e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(6); 1298-1303

Original Research Article

Prognostic value of HbA1c and Magnesium in Non-Diabetic Acute Myocardial Infarction

G. Pramodini[1], Golagani Kiranmai[2], Divya Gayatri Penupothu[3], Sri Sarada Devi Dasari[4]

[1, 2] Assistant Professor, Department of Biochemistry, Rangaraya Medical College, Kakinada.
[3]Assistant professor, Department of Biochemistry, GSL Medical College, Rajahmundry.
[4] Civil Assistant Surgeon, Blood Bank, Rangaraya Medical College, Kakinada.

Received: 29-04-2026 / Revised: 25-05-2026 / Accepted: 26-06-2026
Corresponding Author: Dr. Sri Sarada Devi Dasari
Conflict of interest: Nil

Abstract:

Background: HbA1c is a marker of chronic glycaemic exposure and may identify occult dysglycaemia in patients without known diabetes. Its role in non-diabetic acute myocardial infarction (AMI) patients requires further evaluation. **Aim:** To estimate admission HbA1c levels in non-diabetic AMI patients and assess their association with clinical and biochemical parameters.

Methods: This prospective case–control study was conducted at Rangaraya Medical College and Government General Hospital, Kakinada, from July 2017 to July 2018. Fifty non-diabetic AMI patients were included as cases and compared with 50 age- and sex-matched controls. Venous blood samples were analysed for HbA1c, random blood sugar, Magnesium, blood urea, serum creatinine and Troponin I. HbA1c was compared between cases and controls and correlated with biochemical parameters. Its association with morbidity and mortality was also assessed.

Results: HbA1c levels were significantly higher among non-diabetic AMI patients than controls. Random blood sugar, blood urea, Magnesium and serum creatinine were also significantly elevated in cases. Patients with HbA1c ≥5.7% had higher morbidity compared with those having HbA1c <5.7%. Mortality was higher in the elevated HbA1c group, but the association was not statistically significant. HbA1c showed positive correlation with random blood sugar, blood urea and serum creatinine, while serum magnesium showed a weak negative correlation with HbA1c.

Conclusion: Admission HbA1c may identify non-diabetic AMI patients at higher morbidity risk, while serum magnesium adds supportive biochemical information on acute electrolyte alterations.

Key words: Acute myocardial infarction; HbA1c; Non-diabetic; Random blood sugar; Morbidity.

DOI: 10.25258/ijcpr.18.6.210

Introduction

Acute myocardial infarction (AMI) remains a major cause of early morbidity and mortality, and risk stratification at admission is essential for identifying patients who may develop complications despite absence of known diabetes. HbA1c reflects average glycaemic exposure over the preceding 2–3 months and may identify occult dysglycaemia, prediabetes, chronic metabolic stress, and vascular risk not captured by random blood sugar alone. Recent evidence suggests that abnormal HbA1c levels in non-diabetic coronary artery disease patients are associated with adverse long-term clinical events, while glucose-related indices including admission glucose, HbA1c, glucose–HbA1c ratio and stress hyperglycaemia ratio have shown prognostic value in AMI cohorts [1,2]. Admission hyperglycaemia has also been reported as an independent predictor of worse short- and long-term outcomes after AMI, including among patients without diabetes [3]. Further, patients with prediabetic-range HbA1c presenting with STEMI have demonstrated poorer survival outcomes, supporting the clinical relevance of HbA1c-based categorisation at presentation [4]. Hence, the present study aims to estimate admission HbA1c levels in non-diabetic AMI patients, compare them with age- and sex-matched controls, and evaluate their association with morbidity, mortality, RBS, blood urea and serum creatinine.

Methods

This prospective case–control study was conducted in the department of Biochemistry, Rangaraya Medical College, Kakinada, Andhra Pradesh, India, in collaboration with the Department of Medicine and Department of Cardiology, Government General Hospital, Kakinada. The study was carried

out from July 2017 to July 2018 after obtaining approval from the Institutional Ethics Committee of Rangaraya Medical College, Kakinada. Written informed consent was obtained from all participants before enrolment. A total of 50 non-diabetic AMI patients were included as cases, and 50 apparently healthy age- and gender -matched individuals were included as controls.

AMI was diagnosed based on presenting history, clinical symptoms, physical examination findings, electrocardiographic changes, 2D echocardiography findings and biochemical confirmation by serum Troponin I. After admission, detailed clinical history was obtained from each patient, including history of hypertension, smoking, obesity, previous stroke, rheumatic heart disease and other relevant cardiovascular risk factors. Patients were included if they had clinical features suggestive of AMI, no past history of diabetes mellitus, arrival to Government General Hospital within 4 hours of onset of chest pain, written informed consent, and Troponin I value >0.04 ng/ml. Patients with known diabetes mellitus, Troponin I value <0.04 ng/ml, delayed presentation beyond 4 hours of chest pain, chronic muscle disease, liver disease, renal disease, recent major surgery, implanted pacemaker or autoimmune disease were excluded from the study. Morbidity parameters and in-hospital mortality among cases were recorded to assess their association with HbA1c levels.

Blood samples were collected immediately after clinical evaluation and before further biochemical analysis. Approximately 5 ml of venous blood was drawn aseptically from the antecubital vein of the upper limb from each case and control. Serum was separated and analysed using standard laboratory methods. In addition, 2 ml of blood was collected in an EDTA vial for HbA1c estimation. The biochemical parameters analysed included HbA1c, random blood sugar, Magnesium, blood urea, serum creatinine and Troponin I. HbA1c was estimated by nephelometry. Troponin I was estimated by chemiluminescent immunoassay using the Beckman Coulter Access 2 analyser. Random blood sugar, blood urea and serum creatinine were estimated using standard biochemical methods. Admission HbA1c values were compared between acute MI patients and controls. Random blood sugar, blood urea and serum creatinine were also compared between both groups to evaluate admission biochemical differences.

The collected data were entered and analysed statistically. Continuous variables such as HbA1c, random blood sugar, blood urea and serum creatinine were expressed as mean ± standard deviation. Categorical variables such as age group, sex, morbidity and mortality were expressed as frequency and percentage. The Student's t-test was used to compare mean biochemical values between cases and controls. HbA1c levels were categorised and analysed for association with complications, morbidity and mortality among non-diabetic acute MI patients. Pearson's correlation coefficient was used to assess the correlation of HbA1c with random blood sugar, blood urea and serum creatinine. A p value of less than 0.05 was considered statistically significant.

Results

A total of 100 participants were included in the study, comprising 50per group. The age distribution was comparable between cases and controls, with the majority of participants belonging to the 51–60 years age group. Male predominance was observed in both groups, but the difference in gender distribution was not statistically significant, indicating adequate matching between cases and controls (Table 1). Admission HbA1c levels were significantly higher among acute MI patients compared with controls, suggesting the presence of underlying dysglycaemic status even in patients without known diabetes. Random blood sugar, blood urea and serum creatinine were also significantly elevated among cases compared with controls, showing that acute MI was associated with metabolic and renal biochemical alterations at admission (Table 2). When HbA1c was categorised using 5.7% as the cut-off, morbidity was more frequent among patients with HbA1c ≥5.7% compared with those having HbA1c <5.7%. This association was statistically significant, suggesting that higher HbA1c levels may be linked with increased in-hospital complications among non-diabetic acute MI patients (Table 3). Mortality was also higher in the HbA1c ≥5.7% group; however, this difference did not reach statistical significance, possibly due to the limited sample size (Table 4). Serum magnesium levels were significantly higher among non-diabetic AMI patients compared with controls (Table 4). Correlation analysis showed a significant positive correlation of HbA1c with random blood sugar, blood urea and serum creatinine. These findings indicate that admission HbA1c may serve as a useful biochemical marker associated with clinical morbidity and metabolic stress in non-diabetic AMI patients.

Table 1: Age and gender distribution of the study members in groups				
Variable	**Cases**	**Controls**	**χ^2 value**	**p value**
Age				
31–40	6 (12.0%)	7 (14.0%)	0.42	0.936
41–50	13 (26.0%)	12 (24.0%)		
51–60	19 (38.0%)	18 (36.0%)		
>60	12 (24.0%)	13 (26.0%)		
Gender				
Male	32 (64.0%)	30 (60.0%)	0.17	0.683
Female	18 (36.0%)	20 (40.0%)		

Table 2: Comparison of HbA1c, RBS, blood urea and serum creatinine among the study members in groups				
Parameter	**Cases**	**Controls**	**t value**	**p value**
HbA1c (%)	5.82 ± 0.42	5.31 ± 0.36	6.51	<0.001
Random blood sugar (mg/dL)	146.28 ± 28.64	104.72 ± 13.85	9.25	<0.001
Blood urea (mg/dL)	38.46 ± 10.92	27.84 ± 6.58	5.89	<0.001
Serum creatinine (mg/dL)	1.12 ± 0.28	0.86 ± 0.19	5.43	<0.001
Serum magnesium (mg/dL)	2.35 ± 0.38	1.86 ± 0.11	2.65	<0.001

Table 3: Association of HbA1c categories with morbidity among non-diabetic acute MI patients					
HbA1c category	**Morbidity**		**Total cases**	**χ^2 value**	**p value**
	Present	**Absent**			
<5.7%	6 (27.3%)	16 (72.7%)	22 (44%)	6.27	0.012
≥5.7%	17 (60.7%)	11 (39.3%)	28 (56%)		
Total	23 (46.0%)	27 (54.0%)	50 (100%)		

Table 4: Association of HbA1c with mortality and correlation with biochemical parameters			
Analysis	**HbA1c <5.7%**	**HbA1c ≥5.7%**	**Statistics**
Survivors	21 (95.5%)	23 (82.1%)	$\chi^2 = 2.08$; P = 0.149
Deaths	1 (4.5%)	5 (17.9%)	

Discussion:

In the present prospective case–control study, admission HbA1c levels were significantly higher among non-diabetic AMI patients compared with age- and sex-matched controls. Although all cases were selected after excluding known diabetes mellitus, the higher HbA1c values suggested the possible presence of previously unrecognised dysglycaemia, prediabetic metabolic status or chronic low-grade glycaemic exposure before the acute coronary event. This finding is clinically important because HbA1c reflects average blood glucose over the previous 2–3 months and is less influenced by acute stress than random blood sugar. Therefore, HbA1c estimation at admission may help distinguish chronic metabolic risk from transient stress hyperglycaemia. Indian studies have shown that a considerable proportion of patients presenting with acute coronary syndrome have previously undetected diabetes or impaired glucose tolerance, supporting the need for routine glycaemic evaluation in apparently non-diabetic ACS patients [5, 6]. Ramachandran et al. also observed high prevalence of glucose intolerance among Asian Indian patients following ACS, indicating that dysglycaemia is frequently underdiagnosed in this population [7]. The present study therefore adds support to the view that HbA1c may be a useful admission marker in non-diabetic AMI patients.

The age and gender distribution were comparable between cases and controls, indicating adequate matching and reducing the possibility that the observed biochemical differences were due to demographic imbalance. Most patients belonged to the middle-aged and elderly groups, and male predominance was observed among AMI cases. This pattern is consistent with Indian data showing that acute coronary syndrome commonly affects productive age groups and continues to show male preponderance, although the burden among women increases with advancing age and metabolic risk exposure [4]. The higher HbA1c observed in cases may reflect the combined influence of insulin resistance, central obesity, hypertension, smoking

and other cardiometabolic risk factors. Rao et al. reported that metabolic risk factors, including high HbA1c, low HDL cholesterol and increased waist–hip ratio, were strongly associated with first ACS events in India [9]. These observations are relevant to the present study because patients with known diabetes were excluded, yet those with risk factors such as hypertension, smoking and obesity were included. Thus, HbA1c may function not only as a glycaemic marker but also as an integrated indicator of adverse metabolic milieu contributing to coronary atherosclerosis and plaque instability.

Random blood sugar was significantly higher among AMI cases compared with controls. This elevation may be explained by acute stress hyperglycaemia due to catecholamine surge, cortisol release, inflammatory activation and increased hepatic glucose output during myocardial infarction. However, the significant positive correlation between HbA1c and random blood sugar in the present study suggested that admission hyperglycaemia was not entirely an acute phenomenon in all patients; rather, individuals with higher baseline glycaemic exposure were more likely to show higher admission glucose levels. This is in agreement with earlier Indian observations that stress hyperglycaemia in non-diabetic AMI patients may predict underlying abnormal glucose tolerance [10]. Mokta et al. also demonstrated a high incidence of abnormal glucose metabolism among non-diabetic ACS patients using admission plasma glucose, fasting plasma glucose, HbA1c and oral glucose tolerance testing [6]. These findings support the concept that admission glucose and HbA1c should be interpreted together. While RBS reflects the acute physiological stress response, HbA1c helps identify chronic dysglycaemic background. Therefore, simultaneous estimation of both markers may improve risk classification in non-diabetic AMI patients.

The present study also observed significantly higher morbidity among patients with HbA1c ≥5.7% compared with those having HbA1c <5.7%. This suggests that even prediabetic-range HbA1c may be associated with greater in-hospital complications after AMI. Higher HbA1c may contribute to endothelial dysfunction, oxidative stress, impaired nitric oxide availability, increased platelet reactivity, inflammation, prothrombotic tendency and microvascular dysfunction. These mechanisms may worsen myocardial perfusion, increase infarct-related complications and delay recovery. Indian studies have shown that abnormal glycaemic status is common in ACS and may influence clinical outcome [5, 6]. Similarly, angiographic studies from India have demonstrated differences in coronary involvement between diabetic and non-diabetic ACS patients, highlighting the role of metabolic status in coronary disease expression [11]. Although our study did not include coronary angiographic severity, the increased morbidity in higher HbA1c categories indirectly supports the association between chronic glycaemic exposure and adverse clinical course. The observed morbidity included arrhythmia, heart failure, cardiogenic shock, recurrent angina and prolonged hospital stay, all of which may reflect greater myocardial injury or poorer physiological reserve among patients with higher HbA1c.

Mortality was higher among patients with HbA1c ≥5.7%, but the association did not reach statistical significance. This may be due to the relatively small sample size of 50 AMI cases and the limited number of deaths during the study period. Nevertheless, the direction of association was clinically relevant, as mortality was proportionately greater in the higher HbA1c group. Indian data from large ACS cohorts have shown that diabetes and metabolic abnormalities adversely influence short-term outcomes after AMI [11]. In the ACS QUIK trial analysis from India, diabetes was evaluated in relation to in-hospital and 30-day major adverse cardiac events, including death, reinfarction, stroke and major bleeding, indicating the importance of glycaemic status in AMI prognosis [12]. Although the present study focused on non-diabetic patients, the results suggest that prediabetic-range HbA1c may represent an intermediate risk state. A larger sample with longer follow-up would be required to confirm whether admission HbA1c independently predicts mortality among non-diabetic AMI patients. Until then, HbA1c may be considered a supportive risk marker rather than a standalone mortality predictor.

Blood urea and serum creatinine were significantly elevated among cases compared with controls, and both showed mild positive correlation with HbA1c. Renal dysfunction in AMI may occur due to haemodynamic instability, reduced renal perfusion, neurohormonal activation, contrast exposure during coronary procedures or pre-existing subclinical renal disease. Even mild renal impairment can worsen cardiovascular prognosis because it is associated with inflammation, endothelial dysfunction, volume overload, electrolyte imbalance and reduced tolerance to cardioprotective drugs. Indian studies have reported that altered renal function and acute kidney injury influence outcomes in cardiac and STEMI patients [13, 14]. Saini et al. evaluated acute kidney injury in nonsurgical cardiac intensive care patients, while Kanabar et al. studied predictors of AKI and outcomes among Indian STEMI patients [13, 14]. The positive correlation between HbA1c and renal parameters in the present study may indicate that chronic metabolic stress contributes to subtle renal dysfunction even before overt diabetes develops. Overall, the present findings suggest that admission HbA1c, along with

RBS, blood urea and serum creatinine, may provide a simple biochemical profile for early assessment of non-diabetic AMI patients. The study was limited by small sample size, single-centre design, short follow-up and absence of angiographic or long-term outcome assessment. However, it highlights the value of HbA1c testing at admission for identifying high-risk non-diabetic AMI patients who may benefit from closer monitoring and follow-up.

Serum magnesium was significantly higher in non-diabetic AMI patients than in controls in the present study, suggesting altered magnesium homeostasis during the acute coronary event. Although many studies describe hypomagnesemia in AMI, higher serum magnesium may occur due to acute cellular injury, reduced renal clearance, haemodynamic instability, or early redistribution of intracellular magnesium into the extracellular compartment. Therefore, this finding should be interpreted along with renal parameters, infarct severity and timing of sample collection. Indian evidence supports the biological relevance of magnesium in coronary disease; Swetha et al. reported altered magnesium status among coronary artery disease patients in a South Indian population, while Mohan et al. described serum magnesium as a prognostic marker in acute myocardial infarction [15, 16]. Thus, serum magnesium estimation may provide supportive information in non-diabetic AMI patients, but its direction of change and prognostic value require confirmation through larger studies.

Conclusion

The present study showed that admission HbA1c levels were significantly higher in non-diabetic acute myocardial infarction patients compared with age- and sex-matched controls. Higher HbA1c levels were associated with increased in-hospital morbidity, suggesting that HbA1c may help identify non-diabetic AMI patients at greater risk of complications. Random blood sugar, blood urea and serum creatinine were also significantly elevated among cases and showed positive correlation with HbA1c, indicating that chronic glycaemic exposure may be linked with acute metabolic and renal stress during myocardial infarction. Serum magnesium levels were also significantly higher among non-diabetic AMI patients compared with healthy controls, suggesting altered magnesium homeostasis during the acute coronary event. Although mortality was higher among patients with HbA1c ≥5.7%, the association was not statistically significant. The major limitations were small sample size, single-centre design, short observation period, absence of long-term follow-up, lack of angiographic correlation, and absence of serial magnesium estimation. Therefore, larger multicentric studies with serial follow-up are required to confirm the prognostic role of HbA1c and serum magnesium in non-diabetic AMI patients.

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