Admission Hyperglycemia in Patients with Acute Myocardial Infarction in Tikrit Teaching Hospital, Tikrit, Iraq

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Abstract

Background: Hyperglycemia is common in critically ill patients; it is no longer considered a benign condition in patients with critical illnesses. Hyperglycemia after acute myocardial infarction (AMI) is associated with an increased risk of in-hospital and long term mortalities in patients with and without diabetes. Although the mechanisms underlying this association are not fully understood, this may be due to impaired left ventricular function, increased incidence of the no-reflow phenomenon, and a tendency for arrhythmias. The aims of this study are to determine the frequency of hyperglycemia during AMI and to show the predictors of adverse events following AMI.

Patients and Methods: Hospital based cross-sectional study. That includes 160 patients with AMI who admitted to CCU in the Tikrit Teaching Hospital from December 2013 to July 2014. Hyperglycemia was defined as admission or non-fasting plasma glucose level equal or above 140 mg/dl (7.8 mmol/L) regardless of past history of diabetes. Patients were divided into 3 groups.

Group 1 (n=77 [48.1%]): Normoglycemic patients: Non-diabetic patients, without stress hyperglycemia and admission plasma glucose level <140 mg/dl (7.8 mmol/L). Group 2 (n=33 [20.6%]): Non-diabetic patients with stress hyperglycemia with admission plasma glucose level equal or above 140 mg/dl (7.8 mmol/L) and Group 3 (n=50 [31.3%]): Diabetic patients, who had a history of treated diabetes mellitus or HbA1c measurement was ≥ 6.5%.

Results: The results indicate that the mean age in normoglycemic, stress hyperglycemic and diabetes mellitus groups were 55.3, 55.1 and 54.4 years respectively with more males' affection in all three groups without significant difference among them. The anterior wall was the most common site of AMI in all 3 groups but without significant difference among them. Killip class 2 was significantly higher in DM group, while Killip class 3 was significantly higher in stress hyperglycemic group. Odds ration and confidence intervals of many variables as predictors of adverse outcome obtained from this study.

Conclusion: This study indicates that admission hyperglycemia is a common finding in patients with AMI and admission hyperglycemia is a predictor of adverse events following AMI.

plasma glucose level equal or above 140 mg/dl (7.8 mmol/L) regardless of past history.

Key Words: Admission Hyperglycemia, Acute Myocardial Infarction, Tikrit, Iraq

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Introduction

Stress hyperglycemia: Is a medical term referring to an elevation of the blood glucose during stressful condition in severely ill patient without evidence of previous diabetes. American Diabetes Association and American Association of Clinical Endocrinologists defined stress hyperglycemia as any plasma glucose concentration ≥ 7.8 mmol/l (140 mg/dl) without evidence of previous diabetes. Although stress hyperglycemia typically resolves as the acute illness or surgical stress abates, it is important to identify and track patients as 60% of patients admitted with stress hyperglycemia had confirmed diabetes at 1 year.(1,2)

Mechanism of stress hypenmrglycemia: Hyperglycemia is a frequent manifestation of critical and surgical illness, resulting from the acute metabolic and hormonal changes associated with the response to injury and stress. Acute illness, surgery, and trauma raise levels of counterregulatory hormones such as glucagon, epinephrine, cortisol, and growth hormone.(2)

The counterregulatory response results in a number of alterations in carbohydrate metabolism; including increase insulin resistance, increased hepatic glucose production, impaired peripheral glucose utilization, enhanced lipolysis, increasing free fatty acids (FFAs) concentration, stimulate protein catabolism and increased circulating amino acids concentration, providing precursors for gluconeogenesis. In addition, acute stress increases pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-6, and IL-1, which increase insulin resistance by interfering with insulin signaling and tissue glucose uptake, the end result is the appearance of hyperglycemia.(1,3)

Hyperglycemia in acute myocardial infarction (AMI): Hyperglycemia during AMI is associated with an increased risk of in-hospital and long term mortalities in patients with and without diabetes. Although the mechanisms underlying this association are not fully understood, evidence that the use of insulin to lower glucose concentrations decreases mortality in diabetic patients who have AMI, suggests that hyperglycemia is not simply an epiphenomenon of a stress response. Unfavorable effects of high blood glucose levels in AMI involve impaired left ventricular function, increased incidence of the no-reflow phenomenon, and a tendency for arrhythmias. Several mechanisms implicated in the detrimental impact of hyperglycemia during acute myocardial ischemia have been postulated, i.e., enhanced oxidative stress, the activation of blood coagulation and platelets, stimulation of inflammation, and endothelial cell dysfunction. Consequently, hyperglycemia at the time of AMI may be an important and potentially modifiable risk factor for poor outcome.(4,5)

Effect of hyperglycemia on ischemic myocardial tissue: In patients with ischemic cardiovascular events, high FFAs levels can aggravate ischemia/reperfusion damage by limiting the ability of cardiac muscle to uptake glucose for anaerobic metabolism. FFAs are toxic to an ischemic myocardium leading to cardiac arrhythmias, sympathetic overactivity, increased blood pressure, oxidative stress and endothelial dysfunction. Increased FFAs levels also produce dose dependent

insulin resistance in peripheral tissues and increase hepatic glucose output in both diabetic and non-diabetic individuals. Acute hyperglycemia may induce cardiac myocyte death through apoptosis or by exaggerating ischemia-reperfusion cellular injury. It also has deleterious effect on endothelial function by suppressing formation of nitric oxide (NO) and impairing endothelium-dependent flow mediated dilation. (1,6)

In humans, elevation of glucose has been associated with increased levels of C-reactive protein, interleukin-6 (IL-6), interleukin-18 (IL-18), and tumor necrosis factor alpha (TNF-α). Elevations of these various inflammatory factors have been linked to detrimental vascular effects. For example, TNF-α extends the area of necrosis following AMI by inducing myocardiocyte apoptosis and its levels are correlate with severity of cardiac dysfunction. IL-18 has been proposed to destabilize atherosclerotic plaques, leading to acute coronary syndrome.(1,7) Interestingly, recent data from human studies suggest that acute fluctuations in glucose levels may have an even more powerful impact on oxidative stress than chronic, sustained hyperglycemia.(6) The aims of this study are to: determine the frequency of hyperglycemia during AMI, study the association between hyperglycemia and inflammatory markers including s.fibrinogen and white cell count (WBC), assess the functional status of patients with hyperglycemia according to Killip classification, and show the predictors of adverse events following AMI.

Patient and Methods

Patients: Hospital based cross-sectional study that includes 160 patients with AMI who were admitted to coronary care unit (CCU) in the Tikrit Teaching Hospital from December 2013 to July 2014. The diagnosis of AMI was made according to WHO and American Heart Association criteria, if the patient fulfilled two of the following criteria:(8,9,10)

1. A history of prolonged ischemic chest pain (> 30 minutes).
2. The presence of an elevated serum level of biochemical cardiac marker (Troponin I).
3. Persistent electrocardiographic changes of AMI.

Hyperglycemia was defined as admission or non-fasting glucose level equal or above 140 mg/dl (7.8 mmol/L) regardless of past history of diabetes. The diagnosis of diabetes in hyperglycemic patients was depended on the patient's reported history of treated diabetes mellitus or the presence of HbA1c ≥ 6.5%. A fasting blood sugar of <100 mg/dl, random blood sugar <140 mg/dl were considered normal.(11,12,13) Patients in this study were classified into 3 groups based on their history of DM and their plasma glucose levels on admission:

Group 1 (n=77 [48.1%]): Normoglycemic patients: Non-diabetic patients without stress hyperglycemia, admission plasma glucose level <140 mg/dl (7.8 mmol/L)

Group 2 (n=33 [20.6%]): Non-diabetic patients with stress hyperglycemia, admission plasma glucose level equal or above 140 mg/dl (7.8 mmol/L)

Group 3 (n=50 [31.3%]): Diabetic patients, who had a history of treated diabetes mellitus or HbA1c ≥ 6.5%.

Killip classification of heart failure (HF) following AMI: Killip classification is a simple clinical tool used to assess the severity of cardiac failure following AMI. Classified into 4 classes (14,15)

1. Class I: No evidence of HF.
2. Class II: Mild to moderate HF: basal crepitation, 3rd heart sound and increased JVP.
3. Class III: Overt pulmonary edema.
4. Class IV: Cardiogenic shock (systolic B.P < 90 mm Hg with oliguria, cyanosis or sweating).

Data collection: The data were collected by data collection sheet that is designed for each patient, which include the demographic characteristic of patients, detailed history information, findings of clinical examination and results of investigations. All patients were followed up during hospitalization for presence of complications and outcome. The study was accepted by ethical committee of College of Medicine, Tikrit University and hospital administration. All patients were instructed about the study and their agreements were taken.

Statistical analysis: The collected data was organized, tabulated, and statistically analyzed using statistical package for social science (SPSS) version 18. The values were reported as mean ± SD and frequencies expressed as percent. Chi ($\chi 2$) square test was used to compare the difference among of variables. The odds ratios with the confidence intervals (CI) were calculated for the following variables: Admission plasma glucose, HbA1C, BMI, DM, hypertension, and smoking to predict the probability of adverse outcome following AMI. P value of ≤ 0.05 was regarded as statistically significant.

Results

Out of the total 160 patients with AMI. 126 (78.8%) patients presented with ST elevation myocardial infarction (STEMI), while 34 (21.2%) patients presented as non- ST elevation myocardial infarction. In this study 77 (48.1%) patients were normoglycemic, 33 (20.6%) patients have stress hyperglycemia and 50 (31.3%) patients were diabetic.

The characteristics of patients are present in Table 1. The mean age in normoglycemic, stress hyperglycemia and diabetes mellitus groups were 55.3, 55.1 and 54.4 years respectively with more males' affection in all three groups without significant difference among them.

The most common risk factor of AMI was smoking which was present in 88.3%, 72.7% and 68% in the three groups respectively. There were significant differences among study groups regarding smoking, hypertension, family history of diabetes mellitus (DM) and obesity.

Regarding therapeutic agents during hospitalization, there was frequent use of B-blockers in normoglycemic group than others and more use of diuretics and insulin in stress hyperglycemia and diabetes mellitus groups than normoglycemic group.

The findings of patients' investigations are present in Table 2. As expected patients with stress hyperglycemia and diabetes mellitus has significantly higher plasma glucose level compared to normoglycemic group.

with excess of adrenaline, glucagon, growth hormone and steroid hormones with activation of circulating cytokines such as tumor necrosis factor alpha that may lead to reduction of insulin sensitivity, thus increase serum level of glucose and FFAs.(21,22)

Furthermore in this study, the percent of diabetic patients was 31.3% and the prevalence of diabetes in the same age group in general population is close to 12%. It is in accordance with other study done by Mansor AA et al of patients with AMI were 25% having diabetes.(16) Other study done by Sahibzada P et al show much higher prevalence of diabetes in association with AMI as 37% in Asian patients than that reported in West as (12-26%), this because Asian population tend to develop diabetes at lower level of BMI than western.(23) However many previous studies show that diabetic patients with ischemic heart disease may have worsen outcome for many reasons, including more advanced atherosclerosis, diabetic cardiomyopathy, autonomic dysfunction and decrease endogenous fibrinolytic activity.(24)

The present study observe more males affection by AMI in 3 groups of patients, similar finding was seen by Musa A et al, which seems to be related genetic factors, increase habit of smoking and increase the psychological and physical stress among males in our country.(25)

The mean WBC count in this study reveals higher level in stress hyperglycemic group than other groups. Marfflla R et al found that hyperglycemic patients had higher circulating level of IL8 and CRP compared to normoglycemic patients. That lead to increase inflammatory process and WBC activation mainly lymphocyte and natural killer cells. The recent demonstration of higher level of natural killer cells in symptomatic plaques compared with asymptomatic plaques suggest a major role of these cells in atherosclerotic plaque destabilization leading to acute coronary syndrome. Moreover Marfflla R et al reveals that inflammatory markers and T-cells activation are lesser response in patients with diabetes who receiving drugs treatment for hyperglycemia before AMI.(4)

There was no significant difference regarding the site of AMI among study groups. Anterior wall was the most common site of infarction in the 3 groups of patient as 49.3%, 48.5% and 42% respectively. The second common site of infarction was an inferior wall. These results were in agreement with those of Tipoo FA et al, who reported that most of the infarcts were anterior (56.6%) in location.(26) Culic V et al, reported it as 47.7%.((27) Left anterior descending artery (LADA) that supplies the anterior cardiac wall seems to be more susceptible to development of atherosclerosis in comparison to right coronary and left circumflex arteries. LADA is exposed to more powerful biochemical and hemodynamic stress resulting from the contraction of the heart, which may be related to greater endothelial and artery wall damage favoring development of atherosclerotic process. Therefore, it seems likely that more extensive atherosclerotic lesions underlay anterior infarction.(28)

Moreover, the clinical assessment for evidence of heart failure in this study was done by applying Killip classification, and patients with Killip class 2, 3 and 4 had

The mean HbA1c, blood urea and serum creatinine were significantly higher in diabetic group. Serum fibrinogen level was higher in stress hyperglycemic group than other groups but without significant difference among them. Furthermore the white blood cell count was significantly higher in stress hyperglycemic group than other groups.

The locations of AMI in the 3 groups of patients are present in Table 3. The anterior wall was the most common site of AMI, which was present in 49.3%, 48.5% and 42% in the 3 groups respectively, but without significant difference among them. This followed by inferior wall AMI, which was presented as 26%, 27.3% and 34% in the 3 groups respectively then followed by lateral wall AMI. While the posterior wall and right ventricular infarction were less frequent in this study.

The clinical manifestations of heart failure following AMI were divided according to Killip classification into 4 classes as shown in Table 4. Killip class 2 was more statistically frequent in diabetic group as 16% compared to 7.8% and 3% in normoglycemic and stress hyperglycemic groups respectively. While Killip class 3 was significantly higher in stress hyperglycemic group as 12.2% compared to 1.3% and 4% in normoglycemic and diabetic groups respectively.

Table 5. Shows the results of odds ration and confidence intervals of many variables as predictors of adverse outcome in this study. The admission hyperglycemia, BMI $\geq$ 29 kg/m2 and smoking were significant predictors of adverse events following AMI as a p- value (< 0.05). The Age $\geq$ 50 years, HbA1c % $\geq$ 6.5, history of hypertension and DM were not statistically significant.

Table 6. Show the results of corrected QT (QTc) interval in the 3 groups of patients. The values of prolonged QTc intervals (> 440ms) were slightly greater in patients with stress hyperglycemic and diabetic groups as 21.3% and 22% respectively compared to 18.2% in normoglycemic group, but without statistical difference.

Discussion

The admission plasma glucose in critically ill patients of 140 mg/dl (7.8 mmol/l) and above was chosen to select the patients with hyperglycemia. This target glucose level appear to be associated with greatest increase in short term mortality risk with or without established diabetes mellitus, so it is reasonable to consider admission random plasma glucose level of 140 mg/dl and more as the definition of hyperglycemia in this study.(16) Furthermore previous history of diabetes mellitus and measurement of HbA1c% during the period of hospitalization provide the opportunity to differentiate patients with stress hyperglycemia from those with DM.(1,17)

In this study, out of 160 patients with AMI, stress hyperglycemia occur in 33 (20.6%) patients, this result goes with many previous results like Zeijko SA et al, present as 20% of patients,(18) Modenesi RF et al as 26.4% (19) and Marfella R et al as 29% patients.(4) Moreover Nordin C et al in a retrospective study of patients admitted with ACS observed as 38% prevalence of stress hyperglycemia and the majority of these hyperglycemic episodes resolved before discharge from the hospital.(20) Stress hyperglycemia in the initial phase of AMI appears to be related to stress mechanism and is a reflection of relative insulin deficiency in association

higher risk profiles and rates of major adverse clinical events following AMI. Moreover there is more severe angiographic coronary artery disease and higher incidence of ventricular dysfunction. The finding in this study were consistent with previous data from Western countries, that show higher Killip class in hyperglycemic patients than normoglycemic patients with significant predictor of short term mortality in acute coronary syndrome.(14,15)

The present study reveals that admission hyperglycemia, BMI ≥ 29 kg/m2 and smoking are significant predictors of adverse outcome in patients with AMI. This was goes with many other studies like Schneider CA et al, and Wong VW et al, show stress hyperglycemia as a predictor of in hospital mortality.(29,30) Moreover this high predictive value for adverse outcome may be due to high adrenergic stimulation following AMI.(13) The high level of stress hyperglycemia has associated with local thrombin generation and platelet activation and altered clot features and abolished ischemic area completely in patients with ACS. (31)

Furthermore Kersten TR et al have shown decrease collateral circulation and increase infarct size with elevation of systolic and diastolic blood pressure and prolongation of QT interval in the sitting of sever hyperglycemia.(32) The BMI ≥ 29 kg/m2 appear to be another predictor of adverse outcome in this study, The same result also reported by Wolk R et al as high BMI on admission is related positively with evidence of ACS, which may be due to endothelial dysfunction.(33)

Moreover one of the important non-invasive methods for assessment of cardiovascular risk is measurement of QTc interval on standard 12 lead ECG which may reflect increased inhomogenety of myocardial repolarization. The QTc prolongation has been proposed as a marker of cardiovascular risk in the clinical setting and it has been particularly associated with arrhythmias, sudden cardiac death and poor survival in apparently healthy subjects. In the present study, the percent of QTc interval prolongation was higher in stress hyperglycemia and diabetic groups but without significant difference. Other study done by Aida JC et al, observe a significant higher percent of prolonged QTc interval in diabetic compared to non diabetic patients as (10.1% vs 4%) and this difference related to cardiac autonomic neuropathy presented in diabetic patients.(34) However other study has not found any significant association. (35)

The present study concluded that admission hyperglycemia is a common finding in patients with AMI. Admission hyperglycemia, smoking and BMI are predictors of adverse outcome in patients with AMI. Higher Killip classes of heart failure are more significantly associated with hyperglycemia. Stress hyperglycemia significantly associated with higher WBC count which may linked to altered immune response. The current study recommended that plasma glucose level should be monitored closely in patients with AMI regardless history of diabetes and further researches and follow up are required to observe the long term complications of hyperglycemia in patients with AMI.

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Table 1. Study characteristics in 3 groups of patients (n=160).

Characteristic	Normoglycemia N=77 (48.1%)	Stress hyperglycemia N= 33 (20.6%)	DM N= 50 (31.3%)	P value
Age(years) mean ±SD	55.3 ± 11.278	55.1 ± 13.2	54.4 ± 10.25	> 0.05
Gender (male)	59 (76.6%)	27 (81.8%)	34 (68%)	> 0.05
Smoking	68 (88.3%)	24 (72.7%)	34 (68%)	< 0.05
Hypertension	40 (51.9%)	12 (36.3%)	32 (64%)	< 0.05
Previous IHD	12 (15.5%)	7 (21.2%)	11 (22%)	> 0.05
Family history of DM	8 (10.3%)	5 (15.1%)	16 (32%)	< 0.05
Hyperlipidemia	3 (3.8%)	2 (6%)	2 (4%)	> 0.05
Obesity	6 (7.8%)	4 (12.1%)	16 (32%)	< 0.05
Therapies in hospital :				
B. blockers	64 (83.1%)	21 (63.6%)	33 (66%)	< 0.05
ACEI or ARB	72 (93.5%)	29 (87.8%)	46 (92%)	> 0.05
Aspirin	77 (100%)	33 (100%)	50 (100%)	NA
Statins	76 (98.7%)	33 (100%)	49 (98%)	> 0.05
Thrombolytic	21 (27.2%)	7 (21.2%)	14 (28%)	> 0.05
Nitrates	74 (96.1%)	33 (100%)	46 (92%)	> 0.05
Diuretic	2 (1.3%)	3 (9%)	9 (18%)	< 0.05
Insulin	0	33 (100%)	50 (100%)	< 0.05

Table 2. The results of investigation in 3 groups of patients (n=160).

Investigation	Normoglycemia n= 77 (48.1%)	Stress hyperglycemia n= 33 (20.6%)	DM n= 50 (31.3%)	P value
APG (mg/dl)	121.2 ± 11.1	216.6 ± 53.56	266.8 ± 66.2	< 0.05
HbA1c %	4.4 ± 0.34	4.6 ± 0.45	7.1 ± 0.99	< 0.05
Fibrinogen (mg/dl)	372.2 ± 73.9	388.3 ± 77.5	381.8 ± 73.8	> 0.05
Urea (mg/dl)	45.3 ± 16.8	48.3 ± 12.3	68.5 ± 30.3	< 0.05
Creatinine (mg/dl)	1 ± 0.3	1 ± 0.2	1.4 ± 0.6	< 0.05
WBC ($\times 10^9$/L)	10 ± 1.4	10.9 ± 1.3	10.3 ± 1.4	< 0.05
Ejection fraction (%)	56 ± 5.4	55.7 ± 4.8	53.7 ± 5.9	> 0.05

Table 3. Location of AMI in 3 groups of patients (n=160).

Location of AMI	Normoglycemia n= 77 (48.1%)	Stress hyperglycemia n= 33 (20.6%)	DM n= 50 (31.3%)	P value
Anterior	38 (49.3%)	16 (48.5%)	21 (42%)	> 0.05
Inferior	20 (26%)	9 (27.3%)	17 (34%)	> 0.05
Lateral	15 (19.5%)	5 (15.2%)	9 (18%)	> 0.05
Posterior	2 (2.6%)	1 (3%)	2 (4%)	> 0.05
Right ventricular	2 (2.6%)	2 (6%)	1 (2%)	> 0.05

Table 4. Killip classes of heart failure in 3 groups of patients (n=160).

Class	Normoglycemia n= 77 (48.1%)	Stress hyperglycemia n= 33 (20.6%)	DM n= 50 (31.3%)	P value
Class 1	70 (90.9%)	27 (81.8%)	39 (78%)	> 0.05
Class 2	6 (7.8%)	1 (3%)	8 (16%)	< 0.05
Class 3	1 (1.3%)	4 (12.2%)	2 (4%)	< 0.05
Class 4	0	1 (3%)	1 (2%)	> 0.05

Table 5. Odds Ratio and Confidence Intervals in patients with AMI (n=160).

Predictors	Odds Ratio	Confidence Intervals	P value
Age ≥ 50 years	1.79	0.68-4.7	> 0.05
APG ≥140 (mg/dl)	0.71	0.64-0.8	< 0.05
HbA1c % ≥ 6.5	0.98	0.4-2.3	> 0.05
BMI ≥ 29 kg/m^2	0.07	0.019-0.23	< 0.05
DM	0.9	0.4-2.1	> 0.05
Hypertension	1.2	0.5-2.5	> 0.05
Smoking	0.3	0.1-0.6	< 0.05

Table 6. Analysis of QTc intervals on standard ECG in 3 groups of patients (n=160).

QTc intervals	Normoglycemia n= 77 (48.1%)	Stress hyperglycemia n= 33 (20.6%)	DM n= 50 (31.3%)	P value
Normal QTc intervals (≤440 ms)	63 (81.8%)	26 (78.7%)	39 (78%)	> 0.05
Prolong QTc intervals (>440 ms)	14 (18.2%)	7 (21.3%)	11 (22%)	> 0.05