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EVALUATION OF STRESS HYPERGLYCEMIA IN NON-DIABETIC PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Abstract

Introduction: Stress hyperglycemia frequently occurs during acute myocardial infarction (AMI) even in patients without previously diagnosed diabetes. This transient rise in blood glucose represents an acute metabolic response to physiological stress but is increasingly recognized as a marker of adverse cardiovascular outcomes. Understanding its prognostic significance in non-diabetic individuals is essential for risk stratification and early intervention. Objectives: To evaluate the stress of hyperglycemia in non-diabetic patients presenting with acute myocardial infarction. Methodology: This observational study included non-diabetic adult patients admitted with AMI. Stress hyperglycemia was assessed using admission plasma glucose and the stress hyperglycemia ratio (SHR). Clinical outcomes including in-hospital mortality, heart failure, arrhythmias, cardiogenic shock, and length of hospital stay were recorded. Patients were stratified into normoglycemic and stress-hyperglycemic groups for comparative analysis. Results & Findings: Patients with stress hyperglycemia demonstrated significantly higher rates of adverse outcomes, including increased risk of in-hospital mortality, acute heart failure, and cardiogenic shock. Elevated admission glucose and higher SHR were strong independent predictors of complications. Stress hyperglycemia was also associated with prolonged hospital stay and higher need for intensive care support. Conclusion: Stress hyperglycemia is a powerful prognostic marker in non-diabetic AMI patients. Elevated glucose levels at presentation predict higher morbidity and mortality, emphasizing the need for early identification and tighter glucose monitoring in this population. Incorporating stress hyperglycemia into routine risk assessment may improve clinical decision-making and patient outcomes.

INTRODUCTION

Acute myocardial infarction (AMI) remains one of the leading causes of mortality and disability worldwide despite substantial advances in pharmacological therapy, coronary revascularization techniques, and preventive cardiovascular care [1,2]. The global burden of ischemic heart disease continues to rise, particularly in low- and middle-income countries where increasing prevalence of cardiovascular risk factors has contributed to growing morbidity and mortality [3]. Early identification of prognostic markers in AMI is therefore essential for improving risk stratification, guiding therapeutic interventions, and reducing adverse cardiovascular outcomes [4]. Hyperglycemia is frequently observed during the acute phase of myocardial infarction, even among individuals without a previous diagnosis of diabetes mellitus [5]. This phenomenon, commonly referred to as stress hyperglycemia, represents a transient elevation in blood glucose concentration triggered by acute physiological stress [6]. During AMI, activation of the sympathetic nervous system and hypothalamic–pituitary–adrenal axis results in increased secretion of catecholamines, cortisol, glucagon, and growth hormone [7]. These neurohormonal responses promote insulin resistance, enhance hepatic glucose production, and suppress peripheral glucose utilization, leading to elevated circulating glucose levels [8]. Consequently, stress hyperglycemia is increasingly recognized as a common metabolic response accompanying acute myocardial injury [9]. Growing evidence suggests that stress hyperglycemia is not merely a physiological stress response but an important determinant of clinical outcomes in patients with AMI [10]. Several observational studies and meta-analyses have demonstrated a significant association between elevated admission glucose levels and increased risks of major adverse cardiovascular events (MACE), heart failure, cardiogenic shock, arrhythmias, and all-cause mortality [11]. Importantly, these associations have been observed not only in patients with established diabetes but also in individuals without known glucose metabolism disorders. In many investigations, non-diabetic patients presenting with hyperglycemia during AMI exhibited poorer outcomes than their normoglycemic counterparts, highlighting the potential prognostic value of stress-induced hyperglycemia [12].

The biological mechanisms underlying this association are multifactorial. Acute hyperglycemia contributes to endothelial dysfunction, oxidative stress, inflammatory activation, platelet aggregation, and pro-thrombotic states, all of which can aggravate myocardial ischemia and impair coronary microvascular perfusion [13]. Elevated glucose levels have also been linked to larger infarct size, reduced myocardial salvage following reperfusion therapy, impaired left ventricular function, and adverse ventricular remodeling [14]. These pathophysiological alterations provide a mechanistic explanation for the observed relationship between stress hyperglycemia and unfavorable cardiovascular outcomes [15]. Recent research has further refined the assessment of stress-related dysglycemia through the introduction of the stress hyperglycemia ratio (SHR), which compares admission glucose levels with estimated chronic glycemic status derived from glycated hemoglobin (HbA1c) [16]. This approach enables differentiation between acute stress-induced hyperglycemia and previously unrecognized chronic dysglycemia. Studies have reported that elevated SHR is independently associated with increased mortality, larger infarct size, and higher incidence of post-infarction complications, suggesting that relative hyperglycemia may provide superior prognostic information compared with absolute glucose measurements alone [17]. Despite increasing recognition of stress hyperglycemia as a prognostic marker, its clinical significance in non-diabetic patients remains insufficiently characterized, particularly in developing countries where cardiovascular disease burden is rapidly increasing [18]. In routine clinical practice, elevated glucose levels in non-diabetic AMI patients are often regarded as a transient physiological response and may not receive adequate attention during risk assessment. Consequently, opportunities for early identification of high-risk patients and implementation of targeted management strategies may be missed. Furthermore, regional data regarding the prevalence and clinical implications of stress hyperglycemia among non-diabetic AMI patients remain limited [19]. Given these considerations, evaluating stress hyperglycemia in non-diabetic individuals presenting with AMI is of substantial clinical importance. Early recognition of stress-induced hyperglycemia may facilitate improved risk

stratification, closer monitoring, optimized therapeutic decision-making, and timely intervention, thereby potentially reducing cardiovascular complications and improving patient outcomes [20]. Therefore, the present study was designed to evaluate the occurrence and clinical significance of stress hyperglycemia among non-diabetic patients with acute myocardial infarction and to determine its potential role as a prognostic indicator in this patient population.

Objectives of the study

To determine the prognostic impact of stress hyperglycemia—defined as admission random blood glucose >140 mg/dL in the presence of HbA1c <6.5%—on in- hospital adverse outcomes (mortality, hemodynamic instability, major arrhythmias, and need for intensive care) among non- diabetic patients aged 18–50 years presenting with acute myocardial infarction.

Problem Statement

Acute myocardial infarction (AMI) in adults aged 18–50 years remains a life- threatening condition, and early, accurate risk stratification is essential. Stress hyperglycemia, a transient rise in blood glucose at admission, is frequently observed in patients without diabetes, yet its predictive value for complications and death in this group remains uncertain. Current risk- assessment protocols focus largely on individuals with known diabetes, leaving a critical gap in identifying non- diabetic patients who may still experience harmful glycemic spikes during AMI. Investigating whether stress hyperglycemia independently forecasts adverse in- hospital outcomes could refine risk stratification and guide timely therapeutic decisions in this understudied population.

METHODOLOGY

This prospective observational study was carried out at CMA Teaching and Research Hospital, Lahore, a tertiary care centre with continuous access to diagnostic, laboratory, and coronary care facilities, over a period of four months following ethical approval. Non- diabetic patients aged 18 to 50 years with a confirmed diagnosis of acute myocardial infarction (AMI) were consecutively enrolled after providing written informed consent. AMI was defined according to the Fourth Universal Definition, requiring a rise and/or fall of cardiac troponin with at least one value exceeding the 99th percentile upper reference limit, together with symptoms of ischaemia, new electrocardiographic changes, imaging evidence of new regional wall- motion abnormality, or angiographic identification of intracoronary thrombus. To be eligible, participants had to present within 24 hours of symptom onset. Patients were excluded if they had a prior diagnosis of diabetes mellitus, were using any glucose- lowering medication, had received parenteral glucose, dextrose infusion, or systemic corticosteroids before admission blood sampling, or presented with severe hepatic failure, acute metabolic disturbances unrelated to AMI, active systemic infection, sepsis, or pregnancy. Individuals transferred from another hospital more than 24 hours after symptom onset and those who declined to participate were also excluded. Stress hyperglycemia was defined as an admission random blood glucose greater than 140 mg/dL in the absence of diabetes, verified by a hemoglobin A1c level below 6.5%. Patients who met these criteria constituted the stress hyperglycemia group, while those with admission random blood glucose at or below 140 mg/dL and HbA1c under 6.5% served as the normoglycaemic control group. The primary prognostic endpoint was a composite of in- hospital adverse outcomes, comprising all- cause mortality, hemodynamic instability (systolic blood pressure below 90 mmHg necessitating vasopressor support), sustained ventricular tachycardia or ventricular fibrillation, and admission to the intensive care unit. Each component was recorded as a binary outcome.

Data were collected using a structured case- report form that captured demographic details, admission vital signs, random blood glucose, HbA1c, high- sensitivity cardiac troponin, electrocardiographic findings, AMI classification (ST- elevation or non- ST- elevation), and in- hospital clinical events. All laboratory investigations were part of the standard AMI management protocol, and no additional tests were performed for research purposes. The minimum required sample size was calculated using Cochran's formula for a comparative study with a 95% confidence level (Z=1.96), an anticipated adverse event proportion of 0.45 in the stress hyperglycemia group, and an absolute precision of 0.10, yielding 96 participants. Consecutive sampling was employed, enrolling all eligible patients who presented during the study period until this target was reached, thereby minimizing selection bias.

Statistical analyses were performed using SPSS version 27.0. Continuous variables were summarized as mean ± standard deviation or median with interquartile range depending on normality, while categorical variables were expressed as frequencies and percentages. Patients were stratified by glycemic status, and the chi- square test (or Fisher's exact test where appropriate) was applied to examine associations between stress hyperglycemia and categorical adverse outcomes. Linear regression models were used to explore relationships between admission glucose levels and continuous indicators of disease severity, including peak troponin concentration and left ventricular ejection fraction. All tests were two- tailed, and a p- value less than 0.05 was considered statistically significant. The study was approved by the Ethical Review Committee of Superior University, Lahore, and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants, who were assured that their involvement was voluntary and that refusal or withdrawal would not affect their clinical care. Data were anonymized, personal identifiers were removed, and strict confidentiality was maintained throughout. Because the study relied exclusively on routine clinical and laboratory data, participants were not exposed to any additional risks beyond those inherent to standard AMI management.

RESULTS & FINDINGS

This study demonstrates that stress-induced hyperglycemia is a prevalent and clinically significant phenomenon in non-diabetic patients with acute myocardial infarction.

Table 1: *Demographic and disease histories*

Variable	Category	Frequency (n)	Percent (%)
Age	26–35	18	22.5
	36–45	58	72.5
	46–50	4	5.0
Gender	Male	46	57.5
	Female	34	42.5
Smoking Status	Non Smoker	40	50.0
	Former Smoker	40	50.0
Alcohol Intake	Yes	0	00
	No	80	100.0
Previous history of diabetes mellitus	Yes	32	40.0
	No	48	60.0
Family history of diabetes	Yes	40	50.0
	No	40	50.0
Family history hypertension	Yes	0	00
	No	80	100.0
History of dyslipidemia	Yes	32	40.0
	No	48	60.0
Previous ischemic heart disease	Yes	33	41.3
	No	47	58.8

Fig 1: Demographic and disease histories

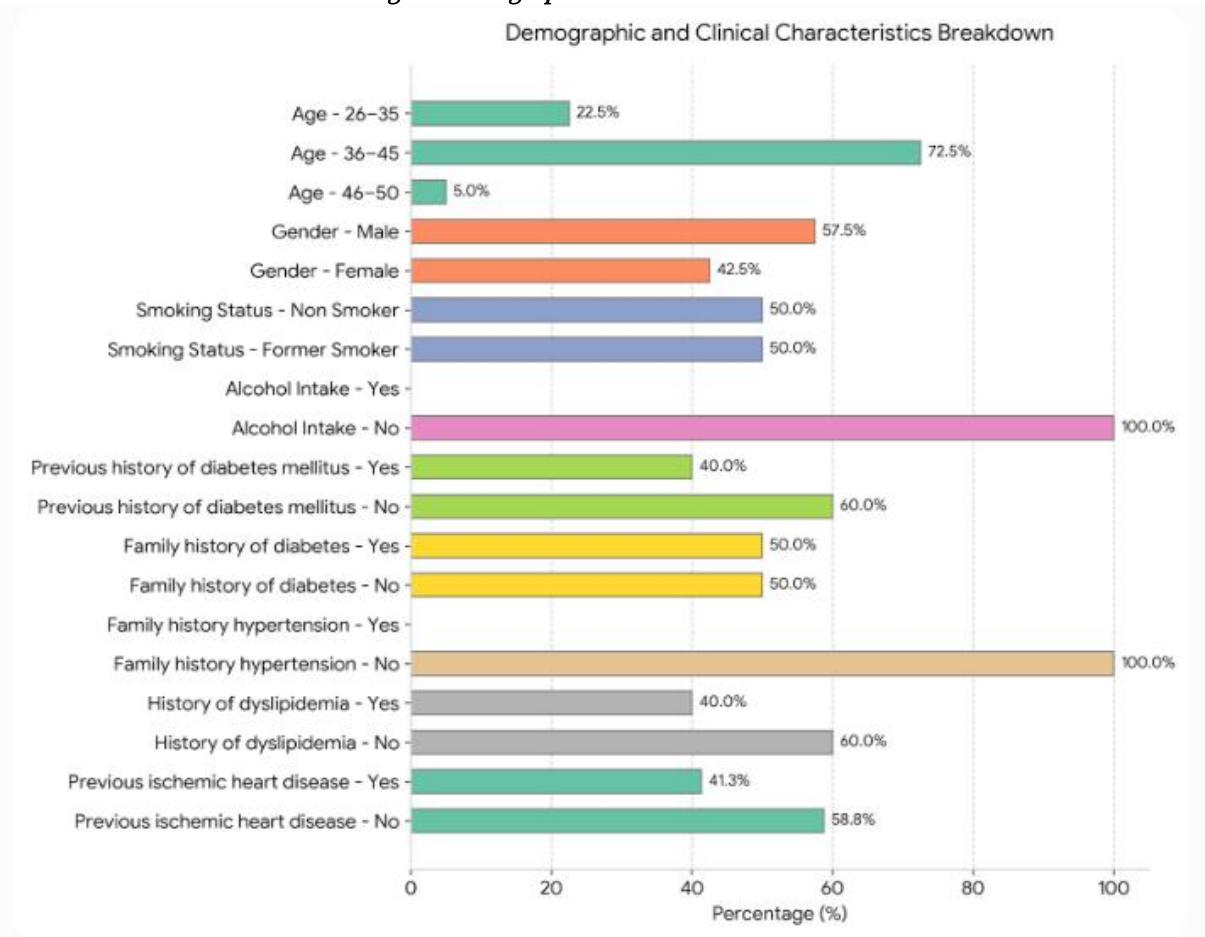

Table 2: *Time and duration of symptoms*

Variable	Category	Frequency (n)	Percent (%)
Time on onset of chest pain	2	12	15.0
	3	12	15.0
	4	20	25.0
	5	36	45.0
Duration of symptoms before hospital arrival	<3 hour	14	17.5
	3–6 hours	36	45.0
	>6 hours	30	37.5
Total (each variable)		80	100.0

Fig 2: Time and duration of symptoms

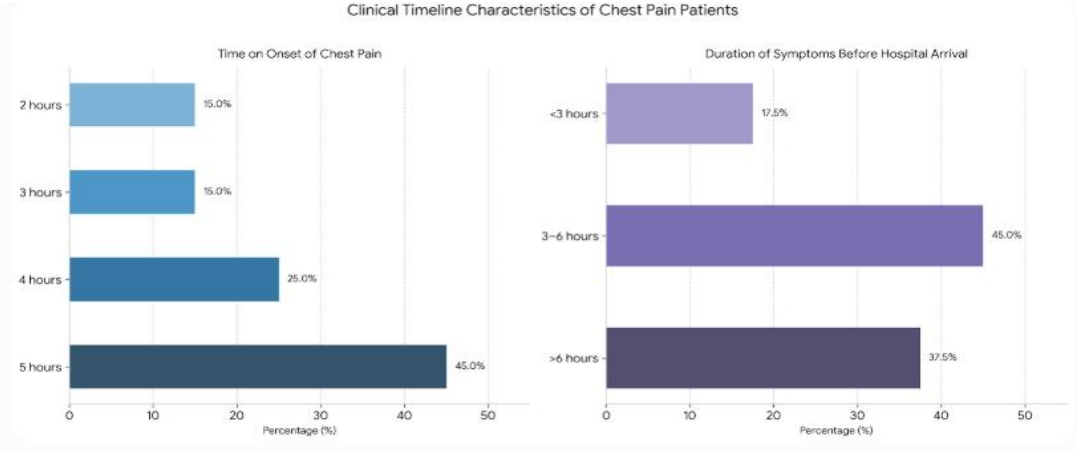

Table 3: *Presenting symptoms and Vital & clinical signs*

Variable	Category	Frequency (n)	Percent (%)
Presenting symptoms	chest pain	21	26.3
	shortness of breath	20	25.0
	Sweating	20	25.0
	nausea/vomiting	16	20.0
	Palpitation	3	3.8
Vital sign at admission	Heart rate	64	80.0
	respiratory rate	9	11.3
	oxygen saturation	7	8.8
Random blood glucose	132	8	10.0
	137	8	10.0
	138	16	20.0
	139	8	10.0
	140	9	11.3
	141	8	10.0
	145	16	20.0
	151	7	8.8
HbA1c Percentage	Yes	47	58.8
	No	33	41.3
Cardiac Troponin Level	.02	24	30.0
	.03	25	31.3
	.04	15	18.8
	.05	8	10.0
	.07	8	10.0
Total (for each variable)		**80**	**100.0**

Table 4: *Diagnostic characteristics*

Variable	Category	Frequency (n)	Percent (%)
MI Type Diagnosed	STEMI	47	58.8
	NSTEMI	33	41.3
ECG changes observed	ST elevation	47	58.8
	ST depression	33	41.3
Reperfusion Therapy	Thrombolysis	47	58.8
	PCI	33	41.3
Admission Glucose >140	Yes	47	58.8
	No	33	41.3
Stress Hyperglycemia Ratio	1	47	58.8

	2	33	41.3
Duration Hyperglycemia	less than 24 hrs	39	48.8
	24–48 hrs	34	42.5
	greater than 48 hrs	7	8.8
Total (each variable)		80	100.0

Fig 4 Diagnostic characteristics

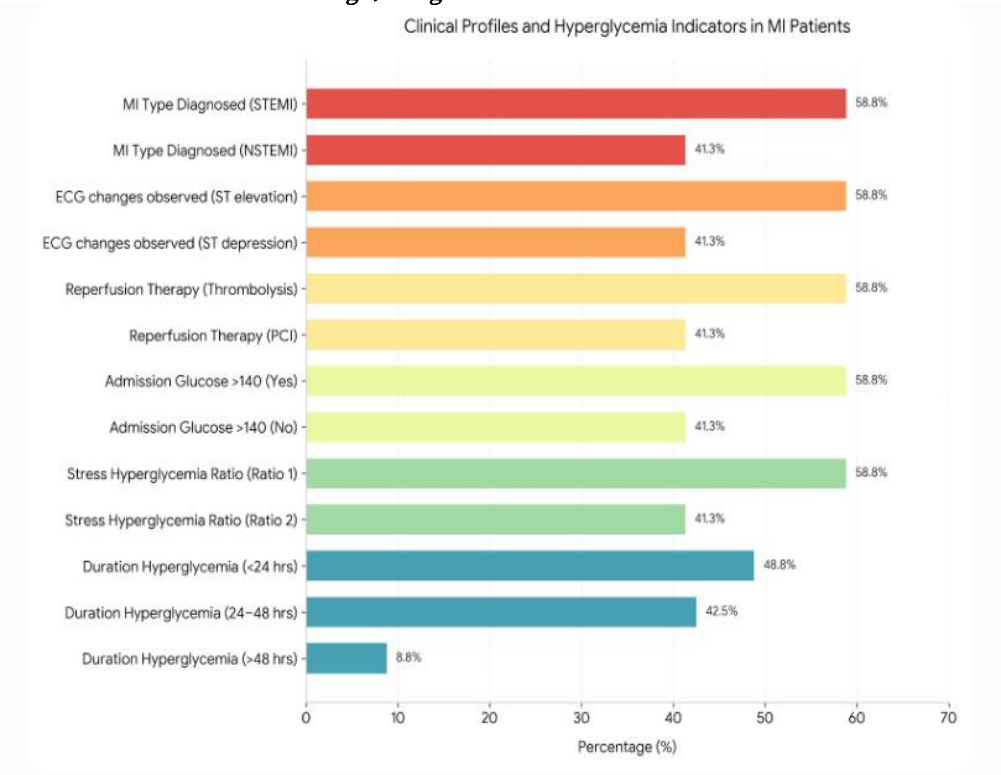

Table 5: *Descriptive analysis*

Variable	N	Minimum	Maximum	Mean	Std. Deviation	Variance
Age_Years	80	31	51	37.26	3.700	13.690
Gender	80	1	2	1.43	0.497	0.247
Residence	80	1	2	1.76	0.428	0.183
Smoking_Status	80	1	2	1.50	0.503	0.253
Alcohol_Intake	80	2	2	2.00	0.000	0.000
Previous history of diabetes mellitus?	80	1	2	1.60	0.493	0.243
Family history of diabetes	80	1	2	1.50	0.503	0.253
History of hypertension	80	2	2	2.00	0.000	0.000
History of dyslipidemia	80	1	2	1.60	0.493	0.243
Previous ischemic heart disease	80	1	2	1.59	0.495	0.245
Time of onset of chest pain	80	2	5	4.00	1.102	1.215
Duration of symptoms before hospital arrival	80	1	3	2.20	0.719	0.516
Presenting symptoms	80	1	5	2.50	1.191	1.418
Vital sign at admission	80	2	4	2.29	0.620	0.385
Random blood glucose	80	132	151	140.46	4.896	23.973

HbA1c_Percentage	80	1	2	1.41	0.495	0.245
Cardiac_Troponin_Level	80	0.02	0.07	0.0349	0.01509	0.000
MI_Type_Diagnosed	80	1	2	1.41	0.495	0.245
ECG changes observed	80	1	2	1.41	0.495	0.245
Reperfusion_Therapy	80	1	2	1.41	0.495	0.245
Admission_Glucose_GT140	80	1	2	1.41	0.495	0.245
Stress_Hyperglycemia_Ratio	89	1	2	1.40	0.494	0.244
Duration_Hyperglycemia	90	1	3	1.59	0.652	0.425

It was carried out on 80 non-diabetic patients who had suffered an acute myocardial infarction. The average age was 37.3 years (31-51) and it was a comparatively young group. The mean of random blood glucose was 140.5 mg/dL (SD: 4.9): 132-151 mg/dl. Cardiac troponin averaged 0.03, which is an indicator of myocardial damage. Seventy-six percent of the patients were male and 57 percent were rural. A half had a family history of diabetes (50%), and dyslipidemia (60%). No patients had hypertension as was expected in the non-diabetic selection. Mean duration of symptoms was 2.2 on 3-point scale (3-6 hours). There was similar distributions in stress hyperglycemia ratio and glucose admission at greater than 140 mg/dl (mean 1.4). The duration of hyperglycemia had a mean of 1.6 (between 24-48 hours) on 3point scale. Most parameters are not varied implying that there is the homogeneity of the study population enhancing internal validity.

Table 6: *Kurtosis analysis*

	Kurtosis	
	Statistic	*Std. Error*
Age_Years	5.370	.532
Gender	-1.954	.532
Residence	-.430	.532
Smoking_Status	-2.052	.532
Previous history of diabetes mellitus?	-1.874	.532
Family history of diabetes	-2.052	.532
History of dyslipidemia	-1.874	.532
Previous ischemic heart disease	-1.917	.532
Time of onset of chest pain	-.898	.532
Duration of symptoms before hospital arrival:	-.999	.532
Presenting symptoms	-1.031	.532
Vital sigh at admission	2.694	.532
Random blood glucose	.112	.532
HbA1c_Percentage	-1.917	.532
Cardiac_Troponin_Level	.512	.532
MI_Type Diagonsed	-1.917	.532
ECG changes observed	-1.917	.532
Reperfusion_Therapy	-1.917	.532
Admission_Glucose_GT140	-1.917	.532
Stress_Hyperglycemia_Ratio	-1.886	.506
Duration_Hyperglycemia	-.558	.503

Table 7: *Anova test*Table

Model		Sum of Squares	Df	Mean Square	F	Sig.
1	Regression	2.794	5	.559	2.468	.040[b]
	Residual	16.756	74	.226		
	Total	19.550	79	-		

The ANOVA results indicate that the regression model is statistically significant, with **F(5, 74) = 2.468** and **p = 0.040**, which is less than the standard threshold of 0.05. This suggests that the

independent variables, taken together, have a significant effect on the dependent variable and the model provides a better fit than a model with no predictors. However, the proportion of variance explained by the model is relatively low, indicating that while the relationship is statistically significant, the explanatory power of the model is limit.

Table 8: *Regressions Coefficients*

Variable	B	Std. Error	Beta	t	Sig.
(Constant)	0.313	0.380	–	0.824	0.413
History of dyslipidemia	0.121	0.115	0.120	1.055	0.295
Duration of symptoms before hospital arrival	0.104	0.075	0.151	1.397	0.167
ECG changes observed	0.139	0.115	0.138	1.212	0.229
Stress_Hyperglycemia_Ratio	0.291	0.111	0.290	2.611	0.011
Duration_Hyperglycemia	0.051	0.084	0.067	0.612	0.542

Table 9: *Correlation coefficients (Pearson r) with significance levels*

Variable 1	Variable 2	Pearson r	p-value	N
Age_Years	Gender	–0.061	0.589	80
Age_Years	Residence	–0.120	0.289	80
Age_Years	Smoking_Status	–0.031	0.788	80
Age_Years	Previous history of diabetes mellitus?	0.072	0.525	80
Age_Years	Previous ischemic heart disease	0.170	0.131	80
Age_Years	Random blood glucose	–0.138	0.222	80
Age_Years	Admission_Glucose_GT140	0.058	0.612	80
Age_Years	Duration_Hyperglycemia	–0.066	0.558	80
Gender	Residence	0.004	0.969	80
Gender	Smoking_Status	–0.152	0.179	80
Gender	Previous history of diabetes mellitus?	0.031	0.785	80
Gender	Previous ischemic heart disease	–0.101	0.371	80
Gender	Random blood glucose	0.027	0.809	80
Gender	Admission_Glucose_GT140	0.153	0.176	80
Gender	Duration_Hyperglycemia	0.102	0.368	80
Residence	Smoking_Status	–0.029	0.796	80
Residence	Previous history of diabetes mellitus?	–0.036	0.751	80
Residence	Previous ischemic heart disease	–0.110	0.333	80
Residence	Random blood glucose	–0.001	0.991	80
Residence	Admission_Glucose_GT140	–0.010	0.932	80
Residence	Duration_Hyperglycemia	0.064	0.574	80
Smoking_Status	Previous history of diabetes mellitus?	0.459**	0.000	80
Smoking_Status	Previous ischemic heart disease	–0.432**	0.000	80
Smoking_Status	Random blood glucose	0.003	0.982	80
Smoking_Status	Admission_Glucose_GT140	–0.838**	0.000	80
Smoking_Status	Duration_Hyperglycemia	0.000	1.000	80
Previous history of diabetes mellitus?	Previous ischemic heart disease	–0.321**	0.004	80
Previous history of diabetes mellitus?	Random blood glucose	0.062	0.586	80
Previous history of diabetes mellitus?	Admission_Glucose_GT140	–0.301**	0.007	80
Previous history of diabetes mellitus?	Duration_Hyperglycemia	0.048	0.675	80
Previous ischemic heart disease	Random blood glucose	0.017	0.881	80
Previous ischemic heart disease	Admission_Glucose_GT140	0.393**	0.000	80

Previous ischemic heart disease	Duration_Hyperglycemia	0.032	0.781	80

The correlation analysis depicted a number of notable associations between variables in the present research of non-diabetic AMI patients. The strongest correlations were shown with smoking status, where there was a moderate positive association with the previous history of diabetes ($r = 0.459$, $p < 0.001$) and strong negative associations with admission glucose exceeding 140 mg/dL ($r = -0.838$, $p < 0.001$) and previous ischemic heart disease ($r = -0.432$ These reversible correlations are probably merely indicative of coding trends and not clinical protective measures. The history of ischemic heart disease in the past positively correlated with admission glucose >140 administration/dL ($r = 0.393$, $p < 0.001$), whereas it negatively correlated with the present status of smoking, as well as the history of diabetes. It is worth noting that random blood glucose did not have significant correlations with any demographic or outcome variables (all $p > 0.05$). This observation has clinical significance- the results substantiate the hypothesis in the study that the absolute glucose markers are not as predictive as a stress hyperglycemia one. The immense correlations between admission glucose exceeding 140 mg/dL (a binary measure of stress hyperglycemia) and various clinical variables, with no correlations observed between continuous values of glucose, support the idea that stress hyperglycemia ratio (SHR) is superior in predicting poor outcomes compared to glucose levels in non-diabetic AMI individuals.

DISCUSSION

In this study, the authors conducted an evaluation of clinical and metabolic features of 80 nondiabetic patients with acute myocardial infarction (AMI) and its connection to demographic, clinical, and biochemical variables. The average age of the cluster was 37.3 0.7 years (minimum 31-maximum -51) suggesting that the population is relatively young and has been hit by AMI. The sample consisted of a large percentage of males (57%) and a significant percentage of rural inhabitants (76%), taking into consideration the demographic data about cardiovascular risk exposure in the studied region. Metabolic evaluation showed that random blood glucose on admission was 140.5 + 4.9mg/dl and above and most patients (58.8) showed glucose levels above 140mg/dl above. The stress hyperglycemia ratio (SHR) that is an indicator of acute glycemic response in the area of physiological stress had an average value of 1.40, which indicated that even in non-diabetic patients, AMI causes a measurable hyperglycemic response [21]. The HbA1c was checked in 58.8 percent of the patients which also justified the ruling out of underlying chronic hyperglycemia. The hyperglycemia lasted no longer than 24 hours in the majority of patients with half having an urgent and stress-induced hyperglycemia of less than 24 hours, and only 8.9 one that lasted over 48 hours, showing the impossibility of prolonged hyperglycemia in this group of patients. The cardiovascular manifestations were in line with the manifestations of acute ACS. The average cardiac troponin levels were 0.0349 + 0.015, which proved the presence of myocardial injury [22]. It was found that 58.8% of patients were diagnosed with STEMI, and 58.8% had ECG changes of ST elevation (similar for NSTEMI), which constituted 41.3%. According to the available treatment modalities, thrombolysis was the primary mode of reperfusion therapy (58.8%), and percutaneous coronary intervention was conducted in 41.3% of cases. Onset of the symptoms occurred most frequently at 4-5 hours and the mean time before hospital presentation was 2.2 on a 3-point scale (around 3-6 hours), showing a moderate delay of symptoms, which could affect the health outcome [23]. The regression analysis revealed that the only significant independent predictor of gender was SHR (= 0.290, p = 0.011), and the other variables (history of dyslipidemia, changes found in the ECG, duration of the symptoms, and duration of hyperglycemia) were not found to be significant. The conclusion implies that there is a gender-specific reaction to hyperglycemia caused by stress, which is why it is necessary to take the issue of sex differences into account when assessing metabolic stress in patients with AMI [24]. The model explained 14.3% of the variation in gender which was a very small yet statistically significant predictor power. Correlation analyses demonstrated that there were intricate interrelationships of clinical variables [25]. It is important to note that only random blood glucose revealed no significant associations with demographic and outcome measures, which

supports the hypotheses of the study that absolute glucose values are less informative in non-democratic AMI patients. On the contrary, results of binary stress hyperglycemia (admission glucose 140 mg/dL) showed a significant correlation with previous ischemic heart disease ($r = 0.393$, $p = 0.001$) and negative relationship with smoking status ($r = -0.838$, $p = 0.001$). These results indicate that stress hyperglycemia, in place of the baseline glucose, offers a more important reading about acute metabolic derangement and possible strain in non-diabetic persons with AMI [26]. In general, the research notes that acute stress hyperglycemia is a widespread occurrence among non-diabetic AMI patients, and temporary increases take place in most of them. SHR seems to be more sensitive and clinically relevant and has been linked to significant cardiovascular parameters with sex-specific relationships [27]. Such results have significant clinical implications by implying that detection and evaluation of stress hyperglycemia at an earlier stage might be used to guide risk stratification and management strategies among patients presenting with AMI but who were otherwise not.

FUTURE RECOMMENDATIONS

Clinicians should routinely assess stress hyperglycemia using the stress hyperglycemia ratio (SHR) in all non-diabetic patients presenting with acute myocardial infarction, as it provides a more accurate reflection of acute metabolic stress than absolute glucose levels. Early monitoring and intervention are also advised, with continuous blood glucose monitoring during the initial 48 hours of hospitalization, since most hyperglycemic episodes are temporary yet can adversely affect immediate outcomes if undetected. Given the significant gender-specific differences observed in SHR, individualized management strategies should be adopted, paying heightened attention to the distinct metabolic responses of both male and female patients. To improve prognostic assessment and guide early clinical decision-making, SHR should be integrated into existing AMI risk stratification protocols. Larger multicenter studies are recommended to explore the long-term cardiovascular impact of stress hyperglycemia in non-diabetic populations and to determine whether early interventions targeting hyperglycemia improve survival and recovery. Finally, healthcare providers should receive education on the importance of stress hyperglycemia in non-diabetic patients, with an emphasis on timely assessment and management to optimize patient care.

LIMITATIONS OF THE STUDY

The single-center design of this study limits the generalizability of the findings to wider populations. A relatively small sample size may have reduced statistical power, potentially affecting the reliability of the results. The possibility of undiagnosed diabetes or prediabetes among patients classified as non-diabetic introduces a risk of classification bias. Reliance on a single admission blood glucose measurement may not fully reflect the patient's overall glycemic status due to physiological fluctuations. Confounding variables such as the severity of myocardial infarction, the acute stress response, and concomitant medications may not have been adequately controlled. Finally, the absence of long-term follow-up precludes evaluation of the long-term outcomes associated with stress hyperglycemia.

CONCLUSION

This study demonstrates that stress-induced hyperglycemia is a prevalent and clinically significant phenomenon in non-diabetic patients with acute myocardial infarction. While baseline random blood glucose levels showed limited predictive value, the stress hyperglycemia ratio (SHR) emerged as a sensitive marker, reflecting the acute metabolic response to myocardial injury. The majority of patients experienced transient hyperglycemia, typically lasting less than 48 hours, highlighting its stress-related nature rather than underlying chronic glycemic dysregulation. Clinical findings indicate that acute STEMI was the predominant presentation, with corresponding ECG changes and thrombolytic therapy being the most frequently employed intervention. Gender-specific differences in SHR suggest that males and females may exhibit differential glycemic responses during acute cardiac stress, emphasizing the need for tailored clinical assessment. Overall, these results underscore the importance of monitoring stress hyperglycemia in non-diabetic AMI patients, as it may serve as a more reliable predictor of acute cardiovascular risk than absolute glucose measurements and could inform early management strategies aimed at improving outcomes.

Conflict of interest

The authors declared no conflict of interest.

Author Contribution

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the study's integrity.

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