International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

A Study on Prognostic Significance of White Blood Cell Count and Blood Glucose Levels at Admission in ST-Elevation Myocardial Infarction

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Received: 07-01-2026
Accepted: 15-01-2026
Published: 15-02-2026

ABSTRACT

Background: Admission leukocytosis and stress hyperglycemia reflect acute inflammatory and neurohormonal activation during ST-elevation myocardial infarction (STEMI) and are increasingly recognized as readily available bedside risk markers.

Objectives: To evaluate the association between admission total white blood cell (WBC) count and random blood sugar (RBS) levels with in-hospital outcomes in STEMI patients.

Methods: This hospital-based observational, cross-sectional analytical study was conducted in the Department of General Medicine, Kamineni Academy of Medical Sciences and Research Centre, among 200 adults with STEMI admitted to the intensive care unit within 48 hours of symptom onset (August 2023–January 2025). Admission WBC count and RBS were recorded and patients were stratified by WBC (<8000, 8000–11000, >11000 cells/mm³) and RBS (<130, 130–180, >180 mg/dL). Outcomes were assessed during hospitalization. Associations were tested using chi-square and analysis of variance, and multivariable models were used to identify independent predictors of in-hospital mortality.

Results: The cohort was predominantly male (78.0%) and aged 55–64 years (36.5%). Leukocytosis (>11000 cells/mm³) was present in 14.0%, and RBS >180 mg/dL in 19.5%. Admission WBC was significantly associated with RBS categories (p=0.0366). In-hospital mortality was 18.0% and was significantly associated with combined WBC–RBS strata (p=0.0034).

Conclusion: Admission WBC count and RBS demonstrate clinically meaningful prognostic associations in STEMI, particularly when interpreted together. Routine integration of these low-cost parameters can support early risk stratification alongside established clinical assessment.

Keywords: ST-elevation myocardial infarction; leukocytosis; stress hyperglycemia; random blood sugar; prognosis; in-hospital mortality.

INTRODUCTION

ST-elevation myocardial infarction (STEMI) remains a leading cause of preventable mortality, despite advances in reperfusion and intensive care. Early risk stratification is central to contemporary STEMI care because it informs triage decisions, intensity of monitoring, and prioritization for timely reperfusion and adjunctive therapies[1–3]. Conventional risk indicators include age, hemodynamic status, Killip class, infarct territory, and left ventricular systolic function; however, easily measurable biological signals that capture the systemic response to myocardial necrosis can add clinically relevant information.

Acute myocardial ischemia triggers a coordinated inflammatory cascade involving endothelial activation, leukocyte recruitment, cytokine release, and downstream microvascular dysfunction. Peripheral leukocytosis is therefore more than a nonspecific stress response; it has been linked to larger infarct size, impaired myocardial perfusion, heart failure, cardiogenic shock, and mortality in acute myocardial infarction cohorts [1–4]. In STEMI populations, admission WBC count has been shown to provide incremental prognostic value beyond established risk indices and is attractive because it is inexpensive, rapidly available, and routinely measured at presentation [3,4].

Hyperglycemia at admission is likewise common in STEMI, occurring in patients with known diabetes and in those without previously diagnosed dysglycemia. Stress hyperglycemia reflects catecholamine surge, cortisol-mediated insulin resistance, and inflammatory activation; it is associated with endothelial dysfunction, prothrombotic tendency, impaired ischemic preconditioning, and adverse remodeling [5–7]. Large observational studies and meta-analytic evidence indicate that admission hyperglycemia correlates with increased in-hospital death and heart failure in myocardial infarction, including STEMI [6–9]. Recent work also emphasizes the importance of interpreting glucose elevation in context, such as through stress hyperglycemia indices, because risk extends to both diabetic and non-diabetic patients [10,11].

Although leukocytosis and hyperglycemia are individually associated with adverse outcomes, their combined prognostic meaning in routine practice warrants careful evaluation. Both markers represent intersecting pathways sterile inflammation and metabolic stress—that evolve early after coronary occlusion and can plausibly interact to influence short-term outcomes. In settings where advanced biomarkers or complex scoring systems are not consistently available, admission WBC count and random blood sugar (RBS) could provide a pragmatic, scalable approach to early risk assessment.
Objectives: This study aimed to assess the association of admission total WBC count and RBS levels with in-hospital outcomes in STEMI patients and to examine whether their combined stratification is related to in-hospital mortality.

METHODOLOGY

Study design and setting

This observational, cross-sectional analytical study was conducted in the Department of General Medicine, Kamineni Academy of Medical Sciences and Research Centre. Patients admitted to the intensive care unit (ICU) with acute ST-elevation myocardial infarction (STEMI) were enrolled over an 18-month period from August 2023 to January 2025.

Study population and sample size

A total of 200 consecutive patients who satisfied the eligibility criteria were included. Adults of either sex aged >18 years were eligible if they presented within 48 hours of symptom onset with typical anginal chest pain lasting >30 minutes and had electrocardiographic evidence of STEMI defined as ST-segment elevation >1 mm in limb leads or >2 mm in two or more contiguous chest leads.

Exclusion criteria

Patients presenting beyond 48 hours after symptom onset were excluded. Additional exclusions comprised ongoing administration of medications or intravenous fluids known to raise blood glucose, recent surgery or trauma within the preceding month, known leukocytosis or active infection, and alternative causes of ST-segment elevation (including myocarditis, pericarditis, stress cardiomyopathy, benign early repolarization, acute coronary vasospasm, spontaneous coronary artery dissection, left bundle branch block, inherited channelopathies, and significant electrolyte disturbances).

Data collection and investigations

At admission, demographic details, risk factors, clinical status, and Killip class were recorded. Blood samples were collected before initiation of therapies that could influence glucose measurements. Random blood sugar (RBS) was measured on admission using the glucose oxidase–peroxidase method. For complete blood count (CBC), 3 mL of venous blood was collected in an EDTA tube. Total leukocyte count and differential count were analyzed using an automated coulter counter with manual smear verification when required. Routine biochemical tests included blood urea and serum creatinine. Urinalysis included albumin, glucose, and microscopic examination. A standard 12-lead ECG confirmed STEMI and documented infarct territory. Two-dimensional echocardiography was performed to assess left ventricular systolic function and ejection fraction.

Exposure stratification

Patients were stratified a priori by admission RBS into three categories: <130 mg/dL, 130–180 mg/dL, and >180 mg/dL. Total WBC count was categorized as <8000 cells/mm³, 8000–11000 cells/mm³, and >11000 cells/mm³. Combined WBC–RBS strata were used to explore joint associations with in-hospital mortality.

Outcome measures

The primary outcome was in-hospital mortality. Secondary outcome assessment focused on the distribution of clinical severity at presentation and descriptive characterization of the cohort.

Statistical analysis

Data were summarized as frequency and percentage for categorical variables and as mean ± standard deviation for continuous variables. Group comparisons were assessed using chi-square for categorical variables and one-way analysis of variance for continuous variables. Multivariable analysis was performed to identify independent associations with in-hospital mortality after accounting for clinically relevant covariates. A two-tailed p value <0.05 was considered statistically significant.

RESULTS

A total of 200 STEMI patients were included. Baseline demographic and clinical characteristics are summarized in Table 1. The cohort was predominantly male (78.0%) and middle-aged to elderly, with the largest proportion in the 55–64-year age group (36.5%). Diabetes mellitus (63.0%), smoking (60.0%), and hyperlipidemia (54.0%) were common comorbidities. Most patients presented in Killip class I–II (77.5%), and 32.0% received thrombolysis.

Table 1. Baseline demographic and clinical characteristics of STEMI patients (n = 200)

Variable	n (%)
Age group (years): 25–44	19 (9.5)
Age group (years): 45–54	46 (23.0)
Age group (years): 55–64	73 (36.5)
Age group (years): ≥65	62 (31.0)
Gender: Male	156 (78.0)
Gender: Female	44 (22.0)
Smoking	120 (60.0)
Diabetes mellitus	126 (63.0)
Hyperlipidemia	108 (54.0)
Killip class I–II	155 (77.5)
Killip class III–IV	45 (22.5)
Thrombolysis received	64 (32.0)

Admission laboratory distributions are shown in Table 2. Nearly half of patients had WBC <8000 cells/mm³ (47.5%), while leukocytosis >11000 cells/mm³ was observed in 14.0%. Admission RBS was <130 mg/dL in 42.5%, 130–180 mg/dL in 38.0%, and >180 mg/dL in 19.5%.

Table 2. Distribution of admission WBC count and random blood sugar (RBS).

Parameter	Category	n (%)
Total WBC count (cells/mm³)	<8000	95 (47.5)
Total WBC count (cells/mm³)	8000–11000	77 (38.5)
Total WBC count (cells/mm³)	>11000	28 (14.0)
Random blood sugar (mg/dL)	<130	85 (42.5)
Random blood sugar (mg/dL)	130–180	76 (38.0)
Random blood sugar (mg/dL)	>180	39 (19.5)

The relationship between WBC categories and RBS strata is presented in Table 3. A statistically significant association was observed between admission WBC count and glycemic category (chi-square = 10.24, p = 0.0366), with higher WBC categories showing greater representation in higher RBS strata.

Table 3. Association between WBC count and RBS levels at admission.

WBC count (cells/mm³)	RBS <130	RBS 130–180	RBS >180
<8000	39	35	21
8000–11000	40	28	9
>11000	6	13	9

Chi-square = 10.24; p = 0.0366.

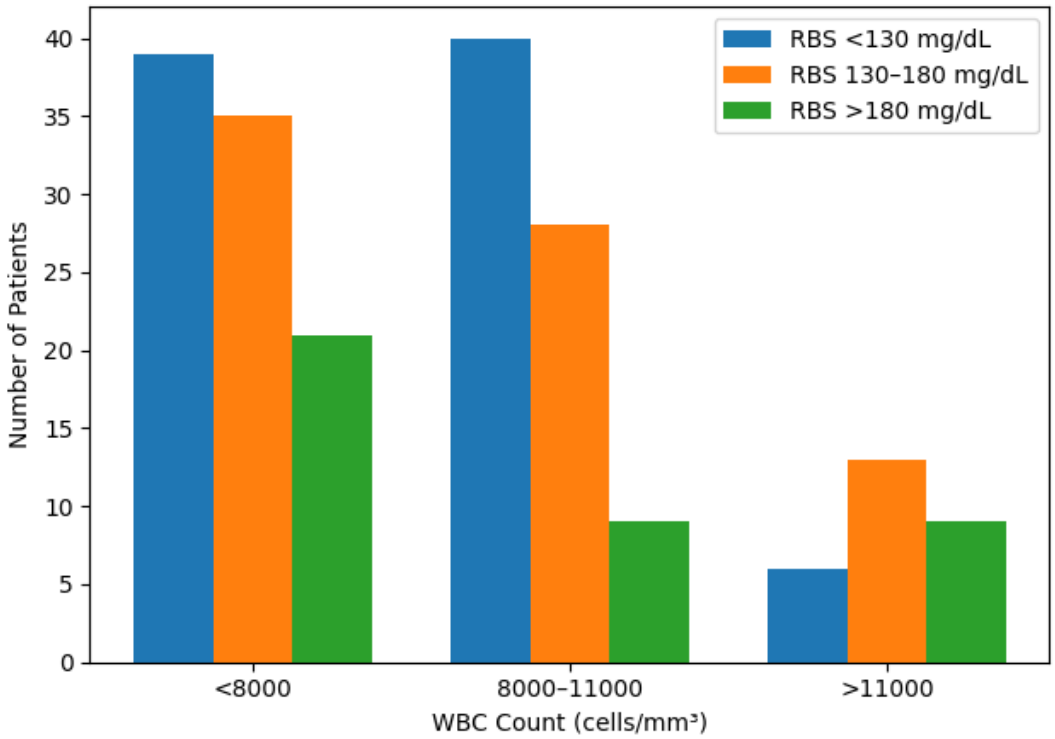

Figure 1: Association between WBC count and RBS levels at admission

Overall in-hospital mortality was 18.0% (36/200). Combined stratification by WBC and RBS demonstrated a significant association with mortality (chi-square = 26.27, p = 0.0034) as shown in Table 4. Deaths were more frequent in strata characterized by concurrent leukocytosis and dysglycemia, indicating that the joint inflammatory–metabolic stress profile at presentation is linked to short-term outcome.

Table 4. Association of WBC count and RBS with in-hospital mortality.

WBC count (cells/mm³)	RBS category (mg/dL)	Death	Alive
<8000	<130	13	28
<8000	130–180	0	33
<8000	>180	4	17
8000–11000	<130	7	33
8000–11000	130–180	6	22
8000–11000	>180	0	9
>11000	<130	3	3
>11000	130–180	3	10
>11000	>180	0	9

Chi-square = 26.27; p = 0.0034

DISCUSSION

This study evaluated two readily available admission parameters total white blood cell (WBC) count and random blood sugar (RBS) and demonstrated that their combined stratification is significantly associated with in-hospital mortality in STEMI. The cohort profile showed a predominance of men and a peak burden in the 55–64-year age group, consistent with regional STEMI patterns and the established sex gap in premature coronary events.

Inflammation is integral to plaque rupture, thrombus propagation, and reperfusion injury. Elevated leukocyte counts can reflect both the magnitude of myocardial necrosis and the intensity of systemic inflammatory activation. Prior studies have reported that admission leukocytosis is associated with heart failure, cardiogenic shock, and death during hospitalization for acute myocardial infarction [10-12]. In STEMI populations, WBC count has also been shown to add prognostic information to existing risk indices and correlate with adverse outcomes after reperfusion-based strategies [3,4]. The observed association between higher WBC strata and poorer outcome in the present cohort aligns with these mechanistic and clinical observations.

Stress hyperglycemia is another early marker of adverse physiology in STEMI. Admission hyperglycemia has been repeatedly linked with increased in-hospital mortality and complications, even among patients without known diabetes

[6,8,9]. Meta-analytic evidence indicates that stress hyperglycemia is associated with higher risk of death after myocardial infarction in both diabetic and non-diabetic patients [13]. Proposed mechanisms include endothelial dysfunction, oxidative stress, platelet hyperreactivity, and impaired microvascular reperfusion, which together can worsen infarct expansion and left ventricular dysfunction [5,7]. More contemporary studies also highlight that stress hyperglycemia metrics can improve risk stratification beyond absolute glucose values [10,14].

A notable finding in this dataset is that the combined WBC–RBS categorization showed a stronger association with mortality than either marker considered in isolation. This supports the concept that inflammatory and metabolic stress responses are intertwined during acute coronary occlusion. Hyperglycemia can amplify inflammatory signaling, while leukocyte activation can exacerbate insulin resistance and endothelial injury, generating a feed-forward loop that favors microvascular obstruction and pump failure. Therefore, interpreting WBC and RBS together at admission provides a pragmatic clinical framework, especially in settings where rapid access to advanced biomarkers is limited.

Clinically, these results reinforce the value of integrating simple laboratory variables into early bedside assessment alongside Killip class and echocardiographic function. Incorporating admission WBC count and RBS into routine risk documentation can support prioritization for close hemodynamic monitoring, early recognition of deterioration, and timely escalation of care.

Limitations

This study was conducted at a single center and included only ICU-admitted STEMI cases, which limits generalizability to all STEMI presentations. Serial WBC and glucose measurements were not analyzed, so dynamic changes during hospitalization were not captured. HbA1c and stress hyperglycemia indices were not routinely available for all participants, restricting differentiation between chronic dysglycemia and acute stress responses. Treatment heterogeneity and unmeasured confounders influenced outcomes.

CONCLUSION

In this cohort of 200 STEMI patients, admission leukocyte count and random blood sugar provided clinically useful prognostic signals. A significant association existed between WBC categories and glycemic strata at presentation, and the combined WBC–RBS classification demonstrated a strong relationship with in-hospital mortality. These findings support routine interpretation of WBC and admission glucose as low-cost, rapidly available risk markers that complement clinical severity assessment. Early identification of patients exhibiting concurrent inflammatory activation and stress hyperglycemia can strengthen triage decisions, guide intensity of monitoring, and support timely escalation of care within the acute STEMI pathway**.**

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