HbA1c (%)	HbA1c Range (%)
0 vessel (normal)	38 (42.2%)	49.2 ± 10.8	5.45 ± 0.27	4.8 – 5.9
1 vessel	27 (30.0%)	53.5 ± 11.1	5.52 ± 0.32	5.0 – 6.1
2 vessels	13 (14.4%)	57.7 ± 9.9	5.59 ± 0.28	5.1 – 6.2
3 vessels	12 (13.3%)	61.8 ± 8.6	5.64 ± 0.25	5.2 – 6.4
Total (n = 90)	**100%**	**54.36 ± 11.87**	**5.52 ± 0.31**	**4.8 – 6.4**

Table-2 shows that the mean age increased progressively with the extent of vessel involvement. Patients with no affected vessels had the lowest mean, whereas those with three affected vessels had the lowest mean (Table-1). Similarly, the age range shifted toward older ages as the number of involved vessels increased (Table-2). This trend suggests that the severity of vessel involvement increases with age, indicating a possible association between advancing age and the extent of vascular disease.

Table-2: *Mean age according to the number of affected vessels*

	0 Vessel (n=38)	1 Vessel (n=27)	2 Vessels (n=13)	3 Vessels (n=12)	Total (n=90)	p-value
Age (Min–Max)	28–73	36–78	44–70	46–73	28-78	**0.022**
Age (Mean ± SD)	49.2±10.8	53.5±11.1	57.7±9.91	61.8±8.6	54.36±11.87	

No statistically significant differences were detected in fasting glucose, lipid parameters (total cholesterol, LDL, HDL, triglycerides), uric acid or CRP levels across the groups classified by the number of affected coronary vessels ($p > 0.05$ for all) (Table-3). These findings indicate that these classical biochemical parameters did not vary meaningfully with increasing angiographic vessel involvement in this group of patients. Fibrinogen levels differed significantly across groups ($p = 0.018$), with higher values observed among individuals with one or two affected vessels. This suggests a possible association between increased fibrinogen levels and the presence of vascular involvement. A statistically significant overall difference was also observed in mean HbA1c levels across the four vessel-involvement groups ($p = 0.041$). However, post-hoc pairwise comparisons did not show significant differences between individual vessel-involvement groups, indicating that the overall p-value reflected a trend rather than a statistically meaningful separation between specific categories.

Table-3: *Comparison of biochemical parameters according to number of affected vessels*

	Number of Affected Vessels				p
	0	1	2	3	
FPG (mg/dL)	87.61 ± 14.07	92.41 ± 10.06	88.46 ± 13.38	94.33 ± 16.89	0.401
Total Cholesterol	184.58 ± 48.45	197.56±47.77	196.07 ± 35.37	205.58 ± 47.62	0.474
HDL (mg/dL)	43.26 ± 13.32	43.29 ± 14.01	40.77 ± 12.11	44.08 ± 8.89	0.526
LDL (mg/dL)	113.82 ± 36.69	120.70 ± 41.10	124.38 ± 29.66	131.50 ± 38.60	0.429
Triglycerides (mg/dL)	131.86 ± 72.47	164.26 ± 81.58	155.85 ± 68.91	152.92 ± 54.91	0.221
Uric Acid (mg/dL)	5.64 ± 1.67	7.09 ± 2.77	6.33 ± 1.75	11.60 ± 16.71	0.152
Fibrinogen (mg/dL)	335.82 ± 78.52	393.07 ± 79.33	391.31 ±118.79	379.92 ± 111.69	0.018*
CRP (mg/L)	9.59 ± 17.68	17.49 ± 24.19	18.74 ± 21.79	7.60 ± 9.11	0.255
HbA1c (%)	5.45 ± 0.27	5.52 ± 0.32	5.59 ± 0.28	5.64 ± 0.25	0.041*

Kruskal–Wallis H Test, FPG: Fasting plasma glucose, HDL: High density lipoprotein, LDL: Low density lipoprotein, CRP: C reactive protein

Notably, despite the lack of distinct group-wise differences, a positive correlation was found between mean HbA1c levels and Gensini scores (r = 0.46, p<0.001) (Figure-1), indicating thathigher HbA1c values were associated with increased atherosclerotic burden, even in non-diabetic individuals.

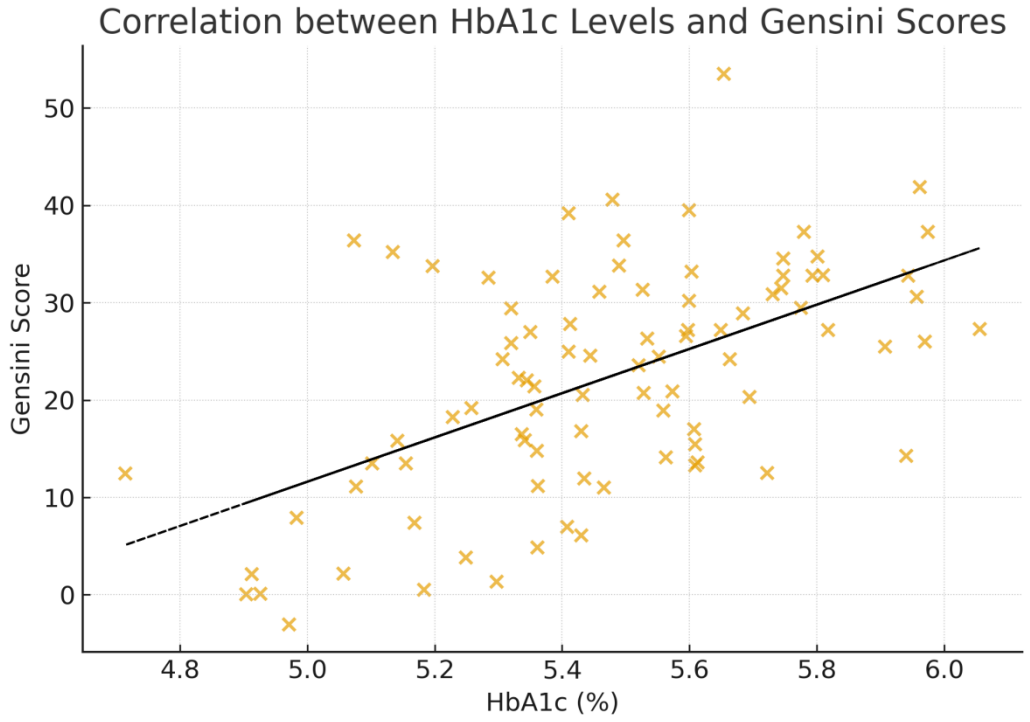

Figure-1: *Correlation between HbA1c Levels and Gensini Scores*

Table-4 shows the mean laboratory values of patients across different HbA1c groups, revealing that most metabolic parameters—including fasting plasma glucose, lipid profile components (total cholesterol, HDL, LDL, triglycerides), and uric acid - did not differ significantly among the groups ($p > 0.05$). This suggests that variations in HbA1c levels were not strongly associated with these markers within the study population. Fibrinogen levels, although not statistically significant ($p = 0.449$), showed a slight upward trend in some subgroups, particularly in patients with HbA1c values between 5.50–5.99 and ≥6%, who exhibited higher mean fibrinogen levels compared to the lower HbA1c categories. This suggests a possible tendency toward increased pro-inflammatory or pro-thrombotic activity with rising HbA1c, even though the differences did not reach statistical significance. However, CRP demonstrated a statistically significant difference across the HbA1c categories ($p = 0.037$), with individuals in the highest HbA1c group (≥6%) exhibiting notably elevated CRP levels compared to the other groups. This finding indicates that higher HbA1c levels may be linked to increased systemic inflammation, even when other metabolic parameters remain relatively comparable.

Table-4: *Mean laboratory values of patients according to HbA1c groups*

	HbA1c				**P**
	<5 (n=22)	**5.0–5.49 (n=24)**	**5.50–5.99 (n=32)**	**≥6 (n=12)**	
FPG (mg/dL)	89.90 ± 15.76	87.38 ± 13.92	92.13 ± 11.12	90.25 ± 13.66	0.664
Total Cholesterol(mg/dl)	197.86 ± 43.40	194.13 ± 56.16	185.69 ± 44.31	200.83 ± 37.50	0.569
HDL (mg/dL)	46.23 ± 11.19	39.75 ± 11.41	43.00 ± 15.04	43.75 ± 10.68	0.102
LDL (mg/dL)	126.91 ± 40.66	120.17 ± 34.34	115.56 ± 41.38	117.08 ± 25.75	0.618
Triglycerides (mg/dL)	129.00 ± 42.52	149.92 ± 82.11	139.31 ± 63.27	201.08 ± 101.91	0.187
Uric Acid (mg/dL)	6.33 ± 1.42	6.11 ± 2.64	8.46 ± 10.46	5.88 ± 1.70	0.502
Fibrinogen (mg/dL)	359.86 ± 95.52	354.79 ± 99.38	378.22 ± 80.51	373.75 ± 110.51	0.449
CRP (mg/L)	9.19 ± 9.59	9.77 ± 19.95	10.92 ± 14.90	32.13 ± 33.16	0.037*

FPG: Fasting plasma glucose, HDL: High density lipoprotein, LDL: Low density lipoprotein, CRP: C reactive protein

These findings strengthen the evidence that HbA1c reflects not only long-term glycemic exposure but also subclinical inflammatory activation, supporting its role as a multifactorial biomarker in cardiovascular risk assessment.

Discussion

In this study, the relationship between HbA1c levels and the severity of coronary artery disease (CAD) was evaluated in non-diabetic individuals, and it was shown that HbA1c was associated with angiographic disease burden independently of classical risk factors. Our findings suggest that HbA1c may not only reflect glycemic control but also serve as an indicator of vascular glycation and endothelial damage [10].

In recent years, large population-based studies have demonstrated that even in non-diabetic individuals, increasing HbA1c levels are significantly associated with a higher incidence of cardiovascular events [11]. Therefore, it has been proposed that HbA1c measurement could be used not only for diabetes diagnosis but also for cardiovascular risk stratification [12]. Particularly, HbA1c levels within the prediabetic range (5.7–6.4%) have been reported to be associated with subclinical atherosclerosis, endothelial dysfunction, and coronary plaque burden [13].

In this study the positive correlation between HbA1c levels and Gensini scores was consistent with numerous findings in the literature. Koushki et al. [14] reported a strong correlation between HbA1c and SYNTAX score in non-diabetic patients

with ST-elevation myocardial infarction (STEMI). Similarly, Jiao et al.[15], in their systematic review, emphasized that HbA1c is an independent marker for predicting coronary lesion severity and prognosis.

Furthermore, it has been demonstrated that HbA1c variability shows a stronger association with cardiovascular events compared to single measurements. Gough et al. [16] reported that intra-individual HbA1c variation significantly increased the risk of major adverse cardiovascular events (MACE). This finding indicates that HbA1c reflects not only average glycemic status but also long-term metabolic instability [17].

The significant increase in CRP and fibrinogen levels with rising HbA1c values in this study supports the connection between HbA1c and inflammation. Chia et al. [18] reported that inflammatory markers, particularly high-sensitivity CRP, play a major role in the atherosclerotic process and that elevated HbA1c levels parallel this inflammatory activity. Therefore, HbA1c can be considered a comprehensive biomarker representing both metabolic dysfunction and subclinical inflammation [19].

This study suggests that HbA1c may be useful in the early detection of CAD severity. Planning early intervention strategies in individuals with elevated HbA1c but without diabetes may contribute to preventing long-term cardiovascular complications. However, the retrospective design and single-center setting of the study constitute important limitations.Larger, prospective, multicenter studies are needed to confirm this relationship [19].

Conclusion

This study demonstrated that HbA1c levels are significantly associated with the severity of coronary artery disease in non-diabetic individuals. The increase in Gensini scores and the parallel rise in inflammatory markers with higher HbA1c values suggest that this parameter may serve not only as an indicator of glycemic control but also as an indirect reflection of atherosclerotic burden. These findings support the notion that HbA1c could be a simple, reproducible, and cost-effective biomarker for early cardiovascular risk detection in the non-diabetic population. Future large-scale prospective studies evaluating HbA1c alongside other inflammatory and metabolic markers will help clarify its role in cardiovascular risk stratification.

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Authors' contributions

SG- Conceptualization of the study, design of the methodology, data collection, statistical analysis and manuscript drafting. MY- Cardiological supervision, interpretation of angiographic findings, critical review of the manuscript, and final approval of the version to be published. BS- Data validation, literature review, and contribution to the interpretation of results and manuscript editing.

Conflict of interest

The authors declare that they have no financial, personal, or institutional conflicts of interest that could have influenced the preparation or outcomes of this study.

No part of this work has been published or is under consideration elsewhere.

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