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Glycemic Comparison Index (GCI): a retrospective analysis of its prognostic value in ICU patients with AMI and diabetes

Yingfang She[1†], Chunfei Wang[2†], Le Fu[3†], Liang Luo[3*] and Yide Li[3*]

Abstract

Background Acute myocardial infarction (AMI) has a significant impact on global health, especially among individuals with diabetes, emphasizing the need for specialized glycemic management. This study examines the glycemic comparison index (GCI), a novel prognostic tool designed for patients with AMI and diabetes, aiming to enhance glucose management in critical care settings.

Methods This retrospective cohort analysis used data from the Medical Information Mart for Intensive Care IV database (version 2.2). The GCI was calculated by comparing mean blood glucose levels in the intensive care unit (ICU) to baseline glucose levels. Patients were stratified into tertiles based on their GCI scores. The primary outcome measured was one-year all-cause mortality, while secondary outcomes included hospital mortality, ICU-free days, and hypoglycemic events. Statistical analyses included time-dependent receiver operating characteristic (ROC), cox proportional hazards models, generalized linear models (GLM), and restricted cubic spline analysis.

Results The patient population comprised 622 individuals, with a mean age of 69.9 years and 64.6% male representation. The high GCI group exhibited the highest one-year mortality rate and fewer ICU-free days, while the low GCI group exhibited a higher incidence of hypoglycemia. Statistical analyses revealed that GCI was a significant predictor of one-year all-cause mortality (hazard ratio: 2.21, 95% confidence interval: 1.51–3.24). Analysis using time-dependent ROC confirmed the consistent predictive accuracy of GCI for survival at 1, 6, and 12 months (area under the curve: 0.671, 0.670, and 0.634, respectively). Furthermore, GLM analysis indicated that a higher GCI was associated with fewer ICU-free days.

Conclusions Higher GCI values are associated with increased one-year mortality and fewer ICU-free days in patients with AMI and diabetes. In comparison, lower GCI values are correlated with a higher risk of hypoglycemia. The GCI demonstrates potential as a personalized prognostic tool, although further validation is needed.

Keywords Acute myocardial infarction, Diabetes mellitus, Glycemic comparison index, Prognosis, Retrospective analysis

†Yingfang She, Chunfei Wang and Le Fu contributed equally to this work and share the first authorship.

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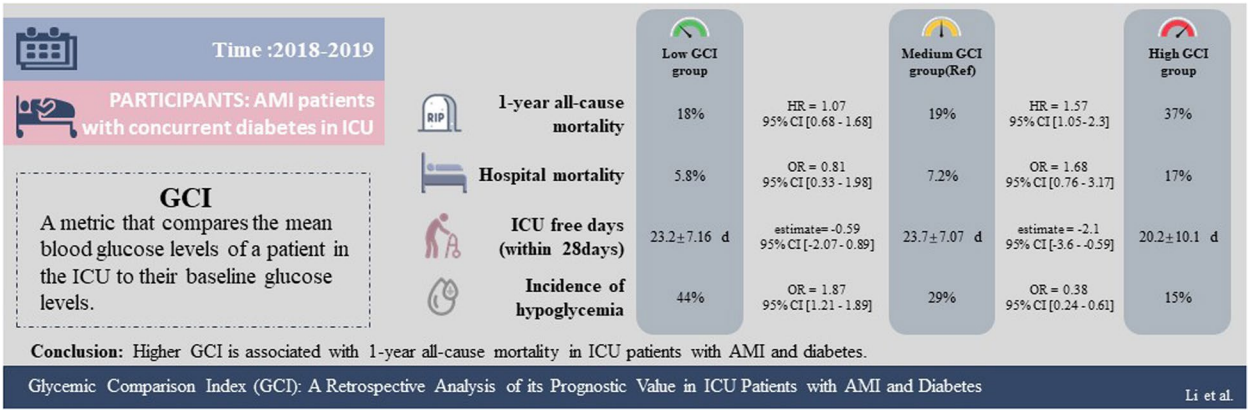
Graphical Abstract



Background

Acute myocardial infarction (AMI) remains a major contributor to the global health burden and mortality rates, with diabetes recognized as a significant risk factor [1–3]. A 2023 meta-analysis by Salari et al. reported that AMI affected 3.8% of individuals younger than 60 years, while the incidence increased to 9.5% in those over 60 [4]. Additionally, a study by Arnold et al. demonstrated that 38% of patients with AMI were diagnosed with diabetes, and another 31% exhibited a prediabetic state [5], highlighting the strong connection between these two conditions.

Elevated glucose levels in patients with AMI and diabetes are associated with harmful outcomes, including increased oxidative stress, inflammation, and a higher risk of thrombosis, which can worsen cardiac damage and delay recovery [6, 7]. Current guidelines recommend keeping blood glucose levels between 140 and 180 mg/dL (7.8–10.0 mmol/L) in intensive care unit (ICU) settings [8, 9]. However, these recommendations are general and not specifically designed for patients with AMI, creating uncertainty regarding the effectiveness of these universal glucose management targets across patients with different comorbidities and pre-admission glucose control [10, 11].

Personalized glycemic management, particularly the use of baseline glycemic control to establish individualized targets, has gained recognition in critically ill patients, especially those with diabetes and AMI [12]. Conventional glycemic indicators, such as mean blood glucose levels, fail to be considered for individual baseline glycemic control, which may result in under- or overtreatment. The Glycemic Comparison Index (GCI) is a novel metric that compares mean blood glucose readings in the ICU with baseline levels derived from glycated hemoglobin (HbA1c) measurements [13]. This study explores whether GCI can more effectively stratify risk and guide glycemic targets than standard glucose measurements. The primary focus is on examining the association between GCI and the prognosis of diabetic patients with AMI, providing a foundation for future research to evaluate the impact of targeted GCI interventions on patient outcomes.

Methods

Design and ethical considerations

This single-center, retrospective analysis utilized a high-quality, de-identified database of ICU patients to investigate the association between GCI and the prognosis of AMI patients with diabetes. The Institutional Review Boards at both the Massachusetts Institute of Technology and the Beth Israel Deaconess Medical Center (BIDMC) approved the use of these de-identified data, ensuring compliance with ethical standards and patient privacy protections.

GCI calculation

The mean blood glucose measurement in the ICU was compared to the baseline glucose levels derived from HbA1c to calculate the GCI. The GCI was calculated using the following formula:

$$\mathrm{GCI} = 100 \times \text{mean blood glucose in ICU (mg/dL)}/[(28.7\mathrm{H} \times \mathrm{A1c}\%) - 46.7]$$

The denominator facilitated the conversion of HbA1c values to average glucose levels in this formula, which

is derived from the study by Nathan et al. [13]. Subsequently, this approach has been used to calculate the stress hyperglycemia ratio, a metric widely applied in various research studies [14–16].

Study population

This retrospective cohort analysis used data from the Medical Information Mart for Intensive Care IV (MIMIC-IV) database (version 2.2). The database contains anonymized health-related data from patients admitted to the ICUs at BIDMC between 2008 and 2019. The MIMIC-IV database is an invaluable resource for clinical research due to its comprehensive patient records, encompassing demographic details, vital signs, laboratory results, and medication data.

The inclusion criteria for the study cohort were as follows: (1) Patients with a concurrent diagnosis of AMI and diabetes mellitus and (2) patients admitted to the ICU for the first time. The exclusion criteria were as follows: (1) ICU stay of less than 2 days and (2) missing HbA1c levels, either during the ICU stay or within the month before ICU admission. The attending physician diagnosed patients and recorded the information in the medical records. The attending physician's diagnoses were converted into International Classification of Diseases (ICD) codes and included in the MIMIC-IV database. The ICD codes were employed to identify and select relevant diagnoses for the cohort. The specific codes are available in the Supplementary Material Table S1.

A total of 622 patients met the study's eligibility criteria. These individuals were subsequently stratified into three categories: low, medium, and high, based on the tertiles of their GCI scores. Figure 1 illustrates the selection and stratification process.

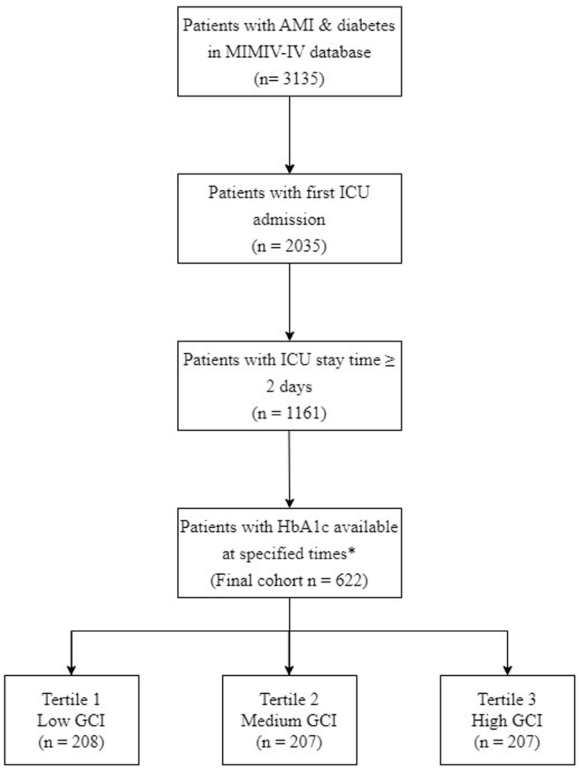


Fig. 1 Flowchart of the cohort selection process. *During the ICU stay or within the month preceding ICU admission. AMI: Acute Myocardial Infarction; GCI: Glycemic Comparison Index

Data extraction

Data for this retrospective analysis were meticulously extracted using the PostgreSQL database (version 11.1, http://www.postgresql.org/). The extraction process focused on collecting demographic information, comorbidities, laboratory data from the first 24-h ICU admission, and dialysis or mechanical ventilation initiation within this timeframe. Additionally, all ICU blood glucose results, procedures such as Percutaneous Coronary Intervention (PCI) or Coronary Artery Bypass Grafting (CABG), severity of illness scores, prognosis, and duration of ICU stay were collected. For tests other than blood glucose, the first recorded result was selected to represent the initial condition of patients upon ICU admission. All blood glucose measurements collected throughout the ICU stay, including fingerstick, venous whole blood, and plasma samples, were used regardless of timing or measurement method. All values were standardized to mg/dL. The first recorded HbA1c measurement in the ICU was prioritized as it best reflected pre-admission glycemic control. If unavailable, the most recent value was used within one month before ICU admission.

Primary and secondary outcomes

This study primarily assessed the all-cause mortality rate at one year. Secondary outcomes included the hospital mortality rate, ICU-free days within the first 28 days following ICU admission, and hypoglycemic events during the ICU stay.

Subgroup analysis

The impact of the GCI on one-year all-cause mortality was analyzed across various patient subgroups. Cox proportional hazard (CPH) models were used to calculate hazard Ratios (HRs), with stratification based on gender, age, HbA1c level, CABG operation, use of vasoactive medications within the first 24 h, and congestive heart failure.

Statistical analysis

Quantitative variables are presented as the median and interquartile range (IQR) and compared using the

Kruskal–Wallis rank sum test. Depending upon applicability, categorical variables are expressed as frequencies (percentages) and analyzed using Chi-square or Fisher's exact tests. Kaplan–Meier survival analysis estimated the incidence of primary outcome events across GCI stratification groups, with differences evaluated using the log-rank test. The association between GCI and one-year mortality was analyzed using four CPH regression models. The first model applied a univariate CPH approach, considering GCI alone, while subsequent models incorporated multivariate CPH models. The second model adjusted for PCI, CABG, and tumor comorbidities. The third model included additional adjusted for Acute Physiology Score III (APSIII) scores and heart failure comorbidities. Stepwise regression within the CPH framework was performed in the fourth model for variable selection. This method iteratively evaluated the inclusion or exclusion of variables based on the Akaike Information Criterion (AIC), optimizing the model for explanatory power and simplicity [17]. Across all models, the intermediate GCI level served as the reference category. Additionally, generalized linear models (GLM) with AIC-guided stepwise regression assessed ICU-free days within a 28-day window. Time-dependent receiver operating characteristic (ROC) curve analysis evaluated the predictive accuracy of GCI for the primary outcomes, with the area under the curve (AUC) values indicating the model performance over time. Restricted cubic spline (RCS) analysis explored the dose–response relationship between GCI and the risk of primary outcomes. Statistical analyses were conducted in R software (version 4.3.2) [18], with a two-tailed *P*-value of < 0.05 considered statistically significant. Missing data were imputed using the Multiple Imputation by Chained Equations package in R software [19].

Sensitivity analysis

As a novel indicator, GCI incorporates an average value in its formula's denominator. A mean value was also applied to the numerator to maintain the index's rationality. The sensitivity analysis compared the GCI calculation method using the average blood glucose with the alternative approach based on the median.

Results

Baseline characteristics

The study cohort included 622 patients with a mean age of 69.93 years, with males comprising 64.6% of the population. Figure 2 illustrates the distribution of mean

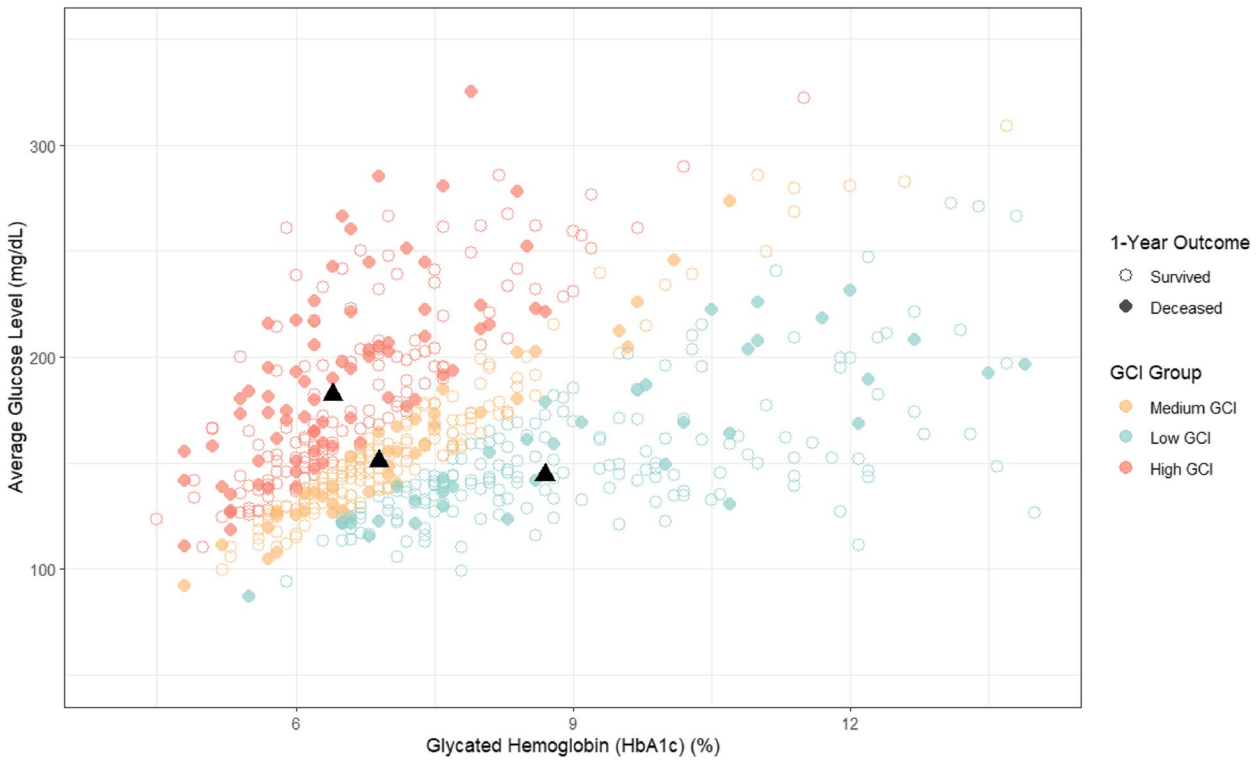


Fig. 2 Scatter plot of mean glucose levels and HbA1c across GCI tertiles, differentiated by one-year outcomes. *This scatter plot illustrates the relationship between mean glucose levels and HbA1c across GCI groups. Each circle represents patients, color-coded by GCI group (low, medium, and high), with open circles indicating survivors and solid circles representing deceased patients. Black triangles denote the median glucose and HbA1c for each group*

glucose and HbA1c levels across the low, medium, and high GCI groups. The scatter plot visually depicts the association between these two variables, using color coding to differentiate the GCI groups. Table 1 presents the baseline characteristics of the cohort stratified by the GCI. Some covariates exhibited significant differences among the low, medium, and high GCI groups. Higher GCI levels were associated with a significant increase in age ($P<0.001$) and hospital and one-year mortality rates, with the highest mortality rates observed in the high GCI group ($P<0.001$). Hypoglycemic events were inversely correlated with GCI and occurred most frequently in the low GCI group ($P<0.001$). The high GCI group underwent interventions such as PCI and CABG more often ($P=0.003$ and $P<0.001$, respectively). Furthermore, greater GCI was associated with increased illness severity, as reflected by higher APSIII scores and a higher prevalence of congestive heart failure and renal disease (both $P<0.001$).

Primary outcome

The Kaplan–Meier survival curves indicated considerably lower one-year survival probability in the high GCI group compared to the intermediate and low GCI groups (Fig. 3). The log-rank test confirmed the statistical significance of this difference ($P<0.001$). Subsequent analysis using four CPH models verified the Kaplan–Meier findings, demonstrating a significantly elevated risk of one-year all-cause mortality in the high GCI group compared to the medium GCI group (Model 1 HRs: 2.21, 95% confidence interval [CI]: 1.51–3.24, $P<0.001$; Model 4 HRs: 1.57, 95% CI: 1.05–2.34, $P=0.028$). These results remained significant even after adjusting for multiple confounding factors. No significant differences in the one-year mortality risk were observed between the low and medium GCI groups across all models. Detailed results from these models are provided in Supplementary Material Table S2.

The RCS model demonstrated an L-shaped relationship between the GCI as a continuous variable and the one-year all-cause mortality rate, with an inflection point at a GCI of 98.54. Figure 4 depicts this relationship. Segmental regression analysis was conducted on both sides of this inflection point to further elucidate the association. The HR per unit GCI was 1.008 (95% CI: 0.988–1.027; $P=0.451$) for GCI values below 98.54 and 1.003 (95% CI: 1.001–1.015; $P<0.001$) for GCI values equal or above 98.54.

The time-dependent ROC curve confirmed GCI as a strong prognostic marker for survival outcomes at 1, 6, and 12 months (Fig. 5). This time-dependent ROC analysis provides a distinct advantage over conventional ROC approaches by enabling the evaluation of GCI's predictive capabilities at various temporal milestones while considering the evolving risk profile over time. The AUC values demonstrated GCI's consistent predictive accuracy, decreasing slightly from 0.671 at 1 month to 0.634 at 12 months, indicating that GCI remains a relevant prognostic indicator throughout the year.

Secondary outcome

A stepwise regression analysis using AIC revealed that GCI did not have a significant impact on in-hospital mortality rates [odds ratio (OR) for low GCI: 0.81, 95% CI: 0.33–1.95, $P=0.65$; OR for high GCI: 1.68, 95% CI: 0.82–3.60, $P=0.17$]. In contrast, GCI emerged as a significant predictor of hypoglycemia in the ICU. A notable association was observed between low GCI and an increased risk of hypoglycemia (OR: 1.86, 95% CI: 1.22–2.86; $P=0.004$), while a high GCI was significantly associated with reduced incidence of hypoglycemia (OR: 0.38; 95% CI: 0.24–0.61; $P<0.001$). Moreover, a GLM regression indicated a significant reduction in ICU-free days in the high GCI group compared to the intermediate group (estimate: –2.07; 95% CI: –2.01 to –0.94; $P=0.007$), while statistically non-significant difference was observed in the low GCI group (estimate: -0.53; 95% CI: –3.57 to 0.56; $P=0.48$). Detailed results are provided in the Supplementary Material Table S3.

Subgroup analysis

Subgroup analysis revealed a significant increase in the one-year all-cause mortality risk within the high GCI group across various patient subgroups. This association was particularly pronounced among male patients, individuals over 70 years of age, patients who had not undergone CABG, those who did not receive vasoactive medications within the first 24 h of ICU admission, and patients without a diagnosis of congestive heart failure. No significant interactions were observed. Detailed outcomes, including HRs and *P*-values for each subgroup, are provided in Supplementary Material Table S4.

Sensitivity analysis

The median ICU blood glucose value was employed in the sensitivity analysis to calculate the median-based GCI (mGCI), which was stratified into tertiles. Kaplan–Meier survival curves for these groups, depicted in Supplementary Material Figure S1, align with the primary analysis and revealed a significantly lower one-year survival rate in the high mGCI group. The log-rank test confirmed this finding with a *P*-value of <0.001.

Table 1 Comparison of patient characteristics by GCI stratification groups

Characteristics	Low GCI $N=208^a$	Medium GCI $N=207^a$	High GCI $N=207^a$	Missing(%)	*P*-value[2]
Range	≤88.82	88.82–108.79	>108.79		
Demographics					
Male	133 (64%)	137 (66%)	132 (64%)	0	0.8
Age (years)	67.97 (61.00, 76.08)	70.56 (61.63, 78.69)	73.99 (66.27, 80.00)	0	<0.001
Race				0	0.2
Asian	6 (2.9%)	2 (1.0%)	4 (1.9%)		
Black/African American	14 (6.7%)	12 (5.8%)	16 (7.7%)		
Hispanic or Latino	13 (6.3%)	6 (2.9%)	7 (3.4%)		
White	122 (59%)	147 (71%)	124 (60%)		
Other	53 (25%)	40 (19%)	56 (27%)		
Prognosis					
Length of ICU stay (days)	3.77 (2.73, 5.65)	3.46 (2.45, 5.19)	4.05 (2.81, 6.94)	0	0.071
Length of hospital stay (days)	10.85 (7.98, 15.68)	10.21 (7.14, 14.57)	11.05 (6.29, 16.01)	0	0.6
Hospital mortality	12 (5.8%)	15 (7.2%)	36 (17%)	0	<0.001
one-year mortality	38 (18%)	40 (19%)	76 (37%)	0	<0.001
Hypoglycemia	91 (44%)	60 (29%)	32 (15%)	0	<0.001
Interventions					
PCI	21 (10%)	25 (12%)	44 (21%)	0	0.003
CABG	148 (71%)	122 (59%)	68 (33%)	0	<0.001
Dialysis within the first 24 h of ICU admission	2 (1.0%)	8 (3.9%)	16 (7.7%)	0	0.003
Mechanical ventilation within the first 24 h of ICU admission	127 (61%)	100 (48%)	96 (46%)	0	0.005
Usage of vasoactive drugs within the first 24 h of ICU admission	124 (60%)	115 (56%)	103 (50%)	0	0.13
GCI-related					
Mean blood glucose in ICU stay (mmol/L)	8.01 (7.29, 9.35)	8.37 (7.51, 9.41)	10.13 (8.56, 12.06)	0	<0.001
Median blood glucose in ICU stay (mmol/L)	7.61 (6.78, 8.76)	8.00 (7.11, 9.06)	9.53 (8.16, 11.32)	0	<0.001
HbA1c (%)	8.70 (7.60, 10.50)	6.90 (6.35, 7.70)	6.40 (5.85, 7.20)	0	<0.001
GCI	76.27 (64.04, 82.77)	99.87 (94.28, 104.68)	125.62 (116.07, 143.08)	0	<0.001
Comorbidities					
Congestive heart failure	113 (54%)	113 (55%)	151 (73%)	0	<0.001
Cerebrovascular disease	50 (24%)	41 (20%)	49 (24%)	0	0.5
Chronic pulmonary disease	44 (21%)	52 (25%)	57 (28%)	0	0.3
Renal disease	65 (31%)	82 (40%)	95 (46%)	0	0.009
Cancer	10 (4.8%)	13 (6.3%)	12 (5.8%)	0	0.8
Illness severity					
APSIII	41.50 (34.00, 53.00)	42.00 (34.00, 55.00)	49.00 (38.00, 62.00)	0	<0.001
Urine output within the first 24 h of ICU admission (mL)	1,525.00 (1,077.25, 1,986.25)	1,575.00 (992.50, 2,323.75)	1,310.00 (782.50, 2,110.00)	11(1.77)	0.13
Laboratory results					
PT (s)	14.15 (13.05, 15.55)	14.13 (13.01, 15.30)	13.65 (12.50, 15.25)	16 (2.27)	0.085
APTT (s)	36.13 (29.19, 49.90)	38.70 (30.28, 62.18)	40.23 (29.91, 62.34)	15 (2.41)	0.049
RDW (%)	14.00 (13.20, 14.95)	14.15 (13.33, 15.23)	14.40 (13.50, 15.70)	1 (0.16)	0.021
Hematocrit (%)	30.63 (27.94, 34.50)	30.70 (27.98, 36.13)	30.70 (27.80, 36.03)	0	>0.9
Hemoglobin (g/dL)	10.23 (9.35, 11.45)	10.05 (9.25, 11.80)	10.15 (9.00, 11.93)	0	0.8
Platelets ($\times 10^9$/L)	184.50 (144.38, 236.38)	190.00 (145.50, 265.25)	203.00 (157.50, 274.75)	0	0.050
WBC ($\times 10^9$/L)	12.75 (9.94, 15.70)	12.25 (10.03, 15.58)	12.65 (10.40, 15.73)	0	0.6

Table 1 (continued)

Characteristics	Low GCI $N=208^a$	Medium GCI $N=207^a$	High GCI $N=207^a$	Missing(%)	*P*-value[2]
Anion Gap (mmol/L)	13.50 (11.38, 16.00)	14.50 (12.00, 16.50)	15.50 (13.00, 18.50)	0	<0.001
Bicarbonate (mmol/L)	23.00 (21.50, 24.50)	23.00 (20.75, 25.00)	22.50 (20.00, 25.00)	0	0.8
BUN (mg/dL)	19.50 (14.50, 30.13)	24.00 (16.50, 35.25)	28.00 (19.50, 50.75)	0	<0.001
Creatinine (mmol/L)	1.05 (0.85, 1.40)	1.15 (0.85, 1.75)	1.45 (1.00, 2.53)	0	<0.001
Potassium (mmol/L)	4.40 (4.10, 4.75)	4.35 (4.03, 4.70)	4.35 (3.95, 4.70)	1 (0.16)	0.5
Sodium (mmol/L)	137.50 (136.00, 140.00)	138.00 (136.00, 139.50)	137.50 (134.50, 140.00)	0	0.7

APSIII Acute Physiology Score III, *APTT* Activated Partial Thromboplastin Time, *BUN* Blood Urea Nitrogen, *CABG* Coronary Artery Bypass Graft, *GCI* Glycemic Comparison Index, *PCI* Percutaneous Coronary Intervention, *PT* Prothrombin Time, *RDW* Red Distribution Width, *WBC* White blood cell

[a] n (%): Median (IQR)

[2] Pearson's Chi-squared test; Kruskal–Wallis rank sum test; Fisher's Exact Test for Count Data (with simulated *P*-value based on 2000 replicates)

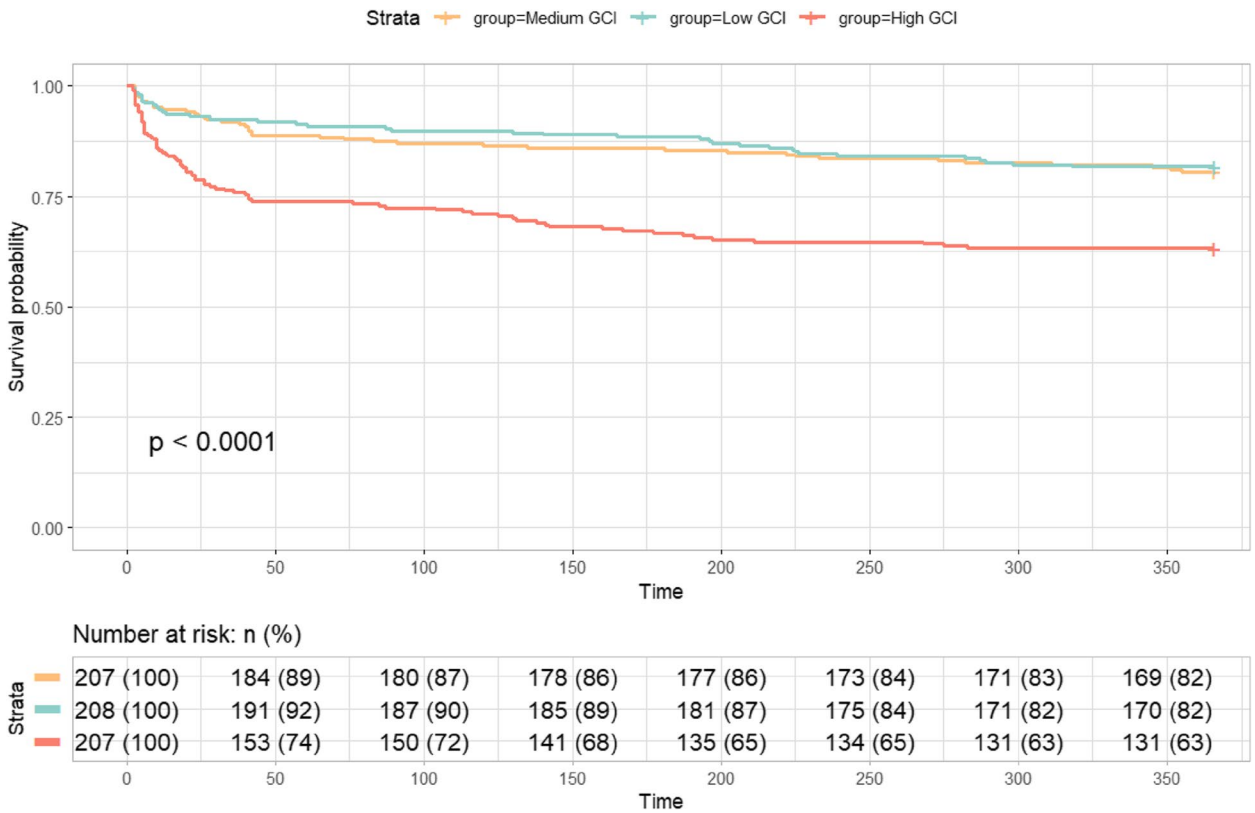


Fig. 3 Kaplan–Meier survival curves for one-year all-cause mortality analysis. *Kaplan–Meier survival curves for one-year all-cause mortality stratified by GCI groups (low, medium, and high), with a table below displaying the number of patients at risk at each time point (in days)*

Discussion

This study is the first to investigate the association between GCI and prognosis in a specific cohort of patients with AMI and concurrent diabetes mellitus. The findings indicate that patients in the high GCI group have a higher one-year mortality rate and fewer ICU-free days within 28 days compared to those in the medium or low GCI, with no significant difference in hospital mortality rates. Furthermore, a lower GCI is associated with a higher incidence of hypoglycemia, highlighting the complex balance required in glycemic management within this patient population.

The prognosis for patients with AMI and diabetes is significantly influenced by the duration and progression of their diabetes and their past glycemic control [20, 21]. One potential explanation is the 'vascular hyperglycemia

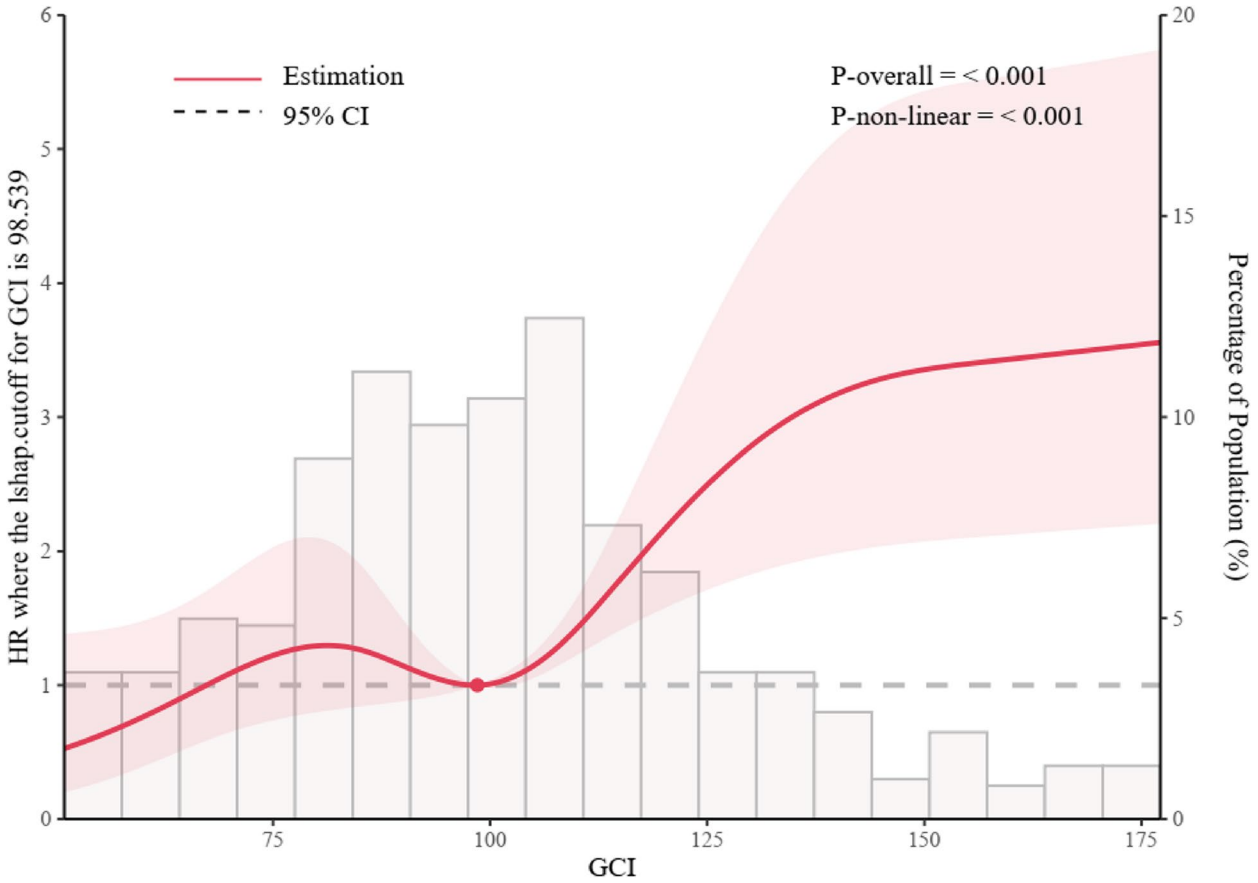


Fig. 4 RCS regression analysis of GCI and one-year all-cause mortality

memory' hypothesis [22, 23], which suggests that prolonged exposure to high glucose levels can create a lasting inflammatory state in the vascular system that cannot be reversed by subsequent glycemic control. This study's findings may support this hypothesis, as higher values could reflect inadequate glycemic management and increased vascular susceptibility, even if glycemic control is normalized during the acute phase. Supporting evidence from multiple studies demonstrates that long-term hyperglycemia can lead to persistent vascular inflammation and endothelial dysfunction due to epigenetic modifications, regardless of later glucose normalization [24, 25]. Hyperglycemia, hypoglycemia, and glycemic variability play significant roles in determining outcomes for patients with AMI [26–29]. Hyperglycemia can intensify oxidative stress and inflammatory responses, hindering cardiac repair and leading to endothelial dysfunction [30, 31]. Conversely, hypoglycemia is associated with heightened platelet activity and coagulation anomalies, which introduce additional risks [32, 33]. These findings highlight the importance of precise glycemic monitoring and management in patients with AMI, emphasizing the delicate balance needed to improve patient outcomes.

This study's findings indicate a worse prognosis for patients with AMI and diabetes in the high GCI group. Elevated GCI, which reflects high mean glucose levels during the ICU stay or low HbA1c levels, suggests two distinct patient profiles. Each profile may have different underlying mechanisms that could influence their prognosis.

1. Elevated ICU Blood Glucose (Large Numerator): High mean glucose levels in this subset of patients may reflect the severity and persistence of stress-induced hyperglycemia, which indicates an intensified or prolonged stress response [34, 35]. Sustained hyperglycemia may intensify oxidative stress [36], promote pro-inflammatory pathways [37, 38], increase thrombotic risk [39], compromise coronary perfusion during PCI procedures [40], and prolonged metabolic stress, which could impede myocardial recuperation [41] and increase susceptibility to infections [42], thereby exacerbating the prognosis for patients with AMI and diabetes [43, 44].

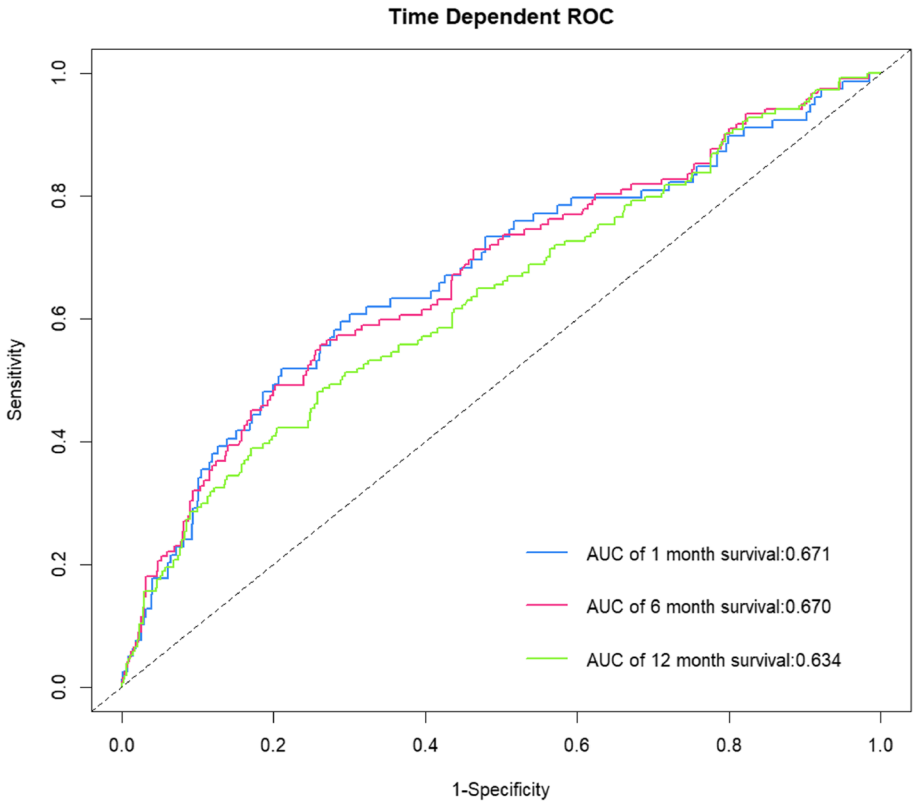


Fig. 5 Time-dependent ROC curves for the GCI predicting 1-, 6-, and 12-month survival outcomes. *Time-dependent ROC curves for GCI predict survival at 1-, 6-, and 12-month intervals, with AUC values of 0.671, 0.670, and 0.634, respectively*

2. Low Baseline Glycemic Control (Small Denominator): Patients who experience elevated hyperglycemia during ICU stays, despite having good baseline glycemic management, may be undergoing a strong stress response, which indicates an adverse prognosis [45, 46]. These acute hyperglycemic episodes could indicate the onset of acute insulin resistance, impaired myocardial reperfusion, and reduced coronary microcirculation function, all of which could adversely impact their prognosis [47, 48]. Moreover, the threshold for harmful glucose levels may vary based on baseline glycemic conditions, where patients with optimal baseline control might experience adverse outcomes at lower glucose thresholds. Eitel et al.'s research demonstrates that the glucose threshold for myocardial injury risk differs based on the patient's diabetic status, with non-diabetic AMI individuals exhibiting significantly lower thresholds than those with diabetes [49].

The study examines the association between GCI and prognosis in patients with AMI and diabetes, but it has some limitations. First, the retrospective design limits the ability to make causal inferences. Second, using a single-center database may limit the generalizability of the findings. Third, using predetermined thresholds GCI requires validation in different clinical settings, and the optimal threshold has not been determined. Fourth, the potential influence of unmeasured confounding factors, such as cardiac function classification and other clinical variables, requires careful interpretation of the results. These limitations highlight the need for further prospective, multicenter studies to confirm and expand the clinical utility of GCI.

Conclusion

This study demonstrates that in ICU patients with AMI and diabetes, a high GCI correlates with higher one-year all-cause mortality and fewer ICU-free days within 28 days. Conversely, a low GCI is linked to an increased risk of hypoglycemia.

Abbreviations

AMI	Acute Myocardial Infarction
APSIII	Acute Physiology Score III
APTT	Activated Partial Thromboplastin Time
BIDMC	Beth Israel Deaconess Medical Center
BUN	Blood Urea Nitrogen
CABG	Coronary Artery Bypass Graft
GCI	Glycemic Comparison Index
ICU	Intensive Care Unit
IRBs	Institutional Review Boards
MIT	Massachusetts Institute of Technology

MIMIC-IV	Medical Information Mart for Intensive Care IV
PCI	Percutaneous Coronary Intervention
PT	Prothrombin Time
RDW	Red Cell Distribution Width
WBC	White Blood Cell

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12902-025-01907-2.

Supplementary Material 1

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Clinical trial number
Not applicable.

Authors' contributions
YS performed data extraction and statistical analysis and drafted the initial manuscript. CW conceptualized the study, developed the methodology, and interpreted the results. LF participated in data curation, formal analysis, and manuscript writing. LL and YL jointly supervised the project, provided critical feedback, and revised the manuscript for intellectual content. All authors read and approved the final manuscript.

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Data availability
Due to the licensing restrictions of the MIMIC database, the data files used in this study are not directly available. The source code for the analyses conducted in this study will be accessible at https://github.com/shaou77/GCI following the publication of this paper. For further inquiries or requests for data access under reasonable conditions, please contact the corresponding author via email.

Declarations

Ethics approval and consent to participate
Given the retrospective nature of this study, which utilizes a de-identified dataset, the need for individual patient consent was waived. This study was conducted in strict accordance with ethical standards, and the Institutional Review Boards at both the Massachusetts Institute of Technology (Protocol No. 0403000206) and the BIDMC (Protocol No. 2001-P-001699/14) granted approval for the use and analysis of the data.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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