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Original Research Article

ASSOCIATION OF ADMISSION BLOOD GLUCOSE-TO-ALBUMIN RATIO WITH CLINICAL OUTCOMES AFTER PERCUTANEOUS CORONARY INTERVENTION IN STEMI: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Admission-based risk stratification is essential in ST-segment elevation myocardial infarction (STEMI) treated with percutaneous coronary intervention (PCI). The blood glucose-to-albumin ratio (AAR) integrates glycaemic stress and inflammatory–nutritional status and may provide prognostic value. **Aim:** This study assessed the association between admission AAR and clinical outcomes in STEMI patients undergoing PCI. **Materials and Methods:** This prospective observational cohort study included 110 STEMI patients undergoing PCI at Government Kilpauk Medical College and Hospital, Chennai, over a six-month period. Admission blood glucose, serum albumin, blood glucose-to-albumin ratio (AAR), left ventricular ejection fraction, major adverse cardiac events, readmissions, mortality, and heart failure outcomes were assessed. Statistical analysis was performed using SPSS version 29. Associations were analysed using the chi-square test, ANOVA/Kruskal–Wallis test, and logistic regression analysis. **Results:** Among the 110 patients, 79 (71.8%) were male, and most were aged 51–60 years. Mean admission blood glucose was 168.4 ± 42.6 mg/dL, mean serum albumin was 3.5 ± 0.6 g/dL, and mean AAR was 1.32 ± 0.41. Mean left ventricular ejection fraction improved from 45.3 ± 6.5% at admission to 55.1 ± 5.2% at 4 months. Adverse outcomes increased with higher AAR, with major adverse cardiac events occurring more frequently in the AAR >1.5 group (31.0%) than in the AAR 1.0–1.5 (17.4%) and AAR <1.0 (8.6%) groups (p <0.01). Higher AAR was independently associated with major adverse cardiac events, readmissions, mortality, and heart failure with reduced ejection fraction. **Conclusion:** Elevated admission AAR was significantly associated with adverse clinical outcomes in STEMI patients undergoing PCI, including higher rates of major adverse cardiac events, mortality, readmissions, and poorer left ventricular recovery. As AAR is derived from routinely available admission parameters, it may serve as a simple and cost-effective marker for early risk stratification in acute STEMI.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is a severe form of acute coronary syndrome that remains a major contributor to cardiovascular mortality worldwide. Cardiovascular diseases account for approximately 17.9 million deaths annually, with myocardial infarction and stroke responsible for more than four-fifths of these deaths.[1,2] It usually results from acute coronary artery occlusion. Prompt reperfusion is required to limit myocardial necrosis and preserve left ventricular function.[3]

Primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy for STEMI and has reduced short-term mortality. Clinical outcomes

remain heterogeneous despite successful epicardial recanalization. Adverse events continue to occur after PCI. One important mechanism is the no-reflow phenomenon, in which myocardial perfusion remains inadequate despite the restoration of coronary patency. No-reflow has been reported in a variable proportion of STEMI patients and is associated with larger infarct size, heart failure, and increased mortality.[4,5] These limitations highlight the need for early and reliable prognostic assessment in STEMI patients undergoing PCI.[6]

The burden of STEMI is influenced by health system- and population-level factors. More than three-quarters of cardiovascular deaths occur in low- and middle-income countries, where delayed presentation and limited access to emergency cardiac care are common.[2] This setting emphasises the importance of prognostic tools that are simple, inexpensive, and available at the time of hospital admission.[6] Early risk stratification guides treatment decisions and monitoring intensity in acute coronary syndromes.[7] Risk scores, such as the GRACE score, are validated and widely recommended. However, bedside use may be limited because some variables may be unavailable or delayed at presentation.[8] These constraints have increased interest in admission-based laboratory markers for prognostic evaluation in PCI-treated STEMI patients.[6]

Admission blood glucose is a readily available marker of metabolic stress and dysglycaemia. Stress hyperglycaemia (≥140 mg/dL) is common in STEMI and predicts adverse outcomes.[9] A systematic review focusing on STEMI reported that elevated admission and fasting glucose levels predicted worse short-term outcomes.[10] Acute hyperglycaemia promotes inflammation, oxidative stress, endothelial dysfunction, and impaired myocardial reperfusion, contributing to poorer prognosis and an increased risk of no-reflow after primary PCI.[11,12]

Serum albumin reflects nutritional status and systemic inflammation. Low albumin levels are associated with adverse outcomes in acute coronary syndromes. A meta-analysis demonstrated that hypoalbuminaemia independently predicts all-cause mortality after adjustment for established risk factors.[13] Similar associations have been observed in cardiac critical care populations.[14] In PCI-treated patients, lower albumin levels are also associated with longer hospital stay and worse clinical outcomes.[15]

Blood glucose and albumin levels reflect complementary physiological processes. Their combination as the admission blood glucose-to-albumin ratio (AAR) integrates metabolic stress and inflammatory–nutritional status. Both parameters are routinely measured during presentation. Previous studies in STEMI patients undergoing PCI have shown that higher AAR values independently predict all-cause mortality and major adverse cardiac events.[6,16,17] However, limited prospective data are available regarding its prognostic utility in Indian populations and in routine clinical settings with short-term follow-up. In addition, evidence regarding the association of AAR with left ventricular functional recovery and heart failure outcomes remains limited. Therefore, this prospective cohort study was conducted to evaluate the association between admission AAR and clinical outcomes in STEMI patients undergoing PCI and to assess its potential role as a simple and cost-effective early risk stratification marker.

MATERIALS AND METHODS

Study design and setting

This prospective observational cohort study was conducted among 110 patients with STEMI who underwent PCI at the Departments of General Medicine and Cardiology, Government Kilpauk Medical College and Hospital, Chennai, Tamil Nadu, India, from [Month Year] to [Month Year]. The study was approved by the Institutional Ethics Committee (Approval No: (1134/2024), and written informed consent was obtained from all participants prior to enrolment. The sample size was calculated using nMaster software version 2.0 based on a sensitivity of 85%, confidence level of 95%, and precision of 7%, yielding a minimum sample size of 99. After accounting for a 10% non-response rate, the final sample size was determined to be 110.

where $Z = 1.96$ at 95% confidence interval, $p = 85$, $q = (100 - 85) = 15$, and $d = 7$.

$$n = \frac{(1.96)^2 \times 85 \times 15}{7^2}$$

$$n = \frac{3.84 \times 1275}{49}$$

$$n = 99$$

After adding 10% for possible non-response, the final sample size was determined to be 110 patients.

Inclusion Criteria

Adult patients aged >18 years presenting with chest pain and ST-segment elevation on electrocardiography who underwent PCI and provided informed consent were included in the study. A total of 110 eligible patients were enrolled.

Exclusion Criteria

Patients aged <18 years, those with non–ST-elevation myocardial infarction, those who did not undergo PCI, patients with incomplete admission laboratory data required for AAR calculation, those who declined consent, and patients with active malignancy or systemic inflammatory disease were excluded from the study.

Methodology

Patients diagnosed with STEMI were evaluated at admission, and data were collected prospectively from case records, laboratory investigations, and imaging findings. Baseline demographic and clinical parameters including age, sex, body mass index, comorbidities, admission and discharge details were recorded. Height and weight were measured at admission, and body mass index (BMI) was calculated for all patients.

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BMI was categorised according to the World Health Organization classification as underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), and obese (≥30 kg/m²).[18] Admission blood glucose and serum albumin levels were measured before PCI, and the blood glucose-to-albumin ratio (AAR) was calculated as glucose (mg/dL) divided by albumin (g/dL). Patients were categorised into three groups based on AAR values: <1.0, 1.0–1.5, and >1.5.[6] Admission blood glucose, serum albumin, and AAR categories were defined based on previously published literature and clinically relevant cut-off values used in STEMI patients undergoing PCI.[6,9,13] Transthoracic echocardiography was performed at admission and repeated at one and four months to assess left ventricular ejection fraction. All patients were followed during hospitalisation and at 1- and 4-month follow-up visits for assessment of MACE, mortality, readmissions, recurrent pulmonary oedema, and heart failure categories based on follow-up echocardiographic left ventricular ejection fraction. Patients developing EF ≤40% during follow-up were classified as incident HFrEF cases. Cause-specific mortality was not separately analysed. Outcomes were confirmed using clinical records, laboratory findings, imaging reports, and discharge summaries. All enrolled patients completed follow-up by clinic visits, and no loss to follow-up was observed.

Statistical Analysis

Data were analysed using SPSS version 29. Categorical variables were expressed as frequency (%) and compared using the chi-square test, while continuous variables were expressed as mean ± standard deviation or median (IQR) and compared using ANOVA or Kruskal–Wallis test. Logistic regression analysis was performed to estimate odds ratios with 95% confidence intervals. A p value <0.05 was considered statistically significant.

RESULTS

The majority of patients were male (79, 71.8%) and belonged to the 51–60-year age group (29, 29%). Most patients had a normal or overweight body mass index (n = 84, 76.3%). Hypertension was the most common comorbidity (48, 43.6%), followed by diabetes mellitus (32, 29.1%) [Table/Fig-1].

Admission glucose was 140–199 mg/dL in 49 (44.5%) patients, and albumin was ≥3.5 g/dL in 59 (53.6%). AAR was most frequently 1.0–1.5 in 46 (41.8%) patients [Table/Fig-2].

The left ventricular ejection fraction improved during follow-up, increasing from 45.3 ± 6.5 to 55.1 ± 5.2. Patients in the AAR <1.0 group showed the greatest improvement from 48.2 ± 5.1 to 56.3 ± 4.2, whereas the AAR >1.5 group improved from 41.8 ± 6.9 to 49.6 ± 5.8 [Table/Fig-3].

Patients with AAR >1.5 had higher rates of major adverse cardiac events (31.0%, $p < 0.01$), readmissions (27.6%, $p < 0.01$), mortality (13.8%, $p < 0.05$), and heart failure with reduced ejection fraction (24.1%, $p < 0.05$) than those with lower AAR. The differences in heart failure with mid-range and preserved ejection fraction were not significant ($p = 0.07$ and $p = 0.12$, respectively). [Table/Fig-4].

The AAR showed a sensitivity of 70.2% and specificity of 78.5% for major adverse cardiac events, and a sensitivity of 75% and specificity of 80% for mortality. For readmissions, the AAR showed a sensitivity of 68.4% and specificity of 75.3%, whereas for heart failure, the sensitivity was 72.5% and the specificity was 77.8%. [Table/Fig-5].

A higher AAR was associated with an increased risk of major adverse cardiac events (OR 2.6, 95% CI 1.4–4.8; $p < 0.01$), readmissions (OR 2.2, 95% CI 1.2–4.1; $p < 0.05$), mortality (OR 3.0, 95% CI 1.5–6.1; $p < 0.01$), and heart failure with reduced ejection fraction (OR 2.5, 95% CI 1.3–4.8; $p < 0.01$). Associations with heart failure with mid-range ejection fraction (OR 1.7, 95% CI 0.9–3.3; $p = 0.08$) and preserved ejection fraction (OR 1.5, 95% CI 0.8–2.9; $p = 0.10$) were not significant. [Table/Fig-6].

Table 1: Baseline demographic and clinical characteristics

Variable	Category	N (%)
Admission blood glucose (mg/dL)	<140	42 (38.2%)
	140–199	49 (44.5%)
	≥200	19 (17.3%)
Serum albumin (g/dL)	≥3.5	59 (53.6%)
	3.0–3.4	36 (32.7%)
	<3.0	15 (13.6%)
Blood-glucose-to-albumin ratio (AAR)	<1.0	35 (31.8%)
	1.0–1.5	46 (41.8%)
	>1.5	29 (26.4%)

Table 2: Admission laboratory parameters and AAR distribution

Time point	Overall EF (%)	AAR <1.0 EF (%)	AAR 1.0–1.5 EF (%)	AAR >1.5 EF (%)
On admission	45.3 ± 6.5	48.2 ± 5.1	44.6 ± 6.0	41.8 ± 6.9
4th week / 1 month	50.2 ± 5.8	52.4 ± 4.8	49.5 ± 5.4	47.1 ± 6.2
4th month	55.1 ± 5.2	56.3 ± 4.2	53.2 ± 4.9	49.6 ± 5.8

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Table 3: Left ventricular ejection fraction during follow-up

Clinical outcome	N (%)	AAR <1.0 n (%)	AAR 1.0–1.5 n (%)	AAR >1.5 n (%)	p-value
MACE	20 (18.2%)	3 (8.6%)	8 (17.4%)	9 (31.0%)	<0.01
Readmissions	15 (13.6%)	2 (5.7%)	5 (10.9%)	8 (27.6%)	<0.01
Mortality	8 (7.3%)	1 (2.9%)	3 (6.5%)	4 (13.8%)	<0.05
Heart failure (HFrEF)	18 (16.4%)	4 (11.4%)	7 (15.2%)	7 (24.1%)	<0.05
Heart failure (HFmEF)	12 (10.9%)	3 (8.6%)	5 (10.9%)	4 (13.8%)	0.07
Heart failure (HFpEF)	7 (6.4%)	2 (5.7%)	3 (6.5%)	2 (6.9%)	0.12

MACE: all-cause mortality, recurrent myocardial infarction, and stroke. HFrEF: EF ≤40%; HFmEF: EF 41–49%; HFpEF: EF ≥50%.

Table 4: Clinical outcomes and distribution by AAR

Clinical Outcome	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
MACE	70.2	78.5	69.1	79.4
Readmissions	68.4	75.3	66.7	76.8
Mortality	75	80	70	83.3
Heart Failure	72.5	77.8	68.4	81

Table 5: Diagnostic performance of AAR for predicting clinical outcomes

Clinical Outcome	Odds Ratio (95% CI)	p-value
MACE	2.6 (1.4-4.8)	<0.01
Readmissions	2.2 (1.2-4.1)	<0.05
Mortality	3.0 (1.5-6.1)	<0.01
Heart Failure (HFrEF)	2.5 (1.3-4.8)	<0.01

Multivariate analysis of the association between AAR and clinical outcomes. Reference category: AAR <1.0 group.
HFrEF: EF ≤40%.

DISCUSSION

This study demonstrated that elevated admission AAR was significantly associated with adverse clinical outcomes in STEMI patients undergoing PCI, including higher rates of MACE, mortality, readmissions, and poorer left ventricular recovery. Patients with higher AAR also demonstrated increased risk of developing HFrEF during follow-up, highlighting the prognostic value of AAR as a simple admission-based risk stratification marker.

In our study, most patients were male (71.8%) and belonged to the 51–60-year age group (29%). Hypertension (43.6%) and diabetes mellitus (29.1%) were the most common comorbidities. Most patients demonstrated moderately elevated admission glucose levels (140–199 mg/dL in 44.5%), preserved serum albumin levels (≥3.5 g/dL in 53.6%), and intermediate AAR values (1.0–1.5 in 41.8%). Similarly, Zhen et al. reported that most PCI-treated ACS patients were male (75.5%–88.9%), with hypertension being the predominant comorbidity and higher admission glucose levels associated with elevated AAR groups.[6] These findings suggest that admission AAR reflects the combined influence of metabolic stress and inflammatory–nutritional status in acute STEMI.

In this study, the left ventricular function improved over time, with better recovery in patients with a lower AAR and poorer function in those with a higher AAR. Similarly, Bo et al. reported that patients with higher fasting stress hyperglycaemia ratio (SHR3 ≥1.01) had significantly impaired left ventricular function, with lower LVEF (45.0% vs. 53.3%), lower LVGFI (25.3 vs. 31.0), reduced myocardial strain parameters, and greater microvascular obstruction; fasting SHR remained independently associated with impaired LVEF (β = −6.815), LVGFI (β = −5.403), and increased risk of MVO (OR 1.591).[19] Meng et al. found that baseline LVEF was similar in the low SHR and high SHR groups (50.8 ± 9.4% vs. 49.1 ± 7.7%). At 6 months, LVEF was higher in the low SHR group (59.4 ± 7.3%) than in the high SHR group (54.0 ± 9.6%, p = 0.001), with SHR independently predicting poorer EF recovery (β = −9.8).[20] These findings support the association between stress-related metabolic dysregulation and impaired ventricular recovery following myocardial infarction. Our study showed that patients with higher AAR experienced more frequent MACE, readmissions, mortality, and HFrEF than those with lower AAR values. Similar findings were reported by Zhen et al., who demonstrated significantly higher in-hospital and long-term adverse cardiovascular outcomes in patients with elevated AAR following PCI.[6] These findings support the role of AAR as an integrated marker of metabolic stress, inflammation, and nutritional status associated with adverse post-infarction outcomes.

In our study, AAR demonstrated good prognostic performance for identifying patients at higher risk of adverse cardiac outcomes, with sensitivity and specificity of 70.2% and 78.5% for MACE, and 75% and 80% for mortality, respectively. Previous studies evaluating stress hyperglycaemia ratio have also demonstrated moderate predictive accuracy for adverse outcomes following PCI.[20,21] However, AAR may provide additional prognostic value over

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isolated glucose or albumin measurements because it simultaneously reflects acute metabolic stress, inflammatory burden, and nutritional status. Unlike complex risk scores such as GRACE and TIMI, AAR can be rapidly calculated using routinely available admission laboratory parameters, making it potentially useful for early bedside risk stratification in resource-limited settings.

In this study, higher admission AAR independently predicted increased risk of MACE, mortality, readmissions, and HFrEF. Similar findings were reported by Zhen et al., who demonstrated that elevated AAR independently predicted in-hospital mortality, MACEs, and lower LVEF in ACS patients undergoing PCI.[6] Jiang et al. also reported that higher AAR was associated with increased adverse cardiovascular events, higher all-cause mortality, and reduced ventricular function following PCI.[22] Elevated AAR may reflect increased oxidative stress, inflammatory activation, endothelial dysfunction, impaired myocardial perfusion, and microvascular dysfunction, thereby contributing to adverse post-infarction outcomes and poorer ventricular recovery. Patients with elevated AAR may benefit from closer haemodynamic monitoring, aggressive secondary prevention strategies, and early follow-up for ventricular dysfunction and heart failure progression. The simplicity, low cost, and wide availability of admission glucose and albumin measurements may support the clinical applicability of AAR in routine STEMI management.

Using admission AAR could help identify high-risk patients early. Larger, multicentre studies are needed to confirm its value and guide treatment decisions.

Limitation(s)

This single-centre observational study cannot establish causality and may be affected by residual confounding. The sample size was modest, and follow-up was limited to four months. Important clinical variables, repeated measures echocardiographic analysis, ROC-derived cut-off assessment, and cause-specific mortality analysis were not systematically evaluated.

CONCLUSION

Elevated admission AAR was significantly associated with adverse clinical outcomes in STEMI patients undergoing PCI. Patients with higher AAR demonstrated increased rates of MACE, mortality, readmissions, and poorer left ventricular recovery during follow-up. Higher AAR also independently predicted the development of HFrEF and adverse cardiovascular outcomes. As AAR is derived from routinely available admission glucose and albumin measurements, it may serve as a simple, inexpensive, and clinically useful marker for early risk stratification. Using admission AAR could help identify high-risk patients who may benefit from closer monitoring and intensive follow-up.

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