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# Assessment of stress hyperglycemia in acute coronary syndrome cases

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**Abstract**
**Background:** In acute coronary syndrome (ACS), admission hyperglycemia is associated with adverse cardiovascular events in patients.
**Objective:** To assess the prognostic value of stress hyperglycemia for the in-hospital outcome of patients admitted due to ACS.
**Methods:** This study was conducted on 100 patients admitted with Acute Myocardial Infarction in a tertiary care hospital. Patients were categorized according to their blood sugar level stress hyperglycemia present and absent.
**Results:** 64% of the patients were diagnosed with non-ST-elevation myocardial infarction NSTEMI and 36% were diagnosed with STEMI. Lesions most frequently (84.9%) in the left anterior descending artery (LAD) followed by right coronary artery 65% followed by Circumflex artery (54%) followed by Left Main Coronary Artery (3%). Death in stress hyperglycemia group was four times that of those who were not having hyperglycemia.
**Conclusion:** Stress hyperglycemia is an independent predictive factor for in-hospital complications after ACS. The results highlight the need to assess admission blood glucose concentration in all patients admitted due to ACS, including nondiabetic ones, aiming at identifying those at higher risk for complications.

**Keywords:** Stress hyperglycemia, STEMI, NON-STEMI, prognosis.

## Introduction
Stress hyperglycemia, defined as a high blood glucose concentration on hospital admission, is a common condition in acute coronary syndrome (ACS) [1], affecting 25% to 50% of patients admitted [2]. In patients with or without diabetes mellitus, stress hyperglycemia is associated with the presence of cardiovascular adverse events and increased mortality (DM) [3-6]. High blood glucose levels are caused by an inflammatory and adrenergic response to ischemic stress, which results in catecholamine release and glycogenolysis [1, 7]. Hyperglycemia causes an increase in free fatty acids, which causes cardiac arrhythmias and insulin resistance, as well as the chemical inactivation of nitric oxide and the production of oxygen reactive species, resulting in oxidative stress, which causes microvascular and endothelial dysfunction, a prothrombotic state, and vascular inflammation [8-10]. It is linked to myocardial metabolic disorders, which cause thrombosis, damage extension, decreased collateral circulation, and ischemic preconditioning [1, 7]. However, the precise pathophysiological mechanism has not been determined [2].
However, there is no agreement on the minimum blood glucose concentration that poses a risk Blood glucose concentrations greater than 110 mg/dL in non-diabetic patients and equal to or greater than 180 mg/dL in diabetic patients, according to Gois [12] and Capes *et al*. [13], are a risk for hospital complications in ACS. Timmer *et al*. [14] considered nondiabetic patients with values above 140 mg/dL. Six-month mortality was significantly higher in patients with acute myocardial infarction (AMI) whose mean blood glucose concentration was greater than 144 mg/dL [15] in the HI-5 study.
The purpose of this study was to determine the prognostic value of stress hyperglycemia in the in-hospital outcome of patients admitted for ACS.

## Material and Methods
This study was conducted on 100 patients admitted with Acute Myocardial Infarction in

SAIMS Hospital-Indore (M.P.). Written consent was taken for participation and was included in this study. We analyzed demographic data, Echocardiography changes, selected lab parameters like Cardiac Enzymes (Troponin T and CPKMB), and blood glucose at the time of admission. We also assessed the presence of Risk factors like history of smoking, obese having BMI >30, history of hypertension with Systolic >114mmHg, and Diastolic >90 mmHg. Myocardial Infarction was to the European Society of Cardiology and the American College of Cardiology, by the presence of clinical symptoms like persistent Angina (>15 min), elevated Cardiac Enzymes (Troponin T and CK-MB), presence of ECG changes indicating acute ischemia (ST-elevation, ST-depression, or T-wave inversion).

## Results

When the ECG of patients at the time of admission to the hospital were reviewed, 34% of the patients (*n* =34) were found to have normal sinus rhythm as their first ECG finding. In the ECG of ST-elevation myocardial infarction (STEMI) patients, the most frequent finding was anterior (30%; 30 patients) and lower wall infarction (30%; 30 patients) (Table I). 64% of the patients (*n* = 64 were diagnosed with non-ST-elevation myocardial infarction NSTEMI and 36% (*n* = 36) were diagnosed with STEMI.

**Table I:** Distribution of ECG findings of patients

| Parameter | Number | Percentage |
|---|---|---|
| Normal sinus rhythm | 34 | 34 |
| Anterior wall MI | 30 | 30 |
| Inferior wall MI | 30 | 30 |
| Posterior wall MI | 1 | 1 |
| Anterior + inferior wall MI | 1 | 1 |
| Anterior + lateral wall MI | 1 | 1 |
| Inferior + posterior wall MI | 1 | 1 |
| Left branch block | 1 | 1 |
| Asystole | 1 | 1 |
| Total (*n*) | 100 | 100.0 |

Table 2 depicts Patients were found to have lesions most frequently (84.9%) in the left anterior descending artery (LAD) followed by right coronary artery 65% followed by Circumflex artery (54%) followed by Left Main Coronary Artery (3%).

**Table 2:** Comparison of coronary angiography results of patients

| Parameter | Lesion Present | | Lesion Absent | |
|---|---|---|---|---|
| | N | % | N | % |
| LMCA lesion | 3 | 3 | 97 | 97 |
| LAD lesion | 84 | 84 | 16 | 16 |
| CXA lesion | 54 | 54 | 46 | 46 |
| RCA lesion | 65 | 65 | 35 | 35 |

**Table 3:** Comparison of patients with and without stress hyperglycemia

| Parameter | | Stress hyperglycemia | | *P*-value |
|---|---|---|---|---|
| | | Yes (%/*n*) (42) | No (%/*n*) (58) | |
| WBC elevation | | 55/23 | 36.3/21 | 0.016 |
| | NSTEMI | 71.5/30 | 60/35 | 0.49 |
| | STEMI | 28.5/12 | 40/23 | |
| LCMA lesion | | 7.1/3 | 1.75/1 | 0.033 |
| Outcome in patients | Death | 9.5/4 | 1.75/1 | 0.002 |
| | Discharge | 89.9/38 | 98.25/57 | |

Table 3 depicts Stress hyperglycemia was found in 42% of the patients studied. A higher WBC value was found in 55% of stress hyperglycemia patients and 36.3% of non-stress hyperglycemia patients. There was a statistically significant difference in high WBC values between the groups of patients with and without stress hyperglycemia. NSTEMI was found in 71.5% of patients with stress hyperglycemia and STEMI in 28.5%; NSTEMI was found in 60% of patients without stress hyperglycemia and STEMI in 40%. In patients with stress hyperglycemia, the NSTEMI detection rate was statistically significantly higher than the rate of STEMI detection. 7.1% of patients with stress hyperglycemia and 1.75% of patients without stress hyperglycemia had left main coronary artery stenosis (LMCA). When patients with and without stress hyperglycemia were compared, there was a statistically significant difference in stenosis in the LMCA. It was discovered that 9.5% of patients with stress-related hyperglycemia died, while only 1.75% of patients without stress-related hyperglycemia died. There was a statistically significant difference in death rates between patients with stress-related hyperglycemia and those who did not (p 0.05).

## Discussion

Cardiovascular diseases are the major cause of death in industrialised nations and will be in developing nations by 2020 [16]. Coronary artery disease causes the highest number of deaths among cardiovascular diseases [17]. Coronary artery disease is a primary cause of death and morbidity .

It's Turkey's top killer [19, 20]. Many studies show that stress hyperglycemia increases mortality and hospitalisation time in patients with acute cardiovascular illness [21–23].

Rafael *et al*. studied 834 individuals with a mean age of 64 13 (25–94) [24]. The mean age was 63.3 13.8 in another study [25]. Our study's average age matched previous research. Aggarwal *et al*. studied non-diabetic patients with myocardial infarction and found a 4:1 male-to-female ratio [22]. Similar research revealed a 3:1 female-to-male ratio [24]. After menopause, women have a higher risk of cardiovascular disease due to lower oestrogen levels. The higher rate of women in our study data may be connected to female gender risk. Our country's women live longer than men [26]. Most patients were over 60. Increasing age is another reason why more cardiac incidents occur in women.

WBC rise in AMI promotes a bigger thrombus, greater inflammation in minor lesions, and more heart failure, which may need aggressive treatment [27, 28]. Açkel associated high WBC early admission with severe heart failure and high hospital mortality [29]. In our study, stress hyperglycemic AMI patients had increased WBC. In our investigation, stress hyperglycemia increased mortality in AMI patients.

The anterior wall myocardium is supplied by the LAD, and the lower wall myocardium is nourished by the CXA in 85% of instances and the RCA in 15%. In most circumstances, the RCA supplies most of the posterior wall myocardium's blood [30]. 158 patients in our study with aberrant ECGs had anterior or posterior wall MI. Our patients' angiograms showed LAD and CXA lesions. According to this angiography, patients' ECGs should reveal intense anterior and inferior wall MI.

Left main coronary artery lesions occur in 4% to 10% of individuals having coronary angiography [31, 32]. 3.5% of research participants had left main coronary artery stenosis.

Angiographic results of stenosis patients also exhibited LMCA and RCA stenosis. Most coronary angiography and stenting lesions were in the LAD [33]. According to coronary artery involvement, the lesion distribution is identical to that reported in the literature. In our investigation, the LAD was most prevalent. Comparing male and female coronary angiography findings was not statistically significant.

In one study, hyperglycemia at the time of emergency service admission was a substantial predictor of mortality in STEMI patients [24]. Stress hyperglycemia after MI in non-diabetic patients may predict risk categorization and treatment choices [34]. Budzyski *et al*. showed that patients with abnormal HDL cholesterol had increased LDL, non-HDL, triglycerides, and fasting blood sugars [35]. Fasting blood glucose levels were high in both live and dying patients in research by Dönmez *et al*. on 86 patients who underwent transcatheter aortic valve implantation [36].

In our study, stress hyperglycemia patients had a greater mortality rate than those without it. In normal aerobic conditions, free fatty acid oxidation helps meet cardiac energy needs [37]. Oxygen in veins and on myoglobin may last 2 to 6 heartbeats. In humans, contractile function decreases in the 10th pulse after coronary occlusion [38]. Thus, the myocardial oxygen reserve is depleted within seconds of ischemia, and oxidative phosphorylation, electron transport, and mitochondrial ATP synthesis stop below 5 mm Hg. Reduced mitochondrial activity causes anaerobic glycolysis to replace aerobic metabolism [39]. Thirty minutes after AMI, stress factors increase plasma epinephrine and free fatty acids (FFA) due to TNF- and IL-6. Increased plasma FFA/albumin ratio and lack of FFA - oxidation in cardiac mitochondria promote acyl-CoA and acylcarnitine accumulation. These chemicals activate Ca channels and block the sarcoplasmic Ca pump, Na-Ca and Na-K pumps. Increasing cytosolic Ca. Increased Ca causes tachycardia [40–42]. Arrhythmias are a common AMI complication. ventricular fibrillation is deadly. Stress-induced hyperglycemia may produce arrhythmias.

AMI has necrotic and apoptotic dead tissue in the infarct region [43]. Hyperglycemia-formed free oxygen radicals (FOR) cause apoptosis in cardiomyocytes via cytochrome c and KASPAS-3 [44]. Insulin promotes endothelial nitric oxide (NO) production via PI-3 kinase. NO-induced vasodilation increases tissue glucose absorption. Nitric oxide reduces adhesion molecules such as VCAM-1, E-selectin, and ICAM-1 (ICAM-1). NO decreases platelet adhesion and vascular wall contact and promotes prostacyclin's influence on platelet aggregation. NO's vasculoprotective properties lower insulin in insulin resistance. Accelerating vascular smooth muscle cell proliferation, migration, and PAI-1 production [45]. Clinical experiments demonstrate that insulin resistance may affect PAI-1 expression and regulation, a coronary artery disease risk factor [46]. In hyperinsulinemia, elevated PAI-1 levels may decrease fibrinolytic activity and coagulation [47].

LMCA lesions are rare yet clinically disastrous in unprotected coronary artery disease patients. Cardiogenic shock and a significant death rate have been linked to this lesion [48–52]. 7 of 9 individuals with LMCA stenosis had stress hyperglycemia, a statistically significant difference from patients without stress hyperglycemia. LMCA lesions cause widespread cardiac involvement, ischemia, necrosis, and significant mortality. Increased glucose levels may increase mortality via altering pathophysiology.

## Conclusion

Hyperglycemia is caused by elevated stress hormones, an activated HPA axis, increased cytokine levels, and peripheral insulin resistance. This adaptive mechanism is detrimental to uncontrollable hyperglycemia over time due to undesirable effects on cellular and metabolic levels (mitochondrial damage, oxidative stress-related cell damage, endothelial damage, and cardiac potassium channel dysfunction) and affects the course of critical illness.

## Conflict of Interest

Not available

## Financial Support

Not available

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