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# Impact of Glycemic Gap on Early Adverse Events in Thrombolysed STEMI Patients: A Risk-Adjusted Analysis

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## Abstract

**Background:** *ST-segment elevation myocardial infarction (STEMI) represents the most severe and urgent form of ischemic heart disease (IHD), resulting in significant illness and death. The purpose of the study is to assess the prognostic significance of glycemic gap in predicting early adverse outcomes among thrombolysed STEMI patients.*

**Aim of the study:** *The aim of the study was to evaluate the prognostic significance of glycemic gap in predicting early adverse outcomes among thrombolysed STEMI patients using a risk-adjusted analytical approach.*

**Methods:** *This cross-sectional observational study was conducted at the Department of Cardiology, Mymensingh Medical College Hospital, Bangladesh (October 2019–March 2021), including 287 first-onset STEMI patients thrombolysed with streptokinase within 12 hours. Participants were grouped by glycemic gap (>40 mg/dL vs. ≤40 mg/dL), calculated from admission glucose and HbA1c. Data included clinical assessment, ECGs, echocardiography, and labs. Outcomes were recorded. Analysis was done using SPSS v20.0; $p < 0.05$ was considered significant.*

**Results:** *Among 287 thrombolysed STEMI patients, those with a glycemic gap >40 mg/dl (Group I) had higher rates of heart failure (60.2% vs. 28.3%), cardiogenic shock (35.2% vs. 17.8%), and mortality (13.3% vs. 5.7%) compared to those with ≤40 mg/dl (Group II). Group I also had significantly higher admission glucose, glycemic gap, and lower ejection fraction. Glycemic gap was an independent predictor of heart failure in both unadjusted (OR 7.84, $p = 0.005$) and adjusted (OR 2.75, $p = 0.007$) models.*

**Conclusion:** *A higher glycemic gap independently predicts early in-hospital adverse outcomes, including heart failure, in thrombolysed STEMI patients.*

**Keywords:** *Glycemic Gap, Stress Hyperglycemia, STEMI.*

## 1. Introduction

ST-segment elevation myocardial infarction (STEMI) represents the most severe and urgent form of ischemic heart disease (IHD), resulting in significant illness and death [1]. Prompt reperfusion therapies, including thrombolysis and percutaneous coronary intervention (PCI), have been shown to substantially enhance outcomes by preserving heart muscle viability and reducing the extent of infarction, and are endorsed by current clinical guidelines [2]. Despite advances in these treatments, accurate risk stratification continues to be critical for predicting prognosis in STEMI patients, especially in environments where thrombolytic therapy with agents such as streptokinase remains the mainstay due to limited availability of PCI.

Stress hyperglycemia (SH) refers to a temporary elevation in blood glucose levels during acute physiological stress and has been associated with worse clinical outcomes, especially in cardiovascular diseases [3]. Acute myocardial infarction (AMI) is strongly linked to SH and its negative consequences, irrespective of whether the patient has preexisting diabetes [4,5]. In critically ill individuals, increased secretion of hormones like cortisol, catecholamines, and glucagon contributes to stress-induced hyperglycemia (SIH) [6]. While admission blood glucose (ABG) levels have been used in some studies as an indicator of SIH to predict adverse event risk, ABG is also affected by a patient's chronic glycemic control, which limits its accuracy in representing the acute stress response [7,8].

To address this limitation, the glycemic gap (GG)—calculated by adjusting admission blood glucose (ABG) for chronic glycemic status using glycated hemoglobin (HbA1c)—has been introduced as a more accurate marker of stress-induced hyperglycemia (SIH) [9-11]. The GG is determined by subtracting the A1c-derived average glucose (ADAG) from the measured blood glucose level, thereby minimizing the influence of chronic hyperglycemia on disease severity assessment. Studies have shown that GG offers improved accuracy over ABG alone in evaluating SIH and serves as a more reliable prognostic indicator, as it better captures the acute metabolic stress related to STEMI while reducing confounding from long-term glycemic control.

Despite emerging evidence on the prognostic value of glycemic gap in various critical illnesses, its application in the context of STEMI—particularly among patients receiving thrombolytic therapy—remains underexplored. Most studies have focused on admission glucose or HbA1c independently, without adequately accounting for their combined influence through glycemic gap. Additionally, there is limited data from low- and middle-income countries where thrombolysis is the primary reperfusion strategy. The purpose of the study is to assess the prognostic significance of glycemic gap in predicting early adverse outcomes among thrombolysed STEMI patients using a risk-adjusted analytical approach.

## 2. Objective

- To evaluate the prognostic significance of glycemic gap in predicting early adverse outcomes among thrombolysed STEMI patients using a risk-adjusted analytical approach.

## 3. Methodology & Materials

This cross-sectional observational study enrolled 287 patients with first-onset ST-elevation myocardial infarction (STEMI) who received streptokinase thrombolysis within 12 hours of symptom onset at the Department of Cardiology, Mymensingh Medical College Hospital, Bangladesh, between October 2019 and March 2021. Participants were stratified by glycemic gap (Group I: >40 mg/dL; Group II: ≤40 mg/dL).

### 3.1. Inclusion Criteria

- First attack of acute ST-elevation myocardial infarction (STEMI)
- Thrombolysed with streptokinase
- Presented within 12 hours of onset of chest pain

### 3.2. Exclusion Criteria

- Previous history of myocardial infarction
- History of percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG)
- Chronic obstructive pulmonary disease (COPD), primary pulmonary hypertension, or cor pulmonale
- Valvular heart disease, congenital heart disease, or dilated cardiomyopathy
- Chronic renal failure
- Heart failure
- Unwillingness to participate in the study

Of 325 initially screened patients, 38 were excluded due to referral, early death, or incomplete investigations. Thus, a total of 287 patients meeting eligibility criteria were included in the final analysis. Data were collected using a structured case record form, encompassing clinical history, physical examination, serial 12-lead ECGs (performed twice daily), and continuous rhythm monitoring. Laboratory investigations included admission blood glucose, HbA1c, troponin I, lipid profile, and electrolytes. Estimated average glucose (eAG) was calculated using the formula $eAG = (28.7 \times HbA1c) - 46.7$, and glycemic gap was defined as the difference between admission blood glucose and eAG.

Echocardiography was performed 48–72 hours after admission to assess left ventricular ejection fraction (LVEF). Patients were monitored throughout their CCU stay for in-hospital adverse events including heart failure (Killip class II–IV), cardiogenic shock, arrhythmias, conduction defects, and mortality.

### 3.3. Statistical Analysis

Continuous variables were expressed as mean ± standard deviation (SD) and compared using unpaired t-tests. Categorical variables were presented as frequencies and percentages, analyzed using chi-square tests. Multivariable logistic regression was performed to assess the independent association between glycemic gap and heart failure, adjusting for age, systemic hypertension, and diabetes mellitus. A p-value of $<0.05$ was considered statistically significant. All analyses were conducted using SPSS version 20.0.

### 3.4. Ethical Considerations

Ethical clearance was obtained from the Institutional Review Board of Mymensingh Medical College. Written informed consent was secured from all participants, and strict confidentiality of patient data was maintained throughout the study.

## 4. Results

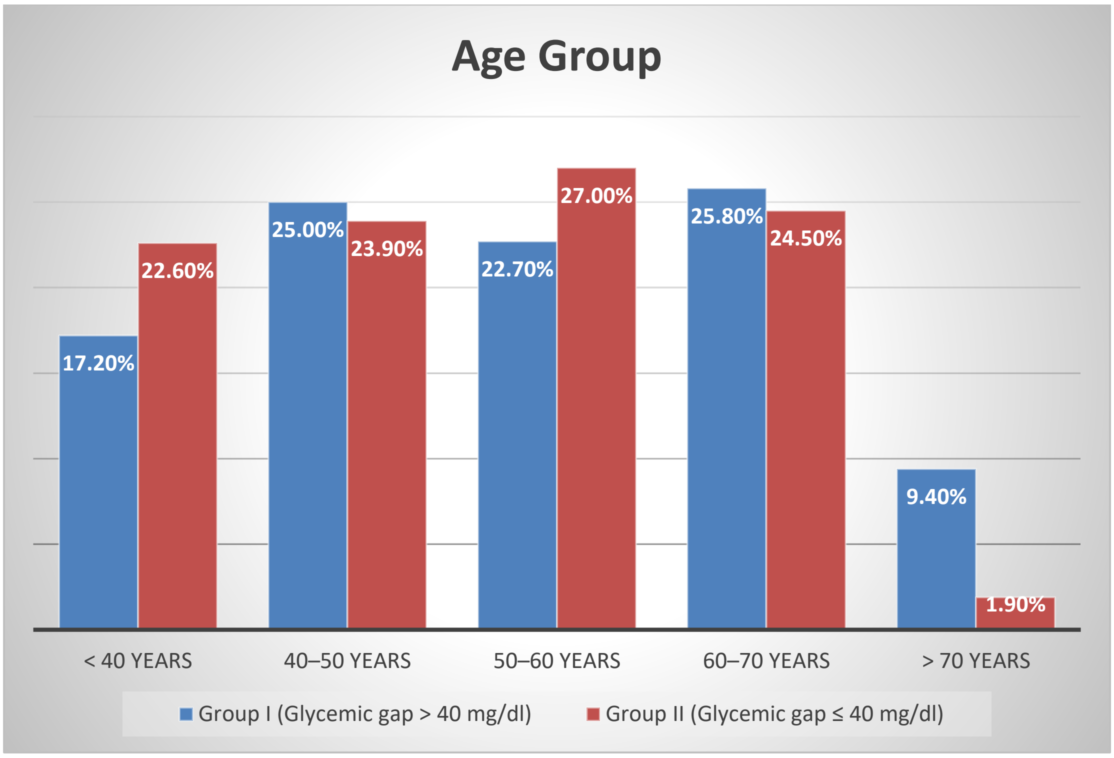


**Figure 1.** *Distribution of Age Group among Study Population (n = 287)*

Figure 1 shows the percentage distribution of age groups in the study population, divided into two groups based on glycemic gap: Group I (glycemic gap > 40 mg/dl) and Group II (glycemic gap ≤ 40 mg/dl). In Group I, the highest proportion of patients fell within the 60–70 year age group (25.8%), whereas in Group II, the 50–60 year group was most common (27.0%). A notable difference was observed in the >70 years category, with 9.4% in Group I compared to only 1.9% in Group II. The mean age was slightly higher in Group I (55.36 ± 13.28 years) than in Group II (53.06 ± 11.64 years), though the difference was not statistically significant ($p = 0.070$).

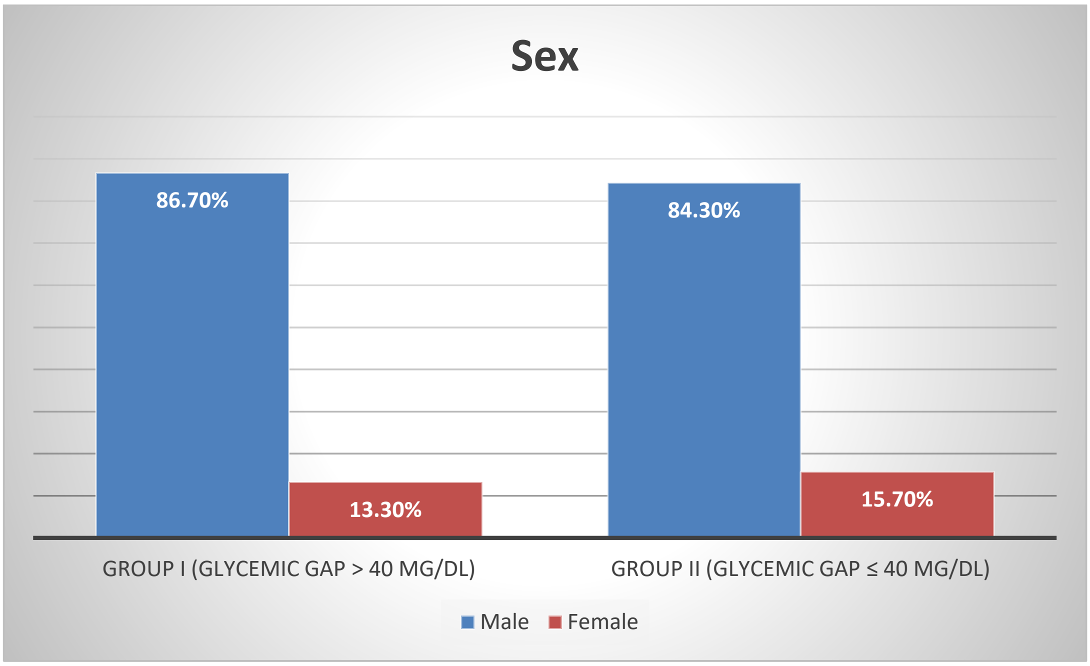


**Figure 2.** *Distribution of Sex among Study Population (n = 287)*

Figure 2 illustrates the distribution of sex among the study population divided by glycemic gap groups. In Group I (glycemic gap > 40 mg/dl, *n = 128*), 111 patients (86.7%) were male and 17 (13.3%) were female. In Group II (glycemic gap ≤ 40 mg/dl, *n = 159*), 134 patients (84.3%) were male and 25 (15.7%) were female. Overall, both groups demonstrated a clear male predominance, with slightly higher male representation in Group I.

**Table 1.** *Types of AMI among the Study Population (n = 287)*

| Site of STEMI | Group I (Glycemic gap > 40 mg/dl) | | Group II (Glycemic gap ≤ 40 mg/dl) | | P-value |
|---|---|---|---|---|---|
| | n | % | n | % | |
| Anterior | 41 | 32.0% | 42 | 26.4% | 0.867 |
| Extensive Anterior | 17 | 13.3% | 23 | 14.5% | |
| Anteroseptal | 14 | 10.9% | 18 | 11.3% | |
| Lateral | 13 | 10.2% | 14 | 8.8% | |
| Inferior | 29 | 22.7% | 45 | 28.3% | |
| Inferior + RV Infarction | 14 | 10.9% | 17 | 10.7% | |

Table 1 shows the distribution of AMI types by glycemic gap groups. In Group I (>40 mg/dl), anterior AMI was most common (32.0%), followed by inferior AMI (22.7%). In Group II (≤40 mg/dl), inferior AMI predominated (28.3%) followed by anterior AMI (26.4%). Other AMI types were similarly distributed. Differences between groups were not statistically significant (p = 0.867).

**Table 2.** *Comparison of the Baseline Characteristics between Groups of the Study Population (n = 287)*

| Parameter | Group I (Glycemic gap > 40 mg/dl) | Group II (Glycemic gap ≤ 40 mg/dl) | P Value |
|---|---|---|---|
| Pulse (beats/min) | 72.02 ± 24.75 | 75.25 ± 18.45 | 0.223 |
| Systolic BP (mmHg) | 112.86 ± 37.86 | 115.16 ± 28.14 | 0.569 |
| Diastolic BP (mmHg) | 72.78 ± 24.91 | 74.65 ± 18.19 | 0.478 |
| Troponin I (ng/ml) | 17.81 ± 14.52 | 14.64 ± 14.03 | 0.064 |
| Admission Blood Glucose (mmol/L) | 10.44 ± 4.35 | 6.59 ± 1.46 | 0.001 |
| HbA1c (%) | 6.51 ± 5.60 | 5.35 ± 1.09 | 0.340 |
| Estimated Average Glucose (EAG) | 105.99 ± 39.81 | 105.27 ± 31.13 | 0.867 |
| Glycemic Gap (mg/dl) | 85.86 ± 54.10 | 13.49 ± 23.25 | 0.001 |

| | | | |
|---|---|---|---|
| Total Cholesterol (mg/dl) | 168.50 ± 36.81 | 163.69 ± 25.87 | 0.214 |
| HDL (mg/dl) | 32.51 ± 6.31 | 32.23 ± 5.30 | 0.690 |
| LDL (mg/dl) | 109.14 ± 27.57 | 105.12 ± 20.25 | 0.171 |
| Triglycerides (mg/dl) | 133.57 ± 56.35 | 133.02 ± 53.33 | 0.933 |
| Sodium ($Na^+$, mmol/L) | 139.34 ± 4.26 | 137.99 ± 14.25 | 0.260 |
| Potassium ($K^+$, mmol/L) | 4.38 ± 0.59 | 5.15 ± 10.80 | 0.370 |
| Chloride ($Cl^-$, mmol/L) | 102.28 ± 8.21 | 102.18 ± 11.67 | 0.931 |
| Ejection Fraction (Echo %) | 45.44 ± 7.01 | 47.06 ± 6.38 | 0.044 |

Table 2 compares the baseline clinical and laboratory characteristics between Group I (glycemic gap > 40 mg/dl) and Group II (glycemic gap ≤ 40 mg/dl). Admission blood glucose and glycemic gap were significantly higher in Group I (p = 0.001 for both). Ejection fraction was significantly lower in Group I compared to Group II (p = 0.044). Other baseline parameters including pulse, blood pressure, troponin I, HbA1c, lipid profile, electrolytes, and estimated average glucose showed no statistically significant differences.

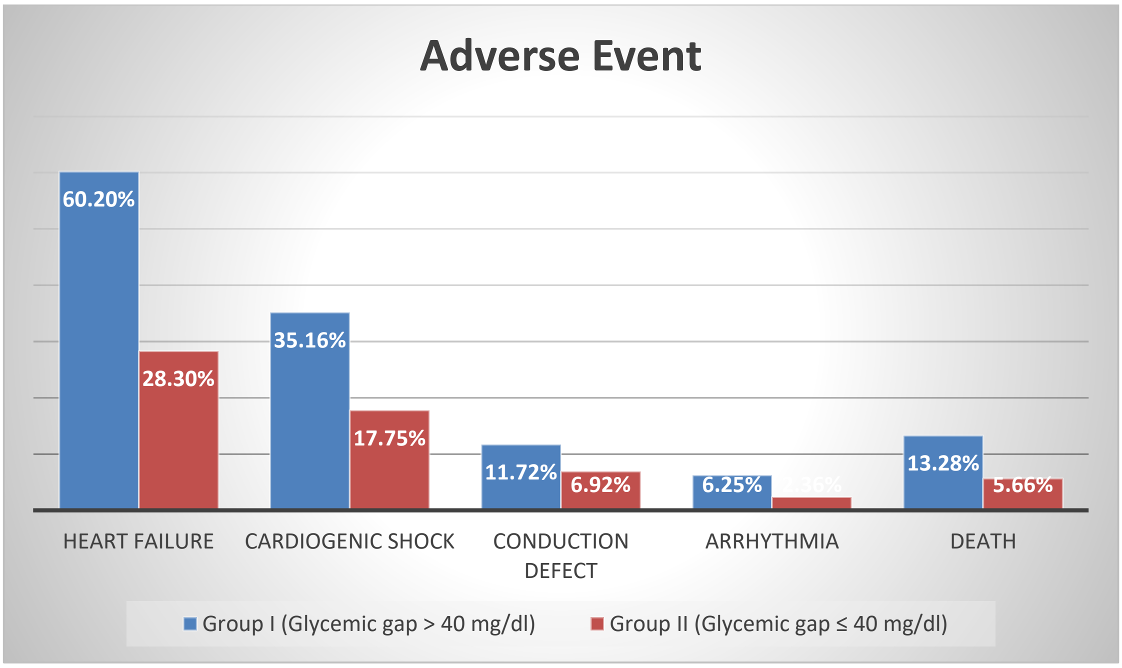


**Figure 3.** *In-Hospital Adverse Events among the Study Population (n = 287)*

Figure 3 illustrates the percentage of in-hospital adverse events in patients stratified by glycemic gap. Heart failure was the most frequent complication, occurring in 60.2% of Group I (glycemic gap > 40 mg/dl) compared to 28.3% in Group II. Cardiogenic shock was also significantly higher in Group I (35.16%) than in Group II (17.75%). In-hospital mortality was more than twice as high in Group I (13.28%) compared to Group II (5.66%). While conduction defects and arrhythmias were more common in Group I, the differences were not statistically significant.

**Table 3.** *Unadjusted Multivariate Logistic Regression for Predictors of Heart Failure*

| Variables | β | S.E. | P value | OR | 95% C.I. |
|---|---|---|---|---|---|
| Age | 0.011 | 0.010 | 0.269 | 1.221 | (0.991 – 1.031) |
| Systemic Hypertension | 0.471 | 0.265 | 0.051 | 3.157 | (0.953 – 2.693) |
| Diabetes Mellitus | -0.439 | 0.433 | 0.310 | 1.030 | (0.276 – 1.506) |
| Dyslipidemia | 0.040 | 0.270 | 0.882 | 0.022 | (0.613 – 1.767) |
| Admission Blood Glucose | -0.023 | 0.054 | 0.665 | 0.187 | (0.879 – 1.085) |
| HbA1c | -0.243 | 0.534 | 0.648 | 0.208 | (0.276 – 2.231) |
| EAG | -0.006 | 0.006 | 0.354 | 0.860 | (0.983 – 1.006) |
| **Glycemic Gap** | **0.893** | **0.319** | **0.005** | **7.837** | **(1.307 – 4.566)** |

Table 3 presents the results of unadjusted multivariate logistic regression analysis to identify predictors of heart failure among the study population. Among the variables analyzed, glycemic gap was found to be a statistically significant independent predictor of heart failure (β = 0.893, p = 0.005), with an odds ratio (OR) of 7.837 and a 95% confidence interval (CI) of 1.307–4.566. Other variables including age, hypertension, diabetes, dyslipidemia, admission blood glucose, HbA1c, and EAG did not show statistically significant associations in this unadjusted model.

**Table 4.** *Adjusted Multivariate Logistic Regression for Predictors of Heart Failure*

| Variables | β | S.E. | P value | OR | 95% C.I. |
|---|---|---|---|---|---|
| Age | 0.022 | 0.103 | 0.832 | 0.045 | (0.835 – 1.251) |
| Systemic Hypertension | 0.386 | 0.257 | 0.133 | 2.257 | (0.889 – 2.434) |
| Diabetes Mellitus | 0.378 | 0.410 | 0.356 | 0.853 | (0.654 – 3.259) |
| Dyslipidemia | -0.083 | 0.267 | 0.757 | 0.096 | (0.545 – 1.554) |
| Admission Blood Glucose | 0.485 | 0.298 | 0.404 | 2.646 | (0.905 – 2.911) |
| HbA1c | -0.686 | 0.452 | 0.129 | 2.304 | (0.208 – 1.221) |
| EAG | -0.012 | 0.294 | 0.968 | 0.002 | (0.556 – 1.758) |
| **Glycemic Gap** | **0.448** | **0.27** | **0.007** | **2.746** | **(1.060 – 2.657)** |

Table 4 displays the results of an adjusted multivariate logistic regression analysis assessing independent predictors of heart failure. After adjusting for potential confounders, glycemic gap remained a statistically significant predictor (β = 0.448, p = 0.007), with an odds ratio (OR) of 2.746 and a 95% confidence interval (CI) of 1.060–2.657. Other variables such as age, systemic hypertension, diabetes mellitus, dyslipidemia, admission blood glucose, HbA1c, and EAG did not show statistically significant associations in the adjusted model.

## 5. Discussion

This hospital-based cross-sectional observational study was conducted in the Department of Cardiology at Mymensingh Medical College Hospital over a period of 18 months, from October 1, 2019 to March 31, 2021. The study explored the prognostic significance of glycemic gap in predicting early adverse outcomes among thrombolysed STEMI patients using a risk-adjusted analytical approach. A total of 287 patients with first-onset ST-elevation myocardial infarction (STEMI), who received streptokinase within 12 hours of symptom onset, were selected based on specific inclusion and exclusion criteria. The patients were categorized into two groups: Group I with glycemic gap > 40 mg/dL and Group II with glycemic gap ≤ 40 mg/dL.

In this study of thrombolysed STEMI patients, those with a glycemic gap >40 mg/dL tended to be older, with a higher percentage in the 60–70 and >70 years age groups compared to patients with glycemic gap ≤40 mg/dL, though this difference was not statistically significant (p = 0.070). This observation is consistent with findings by Liao et al.[12], who reported that higher glycemic gaps were more common among older patients, suggesting age may be a contributing risk factor to stress hyperglycemia severity in acute STEMI. Supporting this, Garcia et al.[3] demonstrated that larger glucose delta values—reflecting greater glycemic gaps—in pharmacoinvasively treated STEMI patients were associated with older age, reduced left ventricular ejection fraction, and larger infarct size. These parallels reinforce the relevance of glycemic gap as a marker influenced by age and linked to worse cardiac outcomes in thrombolysed STEMI patients, underscoring the need for risk-adjusted evaluation in this population.

In this study, a clear male predominance was observed in both glycemic gap groups among thrombolysed STEMI patients, with males constituting 86.7% of Group I (glycemic gap >40 mg/dL) and 84.3% of Group II (glycemic gap ≤40 mg/dL). This finding is consistent with Lu et al. [13], who reported that 75.8% of acute coronary syndrome (ACS) patients were men, with a men-to-women ratio of approximately 3:1, and noted similar trends specifically in STEMI populations. The higher proportion of male patients in STEMI cohorts may reflect underlying biological, behavioral, and epidemiological factors influencing cardiovascular risk and disease manifestation. Our results reinforce this gender distribution pattern, suggesting that male sex remains a dominant characteristic in STEMI presentations regardless of glycemic gap status.

In the present study, anterior wall myocardial infarction was the most common STEMI type among patients with a glycemic gap >40 mg/dL (32.0%), whereas inferior wall infarction predominated in the group with glycemic gap ≤40 mg/dL (28.3%). The distribution of extensive anterior and anteroseptal infarctions was also slightly higher in Group I, indicating a trend toward more extensive myocardial involvement in patients with higher glycemic gaps, though the differences were not statistically significant. These findings are consistent with those reported by Garcia et al.[3], who observed that patients with larger glucose delta values—reflective of greater glycemic gaps—were more likely to present with reduced left ventricular ejection fraction and larger infarct sizes. This supports the notion that higher glycemic gap may be associated with more severe myocardial injury, reinforcing its potential prognostic value in STEMI patients.

In this study, patients with a higher glycemic gap (>40 mg/dl) had significantly elevated admission glucose and lower ejection fraction (45.4% vs. 47.1%, p = 0.044), despite similar HbA1c and estimated average glucose levels. This suggests that acute glycemic excursions, rather than chronic hyperglycemia, may contribute more to myocardial dysfunction. These findings align with Garcia et al.[3], who reported that a higher glucose delta was significantly associated with reduced LVEF and larger infarct size in thrombolysed STEMI patients, reinforcing glycemic gap as a marker of greater infarct severity.

In the present study, thrombolysed STEMI patients with a glycemic gap >40 mg/dL (Group I) experienced markedly higher rates of in-hospital complications, including heart failure (60.2%), cardiogenic shock (35.2%), and mortality (13.3%), compared to those with lower glycemic gaps (Group II). These findings align with Wu et al. [1], who reported a significant stepwise increase in 30-day mortality and major cardiovascular events across higher glycemic gap quartiles in a large multicenter cohort of STEMI patients. The consistency of results underscores the prognostic value of glycemic gap in predicting early adverse outcomes in acute coronary syndromes, particularly in thrombolysis-based settings where early risk stratification remains essential.

In the present study, unadjusted logistic regression analysis identified glycemic gap as a strong and statistically significant independent predictor of in-hospital heart failure among thrombolysed STEMI patients (OR = 7.837, p = 0.005), whereas other variables such as age, systemic hypertension, diabetes, and admission glucose did not show significant associations. This finding aligns with that of Liao et al.[12], who reported that a higher glycemic gap was independently associated with increased risks of adverse outcomes, including heart failure, in AMI patients. Similarly, Zhu et al.[14] found that a larger glycemic gap was closely linked to impaired post-infarct left ventricular systolic function, reinforcing the idea that acute glycemic fluctuations reflect underlying myocardial stress. Zheng et al. [15] also demonstrated that elevated glycemic gap was a significant predictor of both acute heart failure and in-hospital mortality, highlighting its prognostic value across various cardiac populations.

The adjusted multivariate logistic regression analysis in this study identified glycemic gap as an independent predictor of heart failure, with a significant odds ratio of 2.746 (p = 0.007). This finding is consistent with Xu et al.[16], who demonstrated that both absolute and relative glycemic gaps were linked to increased 30-day mortality in patients with cardiogenic shock, regardless of diabetes status. Similarly, Elsayed et al. [17] reported that a glycemic gap cutoff of ≥57 mg/dL strongly predicted systolic heart failure and the severity of coronary artery disease in non-diabetic patients. These studies, alongside the current results, emphasize the critical role of glycemic gap in prognosticating acute cardiac dysfunction, underscoring its value as a marker for risk stratification in thrombolysed STEMI patients.

## 6. Limitations of the Study

This study had some limitations:

- This study was limited to a single center, with a predominantly male population, which may limit the generalizability of the findings to the broader national context.
- The use of purposive sampling may have introduced selection bias.
- Long-term follow-up of patients was not conducted, restricting assessment of extended outcomes.

## 7. Conclusion

The study highlights that a higher glycemic gap is significantly associated with worse early in-hospital outcomes in thrombolysed STEMI patients. Patients with elevated glycemic gaps experienced higher rates of heart failure, cardiogenic shock, and mortality. Even after adjusting for confounding factors, glycemic gap remained an independent predictor of heart failure. These findings suggest that glycemic gap may serve as a valuable prognostic marker in the acute management of STEMI.

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