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Original Article

The Effect of Fasting Blood Glucose (FBG) on Troponin I and Clinical Outcomes in Acute Coronary Syndrome (ACS): A Retrospective Analysis

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ABSTRACT

Background: In the early phase of acute coronary syndrome (ACS), simple biomarkers such as fasting blood glucose (FBG) and cardiac troponin I (cTnI) may offer prognostic insights. However, evidence regarding their relationship remains inconsistent, particularly within Indonesian populations.
Objective: To investigate the correlation between FBG and cTnI and to evaluate the association of FBG with short-term clinical outcomes among patients with ACS at Karsa Husada Hospital, Batu, Indonesia.
Methods: This retrospective observational study included 75 hospitalized patients diagnosed with ACS. Clinical, biochemical, and outcome variables—including length of stay, complications, and cardiovascular comorbidities—were extracted from medical records. Statistical analyses comprised Spearman correlation, linear regression, categorical trend testing, and restricted cubic spline modeling, adjusted for key clinical covariates such as age, sex, diabetes, renal function, and hemodynamic parameters.
Results: The median age was 62 years, and 64% were male. FBG demonstrated a weak, non-significant correlation with cTnI (r_s = 0.19; p = 0.12). Each 10 mg/dL increase in FBG corresponded was associated with an 8.6% higher cTnI level (β = 0.036; 95% CI −4.5 to +23.6%; p = 0.20). Hyperglycemia (≥126 mg/dL) was linked to slightly higher proportions of prolonged hospitalization (>3 days) and in-hospital complications, but these differences were not statistically significant (all p > 0.05).
Conclusion: Elevated fasting glucose in ACS appears to reflect transient metabolic and neurohormonal stress rather than directly determining myocardial injury or short-term prognosis. Larger, multicenter studies are warranted to validate these findings and refine the prognostic utility of FBG in acute cardiac care.

Keywords: fasting blood glucose, troponin I, acute coronary syndrome, stress hyperglycemia, clinical outcomes

INTRODUCTION

Cardiovascular disease remains the leading cause of death worldwide, accounting for an estimated 19.8 million deaths in 2022 (≈32% of all global deaths).[1] According to data from the 2018 Riskesdas survey, the prevalence of Acute Coronary Syndrome (ACS) in Indonesia was recorded at 1.5%. Meanwhile, the 2015 World Health Organization (WHO) report stated that deaths due to ACS in Indonesia reached approximately 17.7 %, or nearly half of the total 39.5 million deaths caused by non-communicable diseases.[2] A meta-analysis study reported that ACS patients with diabetes and hyperglycemia have a mortality risk approximately 1.7 times higher than normoglycemic diabetes patients, while non-diabetic patients with hyperglycemia have a risk 3.8 times higher. Therefore, it is crucial to perform glucose testing when patients first present.[3]

Biologically, acute hyperglycemia has the potential to exacerbate myocardial injury through increased oxidative stress, endothelial dysfunction, inflammatory activation, and a prothrombotic state that contributes to microvascular obstruction.[4] Consequently, hyperglycemia may be associated with larger infarct size and higher troponin release.[5] However, in clinical practice, interpreting FBG is not always straightforward: FBG levels at admission may reflect stress hyperglycemia from sympathetic activation and cortisol release, rather than chronic glycemic control alone.[6] On the other hand, troponin levels are significantly influenced by the duration of ischemia, time to sample collection, reperfusion strategy, and renal function. Therefore, the relationship between FBG and cTnI may be confounded by these factors.[7,8]

Previous studies on the association between acute hyperglycemia and troponin levels have shown inconsistent results.[9] Several studies have demonstrated a positive association between acute hyperglycemia and troponin release in ACS patients; however, the results are inconsistent due to clinical factors such as reperfusion success, intervention timing, and hemodynamic status.[10,11] Most ACS studies assess fasting glucose or 2-hour postprandial glucose (2h-PG), which has proven to be more prognostic than FBG, while recent studies indicate that FBG is also associated with coronary artery disease severity, though its use is limited due to the requirement for fasting.[12,13]

Despite numerous investigations, the prognostic significance of fasting hyperglycemia in ACS remains debated, particularly after adjustment for confounding factors such as diabetes, renal function, and hemodynamic status. Recent studies have suggested that stress-induced hyperglycemia during ACS reflects transient neurohormonal activation mediated by catecholamines and cortisol rather than chronic metabolic dysfunction [14,29]. However, few data are available from Southeast Asian populations, and evidence linking FBG to both biochemical injury markers and in-hospital clinical outcomes remains scarce [30]. Addressing this gap is essential, as understanding whether FBG represents a stress marker or a prognostic determinant may refine risk stratification and early management strategies in ACS patients.

Therefore, this study aims to evaluate the effect of FBG on cardiac Troponin I (cTnI) levels in ACS patients treated at Karsa Husada Hospital, Batu. These findings are expected to provide relevant local context for the initial risk assessment of ACS patients and clarify whether FBG can serve as an additional risk marker for myocardial injury burden as reflected by troponin I.

METHODS

This retrospective observational study reviewed the medical records of patients diagnosed with acute coronary syndrome (ACS) including ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina, who were admitted to Karsa Husada Hospital, Batu, Indonesia, between January and July 2025. The study was conducted in accordance with the principles of the Declaration of Helsinki and received approval from the Health Research Ethics Committee, Faculty of Medicine and Health Sciences, Universitas Islam Negeri Maulana Malik Ibrahim Malang (Approval No. 60/01/EC/KEPK-FKIK/10/2024). Patient confidentiality was maintained, and informed consent was waived due to the retrospective study design.

Eligible participants were adults aged ≥18 years with a confirmed ACS diagnosis established by a cardiologist. Inclusion criteria required available FBG within 24 hours of admission after 8–12 hours of fasting and cTnI measurement during the same period. Exclusion criteria included acute metabolic emergencies (ketoacidosis, hyperosmolar syndrome), ongoing high-dose corticosteroid therapy, type 2 myocardial infarction or non-ischemic myocardial injury (such as myocarditis or sepsis), end-stage renal disease without estimated GFR data, and incomplete key variables.

The primary exposure variable was fasting blood glucose (FBG), evaluated both as a continuous variable (per 10 mg/dL increase) and categorically as normal (<100 mg/dL), prediabetes (100–125 mg/dL), and hyperglycemia/diabetes (≥126 mg/dL or previously diagnosed diabetes). The primary outcome was serum cardiac troponin I (cTnI), which was log10-transformed to approximate a normal distribution. Covariates included demographic characteristics, comorbidities, cardiovascular risk factors, and renal function [estimated glomerular filtration rate (eGFR) calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation], along with admission parameters (blood pressure, heart rate, Killip class), ACS subtype, treatment strategy, medication use, and HbA1c when available. Data were extracted from electronic medical records and laboratory databases, entered into standardized forms and verified by random audits for accuracy.

Continuous variables were summarized as mean ± standard deviation (SD) or median with interquartile range (IQR), whereas categorical variables were expressed as counts and percentages. Group comparisons were performed using ANOVA or the Kruskal–Wallis test for continuous data, and chi-square or Fisher's exact test for categorical data. The association between FBG and cTnI was examined using Spearman's correlation and multivariable linear regression, with results reported as β-coefficients per 10 mg/dL increase in FBG. Additional analyses included trend tests across FBG categories, restricted cubic spline modeling to assess potential non-linear relationships, and subgroup analyses stratified by diabetes status, ACS subtype, and renal function. Sensitivity analyses were conducted using peak cTnI, exclusion of outliers, and multiple imputation for missing data. A two-sided p-value <0.05 was considered statistically significant.

RESULTS

The clinical and biochemical characteristics of ACS patients by FBG categories are summarized in **Table 1**. The median age was 62 years (IQR 56.5–68.5), with males comprising 64.0%. The prevalence of diabetes increased markedly from 8.0% in the normal group, 13.0% in the prediabetes group, to 51.9% in the hyperglycemia group. Blood pressure and admission heart rate were comparable across categories. Median FBG was 115 mg/dL overall, with the highest values in the hyperglycemia group (164 mg/dL). Similar trends were observed for 2h-PPG and random glucose.

Lipid profiles, including total cholesterol, triglycerides, and Low-Density Lipoprotein (LDL), were largely similar, while High-Density Lipoprotein (HDL) was slightly lower in the prediabetes group. cTnI levels were higher in the hyperglycemia group (0.5 ng/mL; IQR 0.1–2.3) compared with the other groups. Renal function markers, including urea, creatinine, and eGFR, showed no meaningful differences.

Table 1. Clinical, biochemical, and demographic characteristics of patients with ACS according to FBG categories

Variable	Total (n=75)	Normal <100 (n=26)	Prediabetes 100–125 (n=22)	Hyperglycemia ≥126/DM (n=27)
Age (years)	62.0 (56.5–68.5)	59.0 (55.0–65.0)	64.0 (57.0–74.5)	64.0 (59.0–69.0)
Male (%)	64%	68.00%	65.20%	59.30%
Diabetes (%)	25.30%	8.00%	13.00%	51.90%
Systolic blood pressure (mmHg)	161.0 (137.0–175.5)	154.0 (138.0–168.0)	159.0 (135.0–164.5)	167.0 (138.5–180.5)
Diastolic blood pressure (mmHg)	98.0 (86.5–110.0)	98.0 (86.0–109.0)	96.0 (88.0–108.5)	102.0 (86.5–110.0)
Heart rate (beats/minute)	88.0 (79.5–98.5)	95.0 (84.0–99.0)	88.0 (73.5–103.0)	87.0 (80.5–97.0)
FBG (mg/dL)	115.0 (96.5–138.5)	88.0 (78.0–96.0)	115.0 (105.5–119.0)	164.0 (136.0–201.0)
2h-PPG (mg/dL)	140.0 (129.5–172.5)	139.0 (113.0–140.0)	137.0 (118.0–141.5)	198.0 (142.5–233.5)
RBG (mg/dL)	128.0 (110.0–155.0)	118.0 (92.0–128.0)	128.0 (114.0–129.5)	153.0 (128.0–195.0)
Total cholesterol (mg/dL)	164.0 (138.0–181.5)	164.0 (136.0–196.0)	168.0 (131.0–203.5)	164.0 (144.0–171.0)
Triglycerides (mg/dL)	98.0 (79.0–130.5)	98.0 (88.0–131.0)	104.0 (92.0–127.5)	92.0 (75.0–133.0)
LDL (mg/dL)	99.4 (77.2–122.8)	99.4 (79.0–131.0)	107.7 (82.2–125.5)	99.4 (74.0–115.3)
HDL (mg/dL)	36.0 (31.0–45.3)	36.0 (26.0–44.5)	35.0 (29.0–39.7)	37.3 (34.3–46.4)
cTnI (ng/mL)	0.1 (0.0–0.8)	0.2 (0.1–0.4)	0.1 (0.0–0.2)	0.5 (0.1–2.3)
Urea (mg/dL)	27.9 (23.0–46.8)	26.0 (19.1–47.2)	27.9 (24.9–43.4)	30.0 (23.6–48.5)
Creatinine (mg/dL)	0.9 (0.6–1.1)	0.9 (0.7–1.2)	0.9 (0.7–1.1)	0.8 (0.5–1.2)
eGFR (mL/min/1,73m^2)	84.4 (61.1–115.5)	86.6 (60.6–115.6)	75.7 (66.7–93.7)	85.9 (57.1–132.3)

***. Note:** FBG = Fasting Blood Glucose; 2h-PPG = 2-hour Postprandial Glucose; RBG = Random Blood Glucose; LDL = Low-Density Lipoprotein; HDL = High-Density Lipoprotein; cTnI = cardiac Troponin I; eGFR = Estimated Glomerular Filtration Rate.

In the correlation analysis, FBG was weakly positively correlated with cTnI ($r_s \approx 0.19$; $p \approx 0.12$). In linear regression, each 10 mg/dL increase in FBG was associated with an 11.2% increase in troponin in the univariate model (β=0.046; 95% CI −0.2% to 9.4%; p=0.059), becoming 9.8% after adjusting for age and sex (β = 0.041; p = 0.086), and 8.6% in the fully adjusted model (β = 0.036; 95% CI −4.5% to 9.2%; p = 0.204) as shown in **Table 2.**

Categorical analysis **(Table 3)** showed that, compared to the normal group, the prediabetes group (100–125 mg/dL) tended to have lower troponin levels by ~58% (β = −0.376; p = 0.266), while the hyperglycemia group ≥126 mg/dL tended to have higher levels by ~51% (β = 0.178; p=0.602); the p-trend between categories was ≈ 0.55. Overall, the direction of the relationship consistently indicated that higher FBG was associated with higher troponin levels, though this did not reach statistical significance after full adjustment—suggesting potential limitations in the sample size.

Table 2. Stepwise linear regression between FBG and log10 troponin I

Model	% change (per 10 mg/dL)	β per 10 mg/dL (log10)	95%CI β low	95%CI β high	p-value
Univariate	11.232	0.046	-0.002	0.094	0.059
+ Age & Sex	9.825	0.041	-0.006	0.087	0.086
+ All variables	8.618	0.036	-0.020	0.092	0.204

***. Note:** p-values are shown for the univariate model, the model adjusted for age and sex, and the fully adjusted model including all variables (age, sex, DM, eGFR, systolic blood pressure, heart rate).

Table 3. Comparison of cTnI levels (log10) according to FBG categories, with normal group (<100 mg/dL) as reference

Category vs normal	% difference	β (log10)	95%CI low	95%CI high	p-value
Prediabetes 100–125	-57.972	-0.376	-1.047	0.294	0.266
Hyperglycemia ≥126/DM	50.611	0.178	-0.502	0.857	0.602

Dose–response analysis using restricted cubic splines after adjustment (age, sex, DM, eGFR, systolic blood pressure, and pulse rate) showed that the relationship between FBG and cTnI levels appeared to be nearly linear across most of the observation range. Increases in FBG were associated with a smooth increase in troponin levels without a clear inflection point; tests for deviation from linearity were not significant (p-nonlinear = 0.72). Estimation uncertainty widened at very high FBG levels, consistent with fewer observations in that range **(Figure 1).**

In subgroup analysis, the direction of association was similar in patients without diabetes and those with diabetes. Each 10 mg/dL increase in FBG was associated with an estimated 17% increase in troponin in the non-DM group (p = 0.17) and a 10% increase in the DM group (p = 0.48). The interaction test did not show a significant difference in effect between the two subgroups (p-interaction = 0.67), indicating that diabetes status does not modify the FBG–troponin relationship in this sample **(Figure 2).**

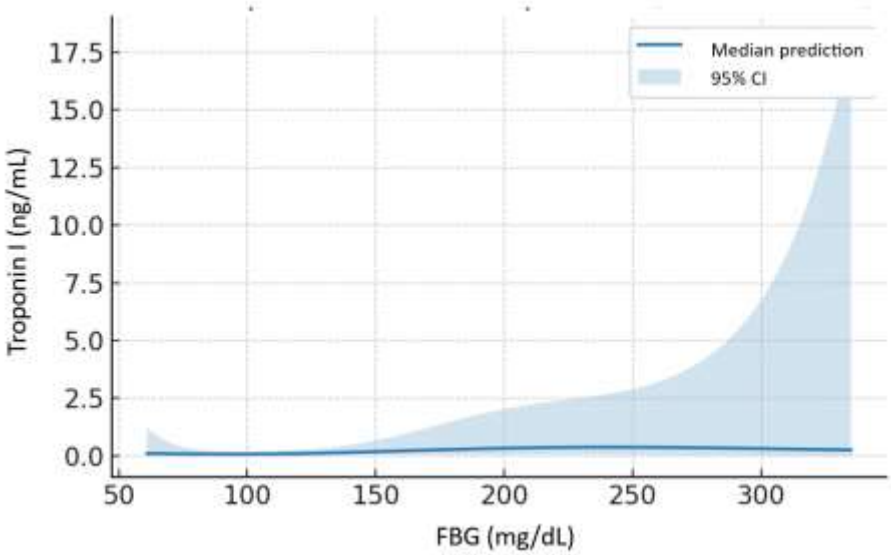

Figure 1. Spline curve of the relationship between FBG and cTnI levels predicted from an adjusted model (age, gender, DM, eGFR, systolic blood pressure, pulse). The line shows the median cTnI prediction (log10 reciprocal value), and the shaded area shows the 95% CI.

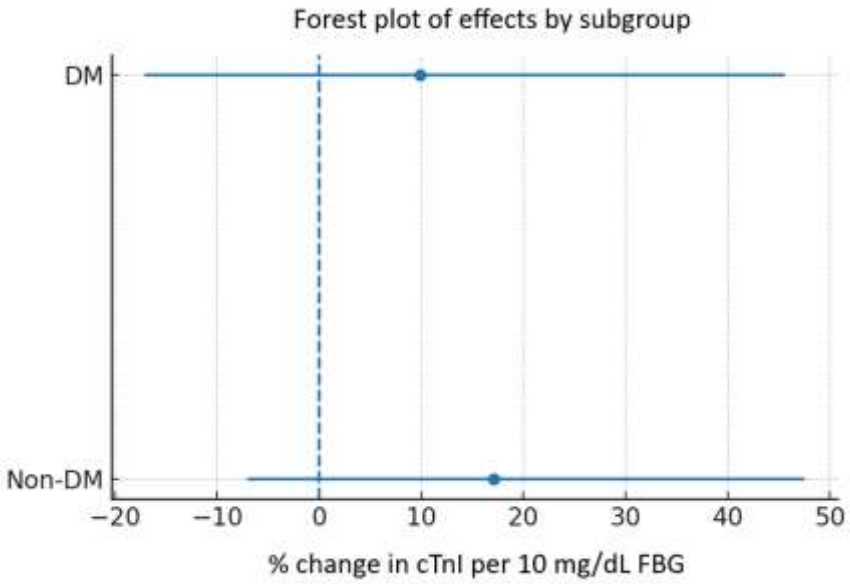

Figure 2. Forest plot of effects by subgroup for the association between FBG and cTnI. Points indicate the % change in cTnI per 10 mg/dL increase in FBG with 95% CI (horizontal lines), adjusted for the same covariates; dashed vertical lines mark zero (no effect). There was no significant difference in effect between subgroups (p-interaction DM×FBG = 0.67).

Table 4. Clinical Outcomes by Fasting Blood Glucose (FBG) Category

Category	Subcategory	Normal (<100)	Prediabetes (100–125)	Hyperglycemia (≥126)	Total (%)	p-value
Length of Stay	≤ 3 days	15 (57.7%)	9 (40.9%)	14 (51.9%)	38 (50.7%)	**0.266**
	> 3 days	11 (42.3%)	13 (59.1%)	13 (48.1%)	37 (49.3%)	
Complications	ADHF	6 (23.1%)	5 (22.7%)	7 (25.9%)	18 (24.0%)	**0.988**
	AF	3 (11.5%)	2 (9.1%)	4 (14.8%)	9 (12.0%)	
	PVC	2 (7.7%)	3 (13.6%)	3 (11.1%)	8 (10.7%)	
	RBBB / AV Block	4 (15.4%)	3 (13.6%)	3 (11.1%)	10 (13.3%)	
Comorbidities	Diabetes Mellitus	0 (0.0%)	4 (18.2%)	27 (100.0%)	31 (41.3%)	**0.999**
	Hypertensive Heart Disease	3 (11.5%)	2 (9.1%)	3 (11.1%)	8 (10.7%)	
	Valvular Disease	2 (7.7%)	1 (4.5%)	2 (7.4%)	5 (6.7%)	
Major Cardiovascular Events	STEMI	10 (38.5%)	8 (36.4%)	11 (40.7%)	29 (38.7%)	—
	NSTEMI	16 (61.5%)	14 (63.6%)	16 (59.3%)	46 (61.3%)	—

The Table 4 demonstrates that among 75 ACS patients, those with normal, prediabetic, and hyperglycemic fasting blood glucose (FBG) levels showed comparable clinical outcomes. Longer hospital stay (>3 days) was more common in the prediabetes group (59.1%) compared to normal (42.3%) and hyperglycemia (48.1%) groups (p=0.266). Complications such as ADHF occurred in 24.0% of patients, with similar frequencies across groups (p=0.988). Diabetes mellitus was present in 100% of the hyperglycemia group, while hypertensive heart disease (10.7%) and valvular disease (6.7%) were evenly distributed. The proportion of STEMI and NSTEMI was also comparable across categories. Although the differences were not statistically significant, these findings highlight that elevated FBG likely reflects acute metabolic stress rather than directly determining adverse in-hospital outcomes providing clinically relevant insight into the metabolic profile of ACS patients.

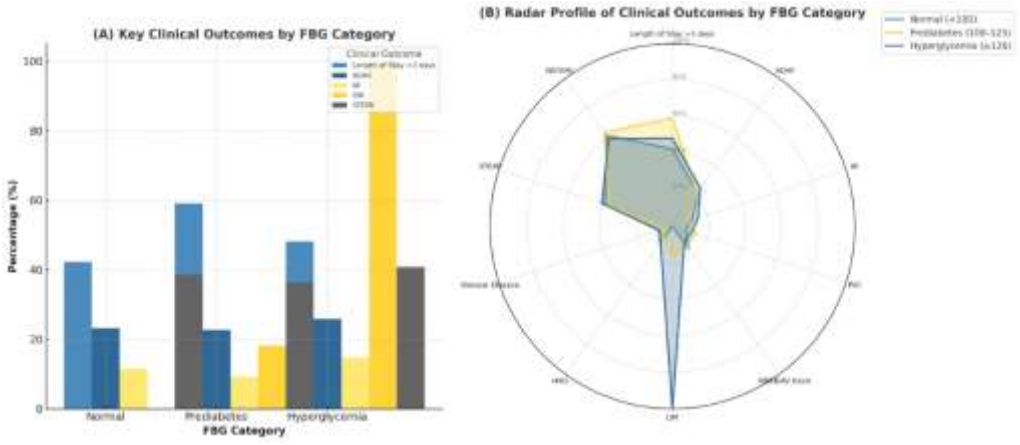

Figure 3. (A) Distribution of key in-hospital clinical outcomes across fasting blood glucose (FBG) categories. **(B)** Radar chart illustrating overlapping clinical profiles among FBG groups, confirming that differences were not statistically significant (all p > 0.05). These findings suggest that fasting glucose levels mainly reflect acute metabolic stress rather than directly determining adverse clinical outcomes in ACS patients.

Figure 3. illustrates the distribution of key in-hospital clinical outcomes across fasting blood glucose (FBG) categories. As shown in Panel A, longer hospital stay (>3

days) was slightly more frequent in the prediabetes group (59.1%) compared to the normal (42.3%) and hyperglycemia (48.1%) groups, while complication rates such as ADHF (23–26%) and STEMI incidence (38–41%) were similar across all categories. Panel B shows overlapping radar profiles, confirming no significant differences (all $p > 0.05$). These findings suggest that elevated FBG primarily reflects acute metabolic stress rather than directly determining adverse outcomes in ACS patients.

DISCUSSION

This study evaluated the relationship between fasting blood glucose (FBG) and cTnI in ACS patients at Karsa Husada Hospital and found a consistent but weak positive association. Although patients with hyperglycemia had higher median troponin levels and each 10 mg/dL increase in FBG corresponded to an estimated 8.6% rise in troponin, the correlations and regression analyses did not reach statistical significance after adjustment for clinical factors ($r_s = 0.19$, $p = 0.12$; $\beta = 0.036$, $p = 0.20$; p-trend = 0.55). Dose–response and subgroup analyses showed a largely linear pattern across the FBG range, with no evidence of a threshold effect or effect modification by diabetes status, suggesting that while higher FBG may be biologically linked to greater myocardial injury, the effect is small and likely overshadowed by other clinical determinants.

In addition to biochemical parameters, clinical outcomes were analyzed across FBG categories. Although patients in the prediabetes group had a higher proportion of prolonged hospitalization (>3 days; 59.1%) compared with those in the normal (42.3%) and hyperglycemia (48.1%) groups, the differences were not statistically significant ($p = 0.266$). Similarly, the incidence of complications, including ADHF (23–26%), arrhythmias, and STEMI (38–41%), was comparable among all FBG categories (Table 4; Fig. 3A,B). These findings indicate that elevated FBG at admission primarily reflects an acute metabolic response rather than serving as a direct determinant of short-term adverse outcomes in patients with ACS.

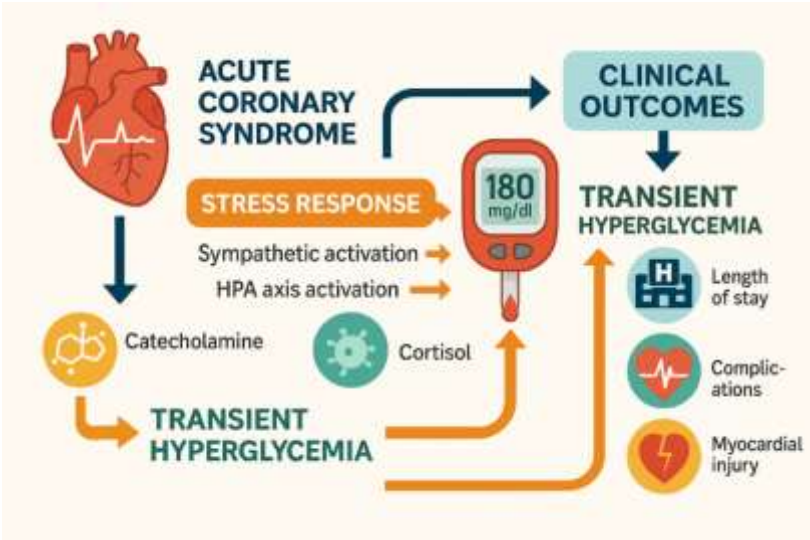

Figure 4. Pathophysiological mechanism illustrating that stress-induced transient hyperglycemia in acute coronary syndrome reflects acute metabolic response rather than a direct cause of adverse clinical outcomes.

Pathophysiologically, acute hyperglycemia can increase oxidative stress, endothelial dysfunction, and prothrombotic activity, contributing to microvascular obstruction, larger infarct size, and higher troponin release.[14] At the molecular level, excessive glucose triggers the formation of advanced glycation end-products (AGEs) and activation of the NF-κB pathway, which can damage cardiomyocyte membranes and thereby accelerate the release of cTnI into the circulation.[15] On the other hand, FBG in the early phase of treatment may reflect hyperglycemic stress due to surges in catecholamines and cortisol, which may more accurately reflect the acute disease stress response than chronic glucose exposure.[16] In this context, the FBG–troponin association may be reduced after adjusting for severity markers (e.g., blood pressure, heart rate) and kidney function (eGFR), as observed in our fully adjusted model.

Furthermore, during acute coronary syndrome, sympathetic nervous system activation and stimulation of the hypothalamic–pituitary–adrenal (HPA) axis lead to elevated catecholamine and cortisol levels, which transiently increase hepatic glucose output and impair insulin sensitivity [20,21]. This physiological response aims to provide sufficient metabolic energy to the ischemic myocardium and vital organs during stress [22]. Consequently, stress-induced hyperglycemia in ACS likely reflects an acute neurohormonal and inflammatory response mediated by catecholamines, cortisol, and cytokine release rather than chronic metabolic dysfunction. This transient elevation in glucose levels may therefore represent an adaptive mechanism to acute stress rather than a determinant of adverse in-hospital outcomes[23,24]. This stress-mediated hyperglycemic response is therefore more reflective of acute neurohormonal activation than of long-term glycemic control, aligning with recent evidence that transient stress hyperglycemia is a reversible, compensatory process rather than a direct driver of poor cardiovascular prognosis [14,23].

Some studies report a positive correlation, for example, Montebello's study of 266 AMI patients in Malta showed that glucose on admission correlated with peak troponin (without diabetes: $r = 0.397$; $p = 0.0001$; with diabetes: $r = 0.29$; $p = 0.004$), and remained significant in multivariate analysis ($\beta = 0.28$; $p = 0.006$ and $\beta = 0.30$; $p = 0.003$).[17] Similar findings were reported in a prospective study in India involving 80 AMI patients, where FBG was positively associated with cTnI ($r = 0.50$; $p < 0.01$) and higher cTnI levels in STEMI compared to NSTEMI (4.2 ± 1.5 vs 2.5 ± 1.2 ng/mL; $p < 0.01$).[18] Additionally, Oktabelia's study in Indonesia noted that the correlation between FBG and cTnI was very weak and not significant ($r = 0.026$; $p = 0.88$), emphasizing the role of ischemia duration, time to reperfusion, and therapeutic strategies as the primary determinants of troponin levels.[19] Our findings, which did not reach significance after adjustment, suggest that clinical factors related to "time" (onset of pain–sample, reperfusion) and ACS phenotype likely have a more dominant role than FBG alone in the early phase.

In this cohort, patients with hyperglycemia (≥126 mg/dL) exhibited a numerically higher proportion of prolonged hospitalization (>3 days; 48.1%) compared with those in the normal (42.3%) and prediabetes (59.1%) groups, although these differences did not reach statistical significance ($p = 0.266$). Consistent findings from multicenter studies have shown that patients with admission hyperglycemia during acute coronary syndrome typically experience prolonged hospitalization compared with normoglycemic counterparts [24,25]. Likewise, a recent meta-analysis involving more than 87,000 patients with acute

myocardial infarction demonstrated that an elevated stress hyperglycemia ratio (SHR) was independently associated with higher in-hospital and long-term mortality, supporting the notion that increased glucose levels at admission primarily reflect acute illness severity and neurohormonal stress rather than chronic metabolic dysfunction [26].

The distribution of in-hospital complications, including acute decompensated heart failure (ADHF), atrial fibrillation (AF), and conduction disturbances such as RBBB or AV block, was similar across FBG groups ($p = 0.988$), and major cardiovascular events (STEMI vs. NSTEMI) also showed no significant variation. Our observations align with previous large-cohort analyses by Yang et al. (2022) and Chen et al. (2023), where stress-related hyperglycemia was identified as a marker of acute illness severity and adverse prognosis rather than as an independent predictor of distinct arrhythmic or mechanical complications. Several studies have suggested that stress hyperglycemia is more reflective of neurohormonal and inflammatory activation than of structural myocardial injury (Vedantam et al., 2022; Chen et al., 2023). The comparable complication rates across glycemic strata in our cohort further support the interpretation that acute glucose elevation represents a physiological stress marker rather than a pathological driver of poor short-term outcomes in ACS.

This study has several strengths, including the use of routine laboratory data in a "real-world" population that reflects everyday clinical practice, pre-specified analysis with log transformation and non-linearity assessment that enhances methodological validity, and adjustment for relevant clinical covariates including eGFR. FBG measurement within 24 hours of admission provides a representative picture of the acute state, while the use of standard biochemical parameters (glucose, lipids, troponin) facilitates comparability with other studies. These findings have practical implications for internists by reinforcing cardiovascular risk stratification in patients with acute hyperglycemia and underscoring the need to monitor renal function and cardiac biomarkers from the onset of care. Several limitations should be acknowledged, such as a relatively small sample size, a retrospective single-center design, variability in measurements, and the absence of HbA1c data necessary for calculating the stress hyperglycemia ratio. Therefore, the findings should be interpreted with caution and confirmed in larger prospective multicenter studies with more comprehensive assessments.

Beyond these methodological strengths, the present study provides novel clinical insight that extends its translational relevance to daily cardiometabolic practice. By integrating fasting blood glucose with short-term clinical outcomes such as hospital stay, complications, and cardiovascular comorbidities, this analysis bridges metabolic and cardiovascular perspectives in acute coronary care. For internists, these findings emphasize the value of early FBG assessment as an accessible marker of systemic stress and hemodynamic burden during acute illness. For cardiologists, they highlight that elevated admission glucose should be interpreted in the context of stress hyperglycemia rather than irreversible myocardial injury. Collectively, these observations underscore the importance of multidisciplinary evaluation in ACS management, supporting a shift from isolated biomarker interpretation toward integrated cardio–metabolic risk assessment.

CONCLUSION

In summary, among patients with ACS, higher FBG levels were modestly associated with cTnI, but the relationship lost significance after adjustment for key clinical factors. Clinical outcomes including length of stay, complications, and major cardiovascular events did not differ significantly across FBG categories. These findings suggest that elevated FBG reflects transient stress-related metabolic responses rather than a direct determinant of myocardial injury or in-hospital prognosis.

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