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The role of hemoglobin A1c as a predictor of major adverse cardiovascular events in patients with type 2 diabetes mellitus after percutaneous coronary intervention: a case-cohort study

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Abstract

Background Few studies have investigated the potential predictive value of glycosylated hemoglobin A (HbA1c) and clinical outcomes in diabetic patients after percutaneous coronary intervention (PCI), and the results of the reports have often been inconclusive and contradictory. We have organized a study to investigate the relationship between HbA1c and the occurrence of major adverse cardiovascular events (MACE) in diabetic patients after PCI.

Methods This case-cohort study was conducted on 563 diabetic patients who underwent PCI. All studied patients had an HbA1c level measured within 24 h before angioplasty. All patients were followed for six months regarding the occurrence of MACE, and the HbA1c level was measured again at the end of the sixth month of follow-up. In the case of MACE, the subjects were considered the case group, and other non-MACE patients were included in the control group.

Results 505 patients remained in the study at the end of follow-up. MACE occurred in 23 (4.6%) patients during the first month and in 57 (11.3%) patients by the end of the sixth month. Baseline HbA1c was an independent predictor of MACE and mortality at the end of month-6 ($P=0.008$ and 0.001, respectively).

Conclusions The level of HbA1c at the time of admission has a significant predictive value for the occurrence of MACE in diabetic patients who undergo PCI. However, post-PCI glycemic control may not effectively reduce the risk of MACE in this population.

Clinical trial registration Not applicable.

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Highlights

- Few studies have investigated the impact of HbA1c and MACE in diabetic patients after PCI, and the reports' results have often been inconclusive and contradictory.
- Baseline HbA1c was an independent predictor of MACE and mortality at the end of month-6 but not for MACE at the end of month-1.
- No significant correlation was observed between sixth-month HbA1c and MACE at the end of month-6.

Keywords Major adverse cardiovascular events, MACE, Percutaneous coronary intervention, Glycated hemoglobin, HbA1c, Diabetes mellitus, Mortality, Acute coronary syndrome

Introduction

Coronary artery diseases (CAD) represent the leading cause of mortality in cardiovascular pathologies. Among the various revascularization techniques employed by clinicians to manage CAD, percutaneous coronary intervention (PCI) has gained significant popularity. However, it is essential to note that patients undergoing this procedure may encounter major adverse cardiovascular events (MACE), contributing significantly to morbidity and mortality rates within this population [1, 2].

Type 2 diabetes mellitus (T2DM) is a prevalent chronic condition that serves as an independent and substantial risk factor for CAD [3]. Individuals with T2DM exhibit an increased susceptibility to MACE [4]. Hemoglobin A1c (HbA1c) is a marker for average blood sugar (BS) levels over 8–12 weeks. Several studies have investigated the prognostic value of HbA1c in relation to MACE, establishing a positive correlation between higher HbA1c levels and elevated rates of MACE [4, 5]. However, research on the impact of blood sugar control on post-PCI MACE occurrences in this specific population is limited, and the findings thus far have been controversial.

Numerous studies have demonstrated a correlation between elevated blood sugar levels upon admission and short-term mortality rates in T2DM patients following myocardial infarction (MI) [6]. Nevertheless, due to the release of catecholamines during the acute phase of CAD, blood sugar measurements may not provide reliable prognostic information. Consequently, HbA1c emerges as a more suitable indicator for long-term blood sugar monitoring.

Only a limited number of studies have examined the association between baseline HbA1c levels and MACE in T2DM patients after undergoing PCI, and the results have yielded conflicting outcomes [7, 8]. Moreover, only some studies have followed patients longitudinally to investigate the relationship between subsequent HbA1c levels and MACE. Furthermore, the efficacy of blood sugar control in preventing MACE following PCI in T2DM patients has yet to be adequately explored. Therefore, the objective of this study is to investigate the correlation between HbA1c levels, both upon admission (HbA1c-0) and six months (HbA1c-6) after PCI, and the occurrence of MACE and mortality, both at the end of month-1 and month-6 post-PCI, in T2DM patients undergoing PCI.

Methods

Population and data collection

This longitudinal case-cohort study was conducted on all T2DM patients undergoing PCI in a tertiary cardiac center from December 2020 to April 2022 using a census sampling method which resulted in 563 total participants). All PCI candidates with T2DM were enrolled. Patients with a history of valvular heart disease, patients with a projected lifespan of fewer than six months, patients without proper post-PCI antiplatelet medications, and patients with a history of previous coronary artery bypass graft (CABG) due to dissimilarities in the pathophysiology of restenosis between graft veins and native arteries, were excluded.

Patient demographic information, including age, gender, drug history, social history, past medical history, electrocardiographic, and echocardiographic data, were recorded at admission. Blood samples were obtained 24 h before PCI, and HbA1c levels were measured. Patients were visited after one month to assess for MACE (including mortality, MI, heart failure (HF), cerebrovascular accidents (CVA), hospitalization due to acute coronary syndrome (ACS), and revascularization after a successful PCI, including target vessel revascularization (TVR) and target lesion revascularization (TLR)) as a short-term endpoint. Patients were revisited six months after PCI and again evaluated for MACE and HbA1c levels. All patients were on dual antiplatelet therapy and high-dose statins during the follow-up period. Finally, at each stage, the case group consisted of MACE patients, and the control group consisted of non-MACE patients. Then, the groups were compared based on their HbA1c levels.

Statistics

Quantitative characteristics were presented using the mean (±standard deviation), while qualitative characteristics were represented by the number (percentage). A t-test (or the Mann-Whitney test if required) was employed to compare quantitative traits between the two groups, whereas a Chi-square test (or Fisher's exact test

if necessary) was used for qualitative traits. Odds ratios (OR) and their corresponding 95% confidence intervals (CI) were calculated individually for high HbA1c levels (HbA1c≥6.5). A P-value less than 0.05 was considered statistically significant.

Declaration of generative AI in scientific writing

The authors disclose that AI and AI-assisted technologies were not used in the writing process of this manuscript.

Results

A total of 563 patients were enrolled, of which 58 patients were excluded during the follow-up period (56 patients due to loss of follow-up and 2 patients due to improper use of antiplatelet medications). Finally, 505 patients (290 male and 215 female) were included in the analysis. Figure 1 demonstrates an overview flowchart of the study process, and Table 1 summarizes the patient's baseline characteristics. During the 6 month follow-up, 23 (4.6%) patients had MACE at the end of month-1 (including 16 mortality, 2 TLR, 2 CABG, 2 HF, and 1 ACS), and at the end of month-6, a total of 57 (11.3%) patients had MACE (including 22 mortality, 3 TLR, 6 TVR, 18 ACS, 3 HF, 4 CABG, and 1 CVA) (Fig. 2).

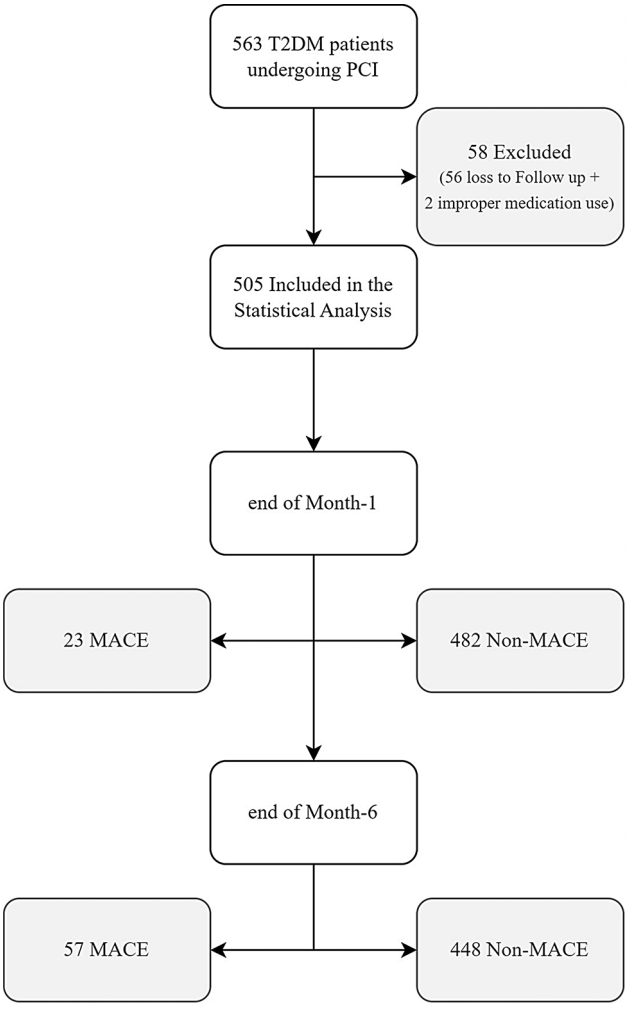

Fig. 1 Study flowchart

Correlation of baseline HbA1c with MACE at the end of month-1 and −6

Correlation of baseline HbA1c with MACE at the end of month-1

Univariate analysis showed that at the end of month-1, mean HbA1c-0 (24 h before PCI) was significantly higher in MACE compared to non-MACE patients (9.29±2.65 vs. 8.14±1.96, $P=0.008$). Additionally, LVEF, clinical presentation, number of VD, and lesion location also significantly differed between MACE and non-MACE patients at the end of month-1 ($P<0.05$) (Table 1). Although HbA1c-0 was significantly higher in MACE patients, the multivariate logistic regression models showed no significant predictive value in HbA1c-0 for MACE at the end of month-1 ($P>0.05$); however, significant correlations were observed in gender, LVEF, number VD, and lesion location with MACE at the end of month-1 ($P<0.05$) (Table 2).

Correlation of baseline HbA1c with MACE at the end of month-6

Univariate analysis showed that at the end of month-6, mean HbA1c-0 was significantly higher in MACE compared to non-MACE patients (9.61±2.36 vs. 8.08±1.93, $P<0.001$). Additionally, age, serum creatinine, HbA1c-6, LVEF, clinical presentation, lesion location, and number of VD also significantly differed between MACE and non-MACE patients at the end of month-6 ($P<0.05$) (Table 1). Multivariate regression models also showed that HbA1c-0 could be an independent predictor of MACE at the end of month-6 (OR: 1.192; 95%CI: 1.046–1.357; $P=0.008$) (Table 2).

Correlation of the sixth month HbA1c with MACE at the end of month 6

Univariate analysis showed that at the end of month-6, mean HbA1c-6 (six months after PCI) was significantly higher in MACE compared to non-MACE patients (8.6±1.95 vs. 7.91±1.78, $P=0.03$). Additionally, age, serum creatinine, HbA1c-0, LVEF, clinical presentation, lesion location, and number of VD also significantly differed between MACE and non-MACE patients at the end of month 6 ($P<0.05$) (Table 1). However, our multivariate regression models did not show any statistically significant predictive value for HbA1c-6 in predicting MACE at the end of month-6 (OR: 1.175; 95%CI: 0.984–1.403; $P=0.07$) (Table 2).

Table 1 Baseline characteristics of patients based on MACE at the end of month-1 and month-6

Variable	Total	Month-1			Month-6		
		MACE (*n* = 23)	Non-MACE (*n* = 482)	*P*-value	MACE (*n* = 57)	Non-MACE (*n* = 448)	*P*-value
Age	60.44 ± 9.7	63.35 ± 11.18	60.3 ± 9.61	0.14	63.4 ± 10.43	60.11 ± 9.56	0.03
Gender				0.07			0.61
Male	290 (57.4%)	9 (39.1%)	281 (58.4%)		31 (54.4%)	259 (57.9%)	
Female	214 (42.6%)	14 (60.9%)	200 (41.6%)		26 (45.6%)	188 (42.1%)	
HTN	296 (58.6%)	11 (47.8%)	285 (59.1%)	0.28	31 (54.4%)	265 (59.2%)	0.49
DLP	191 (37.8%)	10 (43.5%)	181 (37.6%)	0.57	24 (42.1%)	167 (37.3%)	0.48
Prior MI	18 (3.6%)	2 (8.7%)	16 (3.3%)	0.17	2 (3.5%)	16 (3.6%)	0.98
Smoking	94 (18.6%)	4 (17.4%)	90 (18.7%)	0.87	12 (21.1%)	82 (18.3%)	0.62
FBS	210.22 ± 89.19	240.74 ± 86.41	208.77 ± 89.15	0.09	230.89 ± 83.36	207.59 ± 89.65	0.06
Cr	1.11 ± 052	1.48 ± 0.9	1.09 ± 0.49	0.05	1.25 ± 0.62	1.09 ± 0.5	0.02
TG	187.34 ± 121.28	171.43 ± 107.92	188.15 ± 121.97	0.52	183.98 ± 123	187.77 ± 121.2	0.83
Chol	158.29 ± 45.93	163.91 ± 41.32	158 ± 46.18	0.55	161.15 ± 45.57	157.91 ± 46.02	0.62
LDL	92.42 ± 33.36	98.78 ± 36.92	92.09 ± 33.18	0.35	96.58 ± 33.52	91.87 ± 33.51	0.33
HDL	42.67 ± 15.73	42.66 ± 16.45	42.66 ± 15.71	0.98	41.58 ± 13.28	42.81 ± 16.03	0.59
HbA1c-0	8.19 ± 2.01	9.29 ± 2.65	8.14 ± 1.96	0.008	9.61 ± 2.36	8.08 ± 1.93	< 0.001
HbA1c-6	7.96 ± 1.8				8.6 ± 1.95	7.91 ± 1.78	0.03
LVEF	44.6 ± 10.29	32.83 ± 10.53	45.17 ± 9.95	< 0.001	38.25 ± 11.71	45.41 ± 9.82	< 0.001
Clinical presentation				< 0.001			0.001
CSA	178 (35.2%)	1 (4.3%)	177 (36.9%)		9 (15.8%)	169 (37.7%)	
UA	85 (16.8%)	1 (4.3%)	84 (17.4%)		7 (12.3%)	78 (17.4%)	
NSTEMI	38 (7.5%)	3 (13%)	35 (7.3%)		7 (12.3%)	31 (6.9%)	
STEMI	204 (40.4%)	18 (78.3%)	185 (38.5%)		34 (59.6%)	170 (37.9%)	
PCI data							
Number VD	2.04 ± 0.83	2.52 ± 0.66	2.01 ± 0.83	0.004	2.35 ± 0.79	2 ± 0.82	0.001
Culprit vessel				0.65			0.4
LMA	11 (2.2%)	0 (0%)	11 (2.3%)		2 (3.6%)	9 (2.1%)	
LAD	260 (51.5%)	14 (60.9%)	246 (51%)		34 (59.6%)	226 (50.3%)	
LCX	105 (20.8%)	3 (13%)	102 (21.2%)		8 (14%)	97 (21.7%)	
RCA	129 (25.5%)	6 (26.1%)	123 (25.5%)		13 (22.8%)	116 (25.9%)	
Lesion location				0.02			0.001
Proximal	195 (38.6%)	16 (69.6%)	179 (37.2%)		30 (52.6%)	165 (36.9%)	
Middle	273 (54.1%)	7 (30.4%)	266 (55.3%)		24 (42.1%)	249 (55.7%)	
Distal	29 (5.7%)	0 (0%)	29 (6%)		0 (0%)	29 (6.5%)	
Bifurcation	7 (1.4%)	0 (0%)	7 (1.5%)		3 (5.3%)	4 (0.9%)	
Balloon	469 (92.9%)	23 (100%)	446 (92.5%)	0.17	53 (93%)	416 (92.9%)	0.97
Stent number	1.31 ± 0.52	1.22 ± 0.42	1.31 ± 0.52	0.39	1.3 ± 0.53	1.31 ± 0.52	0.87

PMH: Previous Medical History, HTN: Hypertension, DLP: Dyslipidemia, FBS: Fasting Blood Sugar, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, TG: Triglyceride, LVEF: Left Ventricular Ejection Fraction, SA: Stable Angina, UA: Unstable Angina, STEMI: ST-segment Elevation MI, NSTEMI: Non STEMI, VD: Vessel Disease, LMA: Left Main Artery, LAD: Left Anterior Descending artery, LCX: Left Circumflex artery, RCA: Right Coronary Artery

Correlation of baseline HbA1c with mortality at the end of month-1 and −6

Correlation of baseline HbA1c with mortality at the end of month-1

Univariate analysis showed that at the end of month-1, mean HbA1c-0 was significantly higher in deceased patients compared to living patients (9.63±2.64 vs. 8.15±1.97, P=0.004). Additionally, age (68.25±9.01 vs. 60.19±9.62, P=0.001), serum creatinine (1.69±1 vs. 1.09±0.49, P=0.03), LVEF (31.25±9.75 vs. 45.04±10.02, P<0.001), clinical presentation (P=0.003), and number VD (2.5±0.63 vs. 2.02±0.83, P=0.02) also significantly differed between MACE and non-MACE patients at the end of month-1. Regression models also demonstrated that HbA1c-0 could independently predict mortality at the end of month-1 (OR: 1.373; 95%CI: 1.097–1.719; P=0.006) (Table 2).

Correlation of baseline HbA1c with mortality at the end of month-6

Univariate analysis showed that at the end of month-6, mean HbA1c-0 was significantly higher in deceased compared to living patients (9.71±2.53 vs. 8.13±1.96, P<0.001). Additionally, age (66.27±9.76 vs. 60.18±9.63,

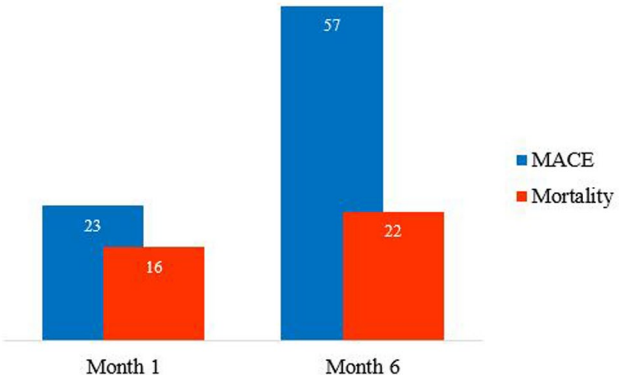

Fig. 2 MACE and mortality at the end of each time point

Table 2 Results of the regression analysis with adjustment for potential confounders

Variable	OR (95%CI)	*P*-value
MACE at the end of month-1 based on HbA1c-0		
Gender	2.685 (1.018–7.081)	0.04
LVEF	0.961 (0.89–0.965)	< 0.001
Clinical presentation	3.917 (0.49-31.306)	0.2
Number VD	1.922 (1.022–9.349)	0.04
Lesion location	3.401 (1.237–9.349)	0.02
HbA1c-0	1.121 (0.931–1.35)	0.23
MACE at the end of month-6 based on HbA1c-0		
LVEF	0.958 (0.933–0.985)	0.002
Clinical presentation	1.978 (0.828–4.722)	0.13
Number VD	1.603 (1.091–2.355)	0.02
HbA1c-0	1.192 (1.046–1.357)	0.008
MACE at the end of month-6 based on HbA1c-6		
Clinical presentation	2.074 (0.829–5.193)	0.12
Number VD	1.61 (1.02–2.541)	0.04
HbA1c-6	1.175 (0.984–1.403)	0.07
Mortality at the end of month-1 based on HbA1c-0		
Age	1.089 (1.018–1.166)	0.01
Cr	2.207 (1.241–3.925)	0.007
LVEF	0.934 (0.889–0.982)	0.007
Lesion location (proximal)	3.933 (1.087–14.235)	0.04
HbA1c-0	1.373 (1.097–1.719)	0.006
Mortality at the end of month-6 based on HbA1c-0		
Age	1.07 (1.02–1.13)	0.1
Cr	1.97 (1.17–3.3)	0.1
LDL	1.01 (1-1.03)	0.05
LVEF	0.958 (0.932–0.984)	0.002
Lesion location (proximal)	2.56 (0.93–3.71)	0.07
HbA1c-0	1.38 (1.14–1.17)	0.001

$P=0.004$), serum creatinine (1.55±0.9 vs. 1.09±0.49, $P=0.03$), LVEF (30.91±8.82 vs. 45.23±9.91, $P<0.001$), clinical presentation ($P=0.001$), lesion location ($P=0.03$), and number VD (2.41±0.73 vs. 2.02±0.83, $P=0.03$) also significantly differed between patients with and without mortality at the end of month-6. Our multivariate regression models also showed that HbA1c-0 may be an independent predictor of mortality at the end of month-6 (OR: 1.38; 95%CI: 1.14–1.17; $P=0.001$) (Table 2).

Discussion

The primary objective of this investigation was to examine the association between levels of HbA1c during admission and six months after PCI and the incidence of MACE and mortality at both one month and six months following PCI in patients with T2DM who underwent PCI. Our findings revealed that HbA1c-0 did not correlate significantly with MACE at the end of month-1; however, this correlation was significant at the end of month-6. Additionally, our results did not demonstrate any significant correlation between HbA1c-6 and MACE at the end of month-6. Regarding mortality, our findings showed a statistically significant correlation between baseline HbA1c and mortality at the end of both month-1 and month-6.

In the present study, HbA1c-0 did not exhibit a significant correlation with MACE at the end of month-1; however, this correlation was significant at the end of month-6. This discrepancy may arise from the inadequate time frame for the manifestation of MACE components, such as revascularization and readmission, within one month. Immediately after the placement of a stent during PCI, mechanical injury to the endothelium prompts the deposition of a platelet and fibrin layer at the site of damage [9, 10]. However, restenosis at the stent site occurs with a delay, gradually developing over several months due to neointimal hyperplasia [11].

A high baseline HbA1c level signifies prolonged inadequate blood glucose control, and chronic hyperglycemia induces damage to the vascular endothelium [12, 13]. Consequently, inflammatory cytokines increase, causing vasomotor activity dysfunction and augmenting extracellular matrix production and cell proliferation [12], which can ultimately lead to vessel narrowing. This pathological observation explains the relationship between baseline HbA1c and MACE at the end of month-6.

Our findings indicated a significant correlation between baseline HbA1c and mortality at the end of month-1 but not between baseline HbA1c and MACE at the end of month-1. This can be explained by understanding that mortality is one component of MACE and can be attributed to stent thrombus, coronary dissection, and electrical disturbances occurring during or immediately after PCI. Conversely, other components of MACE, such as revascularization and readmission, arise from the gradual occurrence of restenosis over six months. The early mortality risk could also be influenced by the prothrombotic and arrhythmogenic effects of hyperglycemia, which amplify cardiovascular vulnerability immediately following PCI [14, 15]. Hyperglycemia promotes oxidative stress via increased Reactive Oxygen Species (ROS)

production, leading to endothelial damage, impaired vasodilation, and increased inflammatory cytokines like IL-6 and TNF-α, which can worsen endothelial dysfunction and promote plaque instability [16, 17]. Advanced glycation end products (AGEs) then form due to prolonged hyperglycemia and lead to stiffening of blood vessels by cross-linking with collagen and other extracellular matrix proteins, smooth muscle proliferation, vascular fibrosis, and reduced elasticity [18]. Also, Hyperglycemia is linked with hyperactivity and aggregation of platelets, and increased fibrinogen levels and plasminogen activator inhibitor-1, which promotes thrombosis [19]. This prothrombotic tendency can contribute to MACE and mortality, particularly in the early post-PCI period.

Furthermore, our study findings revealed no significant association between HbA1c-6 and MACE at the end of month-6. This lack of correlation can be elucidated by the concept of metabolic memory and the gradual reduction of glycation end products over time [7, 20]. These findings suggest that maintaining adequate glycemic control in individuals with T2DM prior to PCI, at least in the setting of elective PCIs, is crucial to preventing the development of MACE. This approach promotes optimal vascular health and improves the condition of blood vessels prior to the procedure.

The results of the present study were consistent with the findings of Ike et al. [7]. They retrospectively evaluated the clinical outcomes in 546 T2DM patients undergoing elective PCI according to their glycemic status before and after PCI. In a 300-day follow-up period, MACE was significantly lower in patients with a baseline HbA1c of less than 6.9 compared to over 6.9, and clinical outcomes were better in patients with proper glycemic control before PCI. However, the multivariate analysis showed no significant relationship between MACE and any of the follow-up HbA1c variables (secondary HbA1c at any time in the follow-up period and the difference between primary and secondary HbA1c). In other words, their findings showed that glycemic control after PCI was not correlated with improved outcomes.

Kasaeeaan et al. [21] prospectively followed 2884 patients, including both diabetic and non-diabetic patients, after PCI. Similar to our findings, they concluded that proper glycemic control significantly correlates with improved clinical outcomes in post-PCI patients with T2DM in such a way that the difference in the risk of MACE between non-diabetics and good glycemic control diabetics was not significant. Similarly, Lao et al. [1] found that in postmenopausal patients with poor prior glycemic control (HgA1c>7%), MACE is more probable and that HbA1c was still an independent MACE risk predictor after adjustment for potential confounders. Ueda et al. [22] also found that HbA1c is an independent risk predictor of MACE in patients with T2DM, with a hazard ratio of 1.40. Consistent with our findings, Pusuroglu et al. [4] demonstrated that HbA1c is significantly correlated with long-term MACE but not with short-term MACE, and Tian et al. [5] showed that HbA1c is not an appropriate prognostic factor for short term MACE in patients undergoing primary PCI. Some studies, such as Çiçek et al. [23], followed up patients for a more extended period compared to similar studies. Çiçek et al. [23] also found a statistically significant association between HgA1c and two-year MACE.

In addition, Baber et al. [24] found that both very tight glycemic control (HgA1c≤5.5%) and poor glycemic control (HbA1c≥8) prior to the PCI procedure could also lead to higher rates of MACE. However, a Bayesian analysis study by Shao et al. [25] found that a tighter glycemic control (HgA1c goal of <6%) leads to a better MACE risk reduction compared to a moderate glycemic control (HgA1c goal of 7–8%). In the present study, we did not evaluate the effect of different glycemic control intensities on MACE.

Some studies' results were in contrast with the present study. Lemesle et al. [8] retrospectively evaluated 952 diabetic patients, and Singla et al. [26] prospectively evaluated 231 people with diabetes, and both found that HgA1c is not a predictor of long-term MACE after a successful PCI in T2DM patients and that glycemic control does not affect long-term clinical outcomes. The discrepancies between our findings and Lamesle et al. [8] study may be due to selection bias and the differences in the definition of MACE used. Lamesle et al. [8] study was conducted in a referral center, making a selection bias probable. Also, their study defined MACE only by mortality, MI, or target vessel revascularization in the previous vessel. Additionally, different populations and sample sizes could explain the discrepancies between our findings and the Singla et al. [26] study. Singla et al. [26] conducted their research only on diabetic patients with acute MI undergoing primary PCI; however, in the present study, we included all patients with ACS and CCS undergoing either elective or emergent PCI. Additionally, Hwang et al. [27] measured HbA1c two years after PCI and showed a statistically significant relationship between lower second-year HbA1c levels and the incidence of MACE. However, our findings showed that the HbA1c level measured during the follow-up period after PCI (HbA1c-6) was not correlated with MACE. This could be explained by the more extended follow-up period in the Hwang et al. [27] study (five years) compared to ours (six months). Thus, the authors suggest that future research be conducted in multiple centers with a more extended follow-up period.

Additionally, the findings of this study should be interpreted in light of several limitations. First, as the study was conducted in a single tertiary cardiac center, its

generalizability may be limited. Patient demographics, comorbidities, clinical practices, and treatment protocols can vary across different institutions, regions, and healthcare systems. Second, although key confounders were addressed, other influential factors impacting MACE and mortality, such as diabetes duration, renal function, insulin resistance, lipid profiles, and medication adherence, were not comprehensively controlled for in the analysis.

Conclusion

HbA1c on admission appears to be associated with long-term MACE and mortality, as well as short-term mortality. However, it does not independently predict short-term MACE, despite being elevated in patients with MACE at the end of month-1. These findings suggest that maintaining adequate glycemic control before cardiac interventions may play a role in reducing the risk of future adverse events.

Abbreviations

CAD	Coronary Artery Diseases
PCI	Percutaneous Coronary Intervention
MACE	Major Adverse Cardiovascular Events
T2DM	Type 2 Diabetes Mellitus
HbA1c	Hemoglobin A1c
BS	Blood Sugar
MI	Myocardial Infarction
CABG	Coronary Artery Bypass Graft
CI	Confidence Interval
OR	Odds Ratio
ACS	Acute Coronary Syndrome

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Author contributions
Conceptualization: Babak Bagheri, Rozita Jalalian, Farima Sadat Mousavi Kiasari; Data curation: Farima Sadat Mousavi Kiasari, Fatemeh Mousavi, Soheil Azizi; Formal analysis: Abbas Alipour; Methodology: Babak Bagheri, Farima Sadat Mousavi, Erfan Ghadirzadeh; Project administration: Babak Bagheri, Farima Sadat Mousavi, Erfan Ghadirzadeh; Resources: Babak Bagheri, Rozita Jalalian, Soheil Azizi; Software: Abbas Alipour, Erfan Ghadirzadeh; Supervision: Babak Bagheri, Farima Sadat Mousavi; Validation: Babak Bagheri, Farima Sadat Mousavi; Visualization: Erfan Ghadirzadeh; Writing–original draft: Erfan Ghadirzadeh, Babak Bagheri, Farima Sadat Mousavi; Writing–review & editing: All authors.

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Data availability
The data are available upon reasonable request from the corresponding author.

Declarations

Ethics approval and consent to participate
This study was conducted without commercial input or involvement in the design, implementation, analysis, or reporting. This study was approved by the Research Ethics Committee of Mazandaran University of Medical Sciences (Ethics Approval Code: **IR.MAZUMS.REC.1399.953**). All procedures performed in this study were in accordance with the ethical standards of the Institutional Research Ethics Committee of Mazandaran University of Medical Sciences and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Written informed consent was obtained from all participants before entering the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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