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ORIGINAL RESEARCH

Relationship Between Glycosylated Hemoglobin Concentration and Prognosis of Acute ST-Segment Elevation Myocardial Infarction in Non-Diabetic Patients

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Objective: To investigate the relationship between glycosylated hemoglobin (HbA1c) concentration at admission and the prognosis of acute ST-segment elevation myocardial infarction (STEMI) in non-diabetic patients.

Methods: A total of 142 non-diabetic patients with acute STEMI who underwent emergency percutaneous coronary intervention (PCI) in our hospital between January 2023 and December 2024 were enrolled in this study. HbA1c levels and baseline data for all patients were obtained at admission. According to the HbA1c level, patients were divided into a normal blood glucose level group (group A, HbA1c & lt; 5.7%, N = 57) and a pre-diabetic group (group B, 5.7% ≤ HbA1c ≤ 6.4%, N = 85). The occurrence of major cardiovascular events (MACE) was compared between the two groups after PCI during one-year follow-up.

Results: The levels of serum FPG, BNP and the number of patients with multivessel diseases in group B were higher than those in group A (all $p < 0.05$). The LVEF in group B was significantly lower than in Group A (P = 0. 017). The total incidence of major cardiovascular events (MACE) within 1 year after PCI was significantly higher in group B than in group A (P = 0. 047). In addition, the risk of MACE in group B was 4.98 times higher than that in group A.

Conclusion: HbA1c can be used as an independent predictor of MACE in non-diabetic STEMI patients. The control of blood glucose levels in pre-diabetic patients with STEMI should be given high emphasis to improve prognosis after PCI.

Keywords: prediabetic lesions, glycosylated hemoglobin, acute ST-segment elevation myocardial infarction, major cardiovascular events

Introduction

Cardiovascular diseases (CVDs) are among the leading causes of morbidity and mortality worldwide and often present clinically as acute myocardial infarction (AMI) for the first time.[1,2] ST-segment elevation myocardial infarction (STEMI) accounts for approximately 40% of all AMI cases despite a relative decrease.[3] STEMI mortality has declined owing to advances in percutaneous coronary intervention (PCI) and pharmacotherapy. However, this decline has reached a point where the mortality remains high.[4]

Glycosylated hemoglobin (HbAlc) was discovered more than 40 years ago by Rahbar et al,[5] and it was found in 1993 in the Diabetes Mellitus (DM) Control and Complications Trial that the concentration of HbAlc is a good predictor of long-term complications associated with DM.[6,7] HbAlc has been reported to be an independent predictor of all-cause mortality and mortality from CVDs[8–10] in populations that include both DM and non-DM cohorts.[7] In DM, a 1% increase in HbAlc was associated with a 30% increase in all-cause mortality and a 40% increase in CVDs mortality.[11] In a meta-analysis of the Reykjavik Study and other Western prospective studies, fasting and postload plasma glucose levels were not significantly associated with coronary heart disease (CHD) risk in non-DM patients,[12] whereas HbAlc appeared to be

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more closely associated with CHD risk in such patients (per 1% increase in HbAlc, the RR of CHD was 1.20). In a community-based population study, it was recently reported that elevated HbAlc predicted CVDs and mortality in patients without DM, independent of fasting glucose levels.[13] Data on the prognostic role of HbAlc in patients with AMI come from different studies that differ mainly in terms of patient selection criteria, treatment (thrombolysis vs mechanical revascularization), and number concordance. Therefore, this study focused on the relationship between HbA1c concentration at admission and the prognosis of STEMI in non-DM patients.

Information and Methodology

1. Participants: A total of 142 non-diabetic patients with acute ST-segment elevation myocardial infarction (STEMI) who underwent emergency PCI in Putuo District Central Hospital of Shanghai from January 2023 to December 2024, including 116 males and 26 females, aged 30–97 years, with an average age of 64.5 + 11.0 years. All patients were administered aspirin, clopidogrel, ticagrelor, statins, low molecular weight heparin, beta-blockers, angiotensin-converting enzyme inhibitors (ACEI), or angiotensin II receptor blockers (ARB) after admission, without contraindications, and signed informed consent.

2. Inclusion and exclusion criteria: Inclusion criteria: 1. Diagnostic criteria of STEMI: ischemic chest pain lasting ≥30 min, ineffective sublingual nitroglycerin, ST segment elevation in two or more adjacent leads, limb leads ≥0.1 Mv, chest leads ≥0.2 Mv; 1. Morbidity within 12 hours. 2. Non-diabetic patients. 3. All the patients underwent PCI after admission. Exclusion criteria: 1. congestive heart failure, myocarditis, cardiomyopathy, valvular heart disease, and 2. severe liver and kidney disease, infection, autoimmune disease, and tumors; 3. Previous PCI or coronary artery bypass grafting, and 4. Incomplete clinical data.

3. Percutaneous coronary intervention (PCI): Percutaneous coronary angiography and PCI were performed by experienced cardiologists through the radial artery or femoral artery, according to conventional techniques. The success criteria for PCI were reduction of infarct-related artery (IRA) obstruction or stenosis to less than 30% and TIMI grade 3 of forward flow.

4. Observation indices: The sex, age, smoking history, history of hypertension, liver and kidney function, myocardial enzymes, number of diseased branches, LVEF, blood glucose level, and other indices of these patients were recorded. According to the HbA1c level, the patients were divided into two groups: the normal blood glucose level group (group A, HbA1c and lt; 5.7%) and the prediabetic group (group B, 5.7% ≤ HbA1c ≤ 6.4%). All patients were followed for 1 year to observe the occurrence of major cardiovascular events (MACE), which were defined as non-fatal myocardial infarction, cardiogenic shock, and severe heart failure.

5. Statistical analysis: SPSS 20.0 statistical software was used for statistical analysis. The measurement data of each clinical index were tested using a normal distribution test and expressed as mean ± standard deviation (X ± s), and the enumeration data were expressed as cases (%). Measurement data in line with the normal distribution using the *t*-test, not in line with the normal distribution using the non-parametric test, enumeration data using the chi-square χ^2 test, two-sided test, P & lt; 0.05, as statistically significant difference.

Result

1. Comparison of baseline data between the two groups: There were 57 patients in group A, including 48 males and 9 females, aged 30–86 years, with an average age of (63.1 ± 10.6) years. There were 85 patients in group B, including 68 males and 17 females, aged 32–97 years, with an average age of (65.4 ± 11.2) years. There were no significant differences in sex, age, BMI, smoking history, blood pressure, or heart rate between the two groups (all P and gt; 0.05). See Table 1.
2. Comparison of biochemical results between the two groups: There were no significant differences in liver and kidney function, blood lipids, myocardial enzymes, CRP and other aspects between the two groups (all P & gt; 0.05), but the fasting blood glucose (FPG) and BNP levels in group B were significantly higher than those in group A, and the differences were statistically significant (all P & lt; 0.01). See Table 2.
3. Comparison of the results of coronary angiography and left ventricular ejection fraction between the two groups: There was no significant difference in the results of coronary angiography between the two groups ($X^2 = 1.181$,

Table 1 Comparison of Basic Information Between Two Groups [(X±s), n (%)]

	Group A (n = 57)	Group B (n = 85)	P value
Male [n (%)]	48 (84.2%)	68 (80.0%)	0.525
Age (years)	63.1±10.6	65.4±11.2	0.224
Smoking history [n (%)]	32(56.1%)	45(52.9%)	0.708
BMI (kg/m2)	24.9±4.2	25.2±3.8	0.657
History of hypertension [n (%)]	34 (59.6%)	46 (54.1%)	0.515
Systolic blood pressure (mmHg)	127.5±19.1	126.8±22.3	0.845
Diastolic pressure (mmHg)	72.7±12.1	73.6±12.0	0.654
Heart rate (BPM)	76.6±15.2	74.5±14.1	0.386

Table 2 Comparison of Biochemical Indexes Between Two Groups (X±s)

	Group A (n = 57)	Group B (n = 85)	P value
CRP (ng/l)	8.9±27.0	5.7±17.4	0.772
PCT (ng/mL)	0.2±0.2	0.2±0.6	0.393
BNP (pg/mL)	97.9±96.8	416.1±386.2	**0.000**
CK-MB (ng/mL)	194.1±58.9	194.1±164.4	0.962
MYO (ng/mL)	2041.8±2752.0	2019.8±1865.2	0.734
TNI (ng/mL)	110.9 ±116.5	114.8±153.8	0.504
FPG (mmol/l)	5.6±1.4	6.4±1.6	**0.001**
TG (mmol/l)	1.4±0.6	1.9±2.9	0.626
TC (mmol/l)	4.9±0.8	4.9±1.1	0.854
LDL-C (mmol/l)	3.2±0.6	3.2±0.8	0.927
ALT (u/l)	59.5±52.9	59.0±35.8	0.709
AST (u/l)	219.1±157.4	266.2±185.1	0.145
Cr (umol/l)	72.6±31.2	75.3±23.5	0.561

P = 0.758), but the number of patients with multi-vessel disease in group B was significantly higher than that in group A, and the difference was statistically significant (X^2 = 6.361, P = 0.042). The left ventricular ejection fraction in group B was significantly lower than that in group A (P = 0.017). See Table 3.

4. Comparison of the occurrence of major cardiovascular events (MACE) between the two groups within one year of follow-up: In group A, 4 cases (7.0%) of MACE occurred within one year of follow-up, including 1 case (1.8%) of non-fatal myocardial infarction and 3 cases (5.2%) of severe heart failure. In group B, 16 patients (18. 8%) with

Table 3 Comparison of Coronary Angiography Outcomes and Left Ventricular Ejection Fraction Between Two Groups [n (%)]

	Group A (n = 57)	Group B (n = 85)	P value
Culprit-Vessels			0.758
LAD [n (%)]	28 (49.1%)	37 (43.5%)	(chi-quare=1.181)
LCX [n (%)]	7(12.3%)	11(12.9%)	
RCA [n (%)]	15(26.3%)	29(34.1%)	
Number of diseased coronary arteries			0.042
Single vessel disease [n (%)]	27(47.4%)	35(41.2%)	(chi-quare=6.361)
Two-vessel disease [n (%)]	25(43.9%)	29(34.1%)	
Multivessel disease [n (%)]	5(8.8%)	21(24.7%)	
Left ventricular ejection fraction EF (%)	50.3±8.7	46.8±8.2	0.017

MACE, including four cases (4.7%) with nonfatal myocardial infarction and 3 patients (3.5%) with cardiogenic shock, and 9 patients (10.6%) had severe heart failure. The incidence of MACE in group B was significantly higher than that in group A, and the difference was statistically significant ($X^2 = 3.930$, P = 0.047), as shown in Table 4.

5. Multivariate regression analysis of major cardiovascular events (MACE): Multivariate COX regression analysis showed that triglyceride, BNP, and glycosylated hemoglobin were significantly associated with MACE. Glycosylated hemoglobin can be used as an independent predictor of MACE, and the risk of MACE in the high glycated hemoglobin group (group B) was 4.98 times higher than that in the low glycated hemoglobin group (group A) (95% CI: 1.04–23.79), as shown in Table 5.
6. Survival analysis: K-M survival analysis showed that the cumulative survival of group A was higher than that of group B during the 1-year observation period (Figure 1).

Discussion

In acute myocardial infarction (AMI), stress hyperglycemia is usually secondary to an increase in catecholamine levels; therefore, examining plasma glucose levels at the time of AMI alone does not predict prognosis.[14] HbAlc, a measure of average blood glucose levels over two months,[15] is minimally affected by acute hyperglycemia, which is common in myocardial infarction.

There are only a few small-scale studies on the prognostic role of HbAlc in patients without a history of DM, with varying methods and results.[16–19] In one study, mortality and cardiogenic shock risk increased with HbAlc in 150 patients with non-DM myocardial infarction.[16] In a high-risk MI population,[18] HbAlc was a risk marker for death at follow-up in patients without DM but not in patients with DM. In a small group of patients with myocardial infarction treated with thrombolysis,[17] there was a significant relationship between admission glucose, HbAlc levels, and follow-up mortality. In contrast, hyperglycemia was a predictor of 30-day outcomes in 504 consecutive patients with STEMI without DM who underwent PCI.[19]

The reliability of HbA1c in predicting cardiovascular events has been increasingly affirmed as the gold standard for determining the level of blood glucose control. Foreign studies have shown that elevated HbA1c levels are a risk factor

Table 4 Comparison of the Occurrence of MACE Between Two Groups During One-Year Follow-Up [n (%)]

	Group A (n = 57)	Group B (n = 85)	P value
MACE [n (%)]	4(7.0%)	16(18.8%)	0.047
Nonfatal myocardial infarction [n (%)]	1(1.8%)	4(4.7%)	(chi-quare=3.930)
Cardiogenic shock [n (%)]	0(0.0%)	3(3.5%)	
Severe heart failure [n (%)]	3(5.2%)	9(10.6%)	

Table 5 Correlation of Indicators with MACE

	B	S.E.	Wald	OR (95% CI)	p-value
Gender(Male, Reference = Female)	−0.472	0.569	0.687	0.624(0.204–1.904)	0.407
Age	0.040	0.023	3.220	1.041(0.996–1.088)	0.073
History of hypertension (NO, Reference = YES)	0.422	0.503	0.703	1.525(0.569–4.085)	0.402
Smoking history (NO, Reference = YES)	−0.669	0.492	1.853	0.512(0.195–1.342)	0.173
Triglycerides	−1.359	0.581	5.475	0.257(0.082–0.802)	0.019
Cholesterol	−0.065	0.248	0.068	0.937(0.576–1.525)	0.794
Low density lipoprotein cholesterol	−0.049	0.338	0.021	0.952(0.491–1.848)	0.885
EF	−1473.116	49,171.300	0.001	0.000(0.000–0.000)	0.976
BNP	0.007	0.001	26.891	1.007(1.004–1.010)	<0.001
Number of diseased vessels	0.020	0.347	0.003	1.021(0.517–2.013)	0.953
HbA1c	1.605	0.798	4.040	4.976(1.041–23.789)	0.044

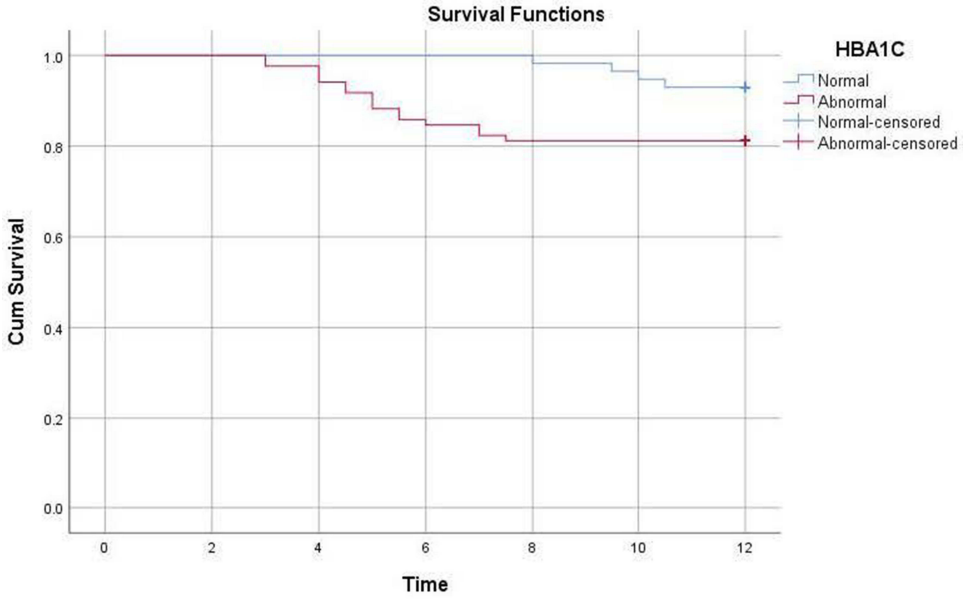

Figure 1 Survival analysis of two groups.

for acute myocardial infarction (AMI) in patients with type 2 diabetes mellitus and affect the hospitalization rate and mortality.[20] Selvin et al[13] reported that HbA1c level was independent of fasting glucose level as a predictor of cardiovascular disease and mortality in non-diabetic patients. According to the revised ADA guidelines for diabetes in 2010, 5.7% ≤ HBA1C ≤ 6.4% were prediabetic lesions, including impaired fasting glucose (IFG) and impaired glucose tolerance (IGT). Studies at home and abroad have found that more than 50% of AMI patients without a history of diabetes have prediabetic lesions.[21,22] Zhu et al[23] observed the impact of HbA1c on the prognosis of non-diabetic AMI patients and found that HbA1c concentration was independently associated with the 1-year mortality risk of patients (95% CI: 1.0–1.4, P & lt; 0.01), and the mortality rate increased by 20% for every 0.5% increase in HbA1c concentration. Timmer et al[24] analyzed 4176 non-diabetic AMI patients and showed that HbA1c concentrations at admission were associated with poor outcomes at 1 year and longer [mean follow-up of (3.3 ± 1.5) years]. However, Tian et al[25] also found that HbA1c was not an independent predictor of short-term (within 7 and 30 days) adverse outcomes in patients with AMI. Cicek et al[20] found that HbA1c levels were significantly associated with in-hospital mortality in patients with STEMI undergoing emergency PCI. However, recent studies have shown that prediabetic lesions are not associated with the prognosis of patients with AMI. Aggarwal et al[22] and Shin et al[26] found no association between prediabetes and hospitalization or 1-year mortality in STEMI patients treated with primary PCI. No association was found between prediabetes and in-hospital mortality in patients in China.[27] In this study, non-diabetic patients with acute STEMI treated by emergency PCI were enrolled, and MACE within 1 year of follow-up was selected as the primary endpoint event. The incidence of MACE in the pre-diabetic group was significantly higher than that in the normal glucose group, and the difference was statistically significant. This may be due to differences in the criteria for admission, follow-up time, follow-up endpoint events, and many other aspects of different studies.

In this study, there was no difference in age, sex, blood pressure, smoking history, liver and kidney function, blood lipids, and other clinical data between the two groups; however, FBG levels were significantly higher in the pre-diabetic group, which supports the hypothesis that some non-diabetic patients with acute STEMI had abnormal glucose metabolism before morbidity. Yin Lu et al[28] found in their analysis of the correlation between HbA1c concentration and cardiac function in patients with AMI that with an increase in plasma HbA1c concentration, the LVEF value gradually decreased, which is consistent with the results of this study, which found that with an increase in HbA1c concentration, BNP increased significantly, and vascular lesions became more serious. It has been suggested that an increase in HbA1c concentration can impair cardiac function, which may be related to the fact that high concentrations of

HbA1c can weaken the anti-platelet aggregation of vascular endothelium, enhance the degradation of collagen by matrix metalloproteinases, weaken the interaction between atherosclerotic plaque and fibrous plaque, and reduce the release of nitric oxide. It can also cause myocardial interstitial fibrosis, decreased ventricular compliance, and restrictive filling.[29–31]

The mechanism of association between elevated HbA1c levels and worsening prognosis in non-diabetic STEMI patients is not fully understood, but existing studies suggest that it may have an impact through the following multi-dimensional pathophysiological pathways: ① Chronic low-grade inflammatory activation. Even if the diagnostic criteria for diabetes are not met, elevated HbA1c levels may reflect the abnormal activation of immune cells due to long-term fluctuations in blood glucose levels. Hyperglycemia promotes the accumulation of AGEs, activates RAGE receptors on the surface of macrophages, triggers the NF-κB pathway, and leads to the release of IL-6, TNF-α, and other pro-inflammatory factors. This persistent inflammatory state accelerates the progression of atherosclerotic plaques and inhibits the repair of cardiomyocytes. Clinical studies have found that C-reactive protein levels increase by 0.8 mg/L for every 1% increase in HbA1c, suggesting a mediating role of inflammatory mediators in prognosis.[32] ② Endothelial dysfunction and oxidative stress High HbA1c level reduces the bioavailability of vasodilator nitric oxide by decreasing the activity of endothelial nitric oxide synthase (eNOS). In animal experiments, fluctuations in blood glucose increased endothelial cell mitochondrial ROS production–3-5 times, resulting in DNA damage and apoptosis. In addition, glycated LDL is more susceptible to oxidation and promotes foam cell formation and plaque destabilization.[33] Platelet hyper-reactivity. Non-enzymatic glycosylation alters the conformation of platelet membrane proteins and enhances the sensitivity of the ADP receptor P2Y12. Flow cytometry showed that the platelet aggregation rate of non-diabetic patients with HbA1c > 5.7% was 22%−35% higher than that of the normal group. This hypercoagulable state may aggravate the coronary microcirculation disorders.[34] Myocardial metabolic remodeling and a sustained hyperglycemic environment led to the shift of myocardial cells from fatty acid oxidation to glucose metabolism; however, insulin resistance reduced the efficiency of glucose uptake. Magnetic resonance spectroscopy analysis showed that the myocardial ATP production rate decreased by 18% and mitochondrial membrane potential decreased in patients with elevated HbA1c, which aggravated myocardial stunning after ischemia-reperfusion injury.[34]

It should be noted that the recent study by Zia-Ul-Sabah et al[35] has some similarities to our study. This study discusses the impact of HbA1c on the left ventricular ejection fraction (LVEF) and cardiac function. Type D personality was an independent risk factor for poor left ventricular remodeling after PCI in patients with STEMI. Combined with the Killip classification, GLS, and the degree of coronary artery disease, it can effectively identify high-risk groups and provide the basis for multi-dimensional intervention. Future cardiovascular risk management needs to integrate psychological assessments and biomedical indicators to achieve precision medicine. By highlighting that psychological factors, such as personality type, also influence left ventricular remodeling and outcome in patients with ST-elevation myocardial infarction (STEMI), this citation will help enhance the discussion and add another layer to the complexity of prognosis after PCI.

In conclusion, HbA1c levels in non-diabetic patients with acute STEMI undergoing PCI can affect their postoperative cardiac function and increase the incidence of MACE. Therefore, for non-diabetic patients with STEMI, HbA1c concentration should be actively monitored, and appropriate lifestyle interventions should be carried out if necessary. However, due to the relatively small sample size of this study, the conclusion needs to be further confirmed by a large multicenter prospective cohort study, and whether appropriate intervention can improve the prognosis of patients with prediabetic lesions should be further explored.

Abbreviations

CVDs, cardiovascular diseases; AMI, acute myocardial infarction; STEMI, ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; HbA1c, glycosylated hemoglobin; DM, diabetes mellitus; CHD, coronary heart disease; RR, relative risk; ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin II receptor blockers; IRA, infarct-related artery; MACE, major cardiovascular events; FPG, fasting blood glucose; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; CRP, C-reactive protein.

Ethical Declaration Statement

This study was approved by the Ethics Committee of Putuo Hospital, Shanghai University of Traditional Chinese Medicine (approval number: No. PTEC-A-2024-40(S)-1.). Patients were consented by an informed consent process that was reviewed by the Ethics Committee of Putuo Hospital, Shanghai University of Traditional Chinese Medicine and certify that the study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki.

Chinese Clinical Trial Registry Under the Registration Number

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Disclosure

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