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Relationship between admission random blood glucose, fasting blood glucose, with severity of coronary lesion and hospital outcomes in patients admitted with ST elevation myocardial infarction

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Abstract
Background: This research addressed the relationships between admission random blood glucose (RBG), fasting blood glucose (FBG), and severity of coronary artery disease (CAD) "assessed by Gensini score "and in hospital outcomes in patient admitted with ST elevated Myocardial Infarction (STEMI).
Methods: The research was conducted on 100 cases who were recruited from Cardiology department in Tanta University presented with STEMI and underwent coronary angiography either primary PCI or Pharmaco-invasive, the cases were categorized firstly, according to RBG into two groups: Group 1: Non hyperglycemia group (<200 mg/dl) and Group 2: Hyperglycemia group (>200 mg/dl). Then after 8hours from admission FBG was tested "regarding FBG Patient is fasting but to be well hydrated" and accordingly the cases were further sub-divided into two groups: Group I: Non-FBG elevated (<126 mg/dl) and Group II: FBG elevated (>126 mg/dl).
Results: It was shown that there was a significant difference between the hyperglycemic and FBG elevated groups in terms of severity of CAD and In-hospital mortality where both the hyperglycemic and the FBG elevated cases had more extensive CAD and higher Gensini score. Also, the occurrence of major adverse cardiovascular events was higher in these two groups in comparison to their non-hyperglycemic, non FBG elevated counterparts. There was statistically significant positive correlation between Gensini score and RBS and fasting blood glucose level (p<0.001).
Conclusions: Our results highlighted the value of both RBS at admission and FBS in correlation to both severity of CAD and in-hospital outcomes.

Keywords: Random blood glucose, fasting blood glucose, coronary lesion, ST elevation myocardial infarction

Introduction
A close link exists between DM and cardiovascular disease (CVD), Type 2 diabetes is regarded as having an equivalent risk of coronary heart disease by the Adult Treatment Panel III of the US National Cholesterol Education Program. As a result, people with type 2 diabetes are just as likely to experience significant coronary events as people with coronary heart disease [1].
Since it is anticipated that the prevalence of DM will rise over the next 30 years and that up to 75% of these cases would die from coronary heart disease, the risk of CVD in cases with DM is at least 2-4 times greater than that faced by non-diabetics of comparable age.
Contrary to non-diabetic cases, cases with DM are at increased risk of developing coronary heart disease (CAD) as an acute coronary syndrome (ACS) with a more difficult hospital course, numerous ischemia recurrences, and a higher risk of death [3]. In cases with hyperglycemia and DM, adverse outcomes include congestive heart failure, cardiac shock, ventricles arrhythmias, and death are more common after an episode of ACS [4].
Coronary atherosclerosis-related ACS is a serious, perhaps fatal cardiovascular disorder. Atherosclerotic plaque rupture, vasospasm, platelet aggregation, and subsequent thrombosis are its main underlying pathophysiological mechanisms.

These might result in significant coronary artery stenosis or blockage and acute myocardial ischemia or, worse, myocardial infarction. Diabetes cases frequently develop coronary artery lesions that are more widespread, diffuse, severe, and calcified [5].

Regardless of previous diabetes history, cases with acute myocardial infarction (AMI) may exhibit hyperglycemia at the time of admission. Stress hyperglycemia brought on by catecholamines is typically the culprit, and this condition is linked to the progression of myocardial lesions and an increase in mortality [6, 7].

Larger infarct size and a higher prevalence of heart failure and cardiac shock in that population may contribute to the increased mortality [8], Elevated glucose levels in some cases may simply be a sign of a condition that was present but undiagnosed, such as type 2 diabetes or glucose intolerance. These conditions can increase lipolysis, produce an excess of free fatty acids in the blood, worsen myocardial damage, and worsen coronary disease [9].

Both diabetics and non-diabetics with high admission plasma glucose (APG) have a poor prognosis and, consequently, a poor course of illness progression, and earlier studies have indicated that the serum glucose level at hospital entry may predict mortality in cases with AMI [11, 12]. Elevated admission blood glucose is an important marker of worse outcome in cases with myocardial infarction (MI) [10], and high admission plasma glucose (APG).

Knowing a patient's entrance blood glucose levels and prior diabetes diagnosis is critical information for optimal patient management because early aggressive treatment of hyperglycemia may improve both short- and long-term outcomes in these cases.

The aim of this work was addressing the associations between admission random blood glucose (RBG), fasting blood glucose (FBG), and their correlations, and severity of coronary artery disease “assessed by Gensini score “and in hospital outcomes in patient admitted with ST elevated Myocardial Infarction (STEMI).

Patients and Methods

This prospective observational research was carried out on one hundred cases presenting by STEMI and treated with primary PCI or pharmaco-invasive technique according to recent ESC guidelines. The cases were assigned according to RBG into two groups; group 1: Non hyperglycemia group (<200 mg/dl), group 2: hyperglycemia group (>200 mg/dl). Then after 8 hours from admission FBG was tested and the cases were divided into two subgroups; group I: Non-FBG elevated (<126 mg/dl), Group II: FBG elevated (>126 mg/dl). The research was recruited from Cardiology department in Tanta University, Egypt in a period of 12 months starting from December 2020.

The research was done after approval from the Ethical Committee Tanta University Hospitals and registration of clinicaltrials.gov. Informed written consent was obtained from the patient or their relatives.

Exclusion criteria were admission more than 24 hours after onset of symptoms, cases less 18 years old and cases with non-cardiovascular causes for the ACS such as trauma, surgery.

All cases were subjected to full history taking, routine laboratory investigations with measuring FBG after 8hours from admission, full clinical examination, resting 12 leads ECG and reperfusion either through: primary PCI for infarct related artery (IRA) or Pharmaco-invasive technique.

PCI

The arterial access: Regarding the femoral approach, the local anesthetic is introduced into an area 3 to 4 cm in diameter 3 to4 cm below the inguinal ligament. Radial approach was done in selected cases with normal Allen’s test and no previous history of abnormal anatomy, generally the choice of the arterial access was up to the operators to decide. The ideal position of entry is approximately 2 cm proximal to the radial styloid.

Imaging: Left coronary imaging: A contrast injection in the left coronary cusp is a reasonable first step to define the ostium of the left main (LM) coronary artery, an antero-posterior (AP) view or a shallow right anterior oblique (RAO) caudal view may be useful to evaluate middle and distal LM coronary artery stenosis. A shallow left anterior oblique (LAO) or LAO cranial view is usually best to visualize osteal LM stenosis. Adequate visualization of the left coronary system commonly requires five or more views: The LAO view, the RAO view, the AP cranial view, the AP caudal view, the spider view [13].

Right coronary imaging

The RCA should be approached in the 30-degree LAO projection. The Judkin Right 4 (JR4) is advanced to the aortic valve level. The LAO view is useful to evaluate the proximal and mid-RCA, the AP view with 30-degree cranial angulation is often the best for evaluating the RCA bifurcation, ostia of the PDA and posterolateral branches and a shallow RAO view is useful to show the entire PDA [14]. The infarcted related artery (IRA) was also identified. PCI with or without stenting was immediately performed with a 6-Fr guiding catheter. When necessary, thrombus aspiration, balloon pre- and post-dilatation, were carried out. The TIMI blood flow grade is used to assess the success of reperfusion: TIMI blood flow grade classified reperfusion as successful (TIMI 3) or abnormal (TIMI 0-1-2) [13].

Evaluation of coronary artery disease severity: Step 1 The coronary artery lesions' degree of stenosis was graded as follows: Step 2: Based on the position of the lesion in the coronary tree and the functional importance of the area that segment fed, the degree of stenosis score was then multiplied by the lesion site score. Step 3: The final Gensini score was determined by adding the lesion scores.

Pharmaco-invasive technique

In pharmaco-invasive technique, cases receive thrombolytic therapy followed by coronary angiography either immediately in case of failed thrombolytic or within 3-24 hours after sign of successful reperfusion [15]. The used type of thrombolytic is: Alteplase (tPA). The accepted time for starting the infusion: According to ESC guidelines 2017 IV bolus of thrombolytic therapy should start within 10 minutes, however thrombolytic therapy can be given within 12 hrs. from onset of chest pain [15].

Echocardiography

All research was conducted utilising (a GE vivid seven Cardiac ultrasound phased array system with tissue Doppler imaging using M4S transducer 4 M.HZ.) Upon admission following reperfusion, an M-mode and modified Simpson echocardiographic evaluation in two dimensions was performed. 2-D Echocardiography was performed in a posture of partial left lateral decubitus to: Evaluate the LV systolic function with either:

Simpson's method in the apical 4 & apical 2 views also we assessed Left ventricle volumes. (End diastolic volume EDV and end systolic volume ESV) or M-mode assessment of LV systolic function through getting the long parasternal axis view and directing the M-mode cursor across the LV & it is measured also in the parasternal short view with directing the M-mode cursor across the mid LV.

Analyze segmental wall motion irregularities SWMA and worldwide wall movement Utilizing a 17 segment model for LV segmentation, regional wall motion anomalies were evaluated. According to current guidelines, the severity of mitral regurgitation was categorised.

Statistical analysis

With the aid of the IBM SPSS software package version 20.0, data were input into the computer and analysed (Armonk, NY: IBM Corp). Number and percentage were used to describe qualitative data. The normality of the distribution was examined using the Kolmogorov-Smirnov test. The range (minimum and maximum), mean, standard deviation, and median were used to characterise quantitative data. The 5% level of significance was used to determine the results' significance [16].

Results

Patient RBG measurements were presented in (Table 1).

Table 1: Distribution of the studied cases according to random Blood Glucose

Random Blood Glucose	No (%)
Non hyperglycemia (<200)	72 (72.0%)
Hyperglycemia (>200)	28 (28.0%)
Mean±SD.	182.09±82.65

The data was presented as frequency and percentage.

The cases in FBG elevated group were significantly elder with diabetes history for longer period and complicated DM in addition to higher BMI and higher prevalence of CKD and previous cardiac surgeries than non-FBG group (Table 2).

Table 2: Comparison between Non-FBG elevated and FBG elevated according to demographic data, History of Diabetes, Prevalence of other risk factors, Clinical Presentation and Gensini Score

	Non-FBG elevated (<126) (n = 57)	FBG elevated (>126) (n = 43)	P
	No (%)	No (%)	
Sex			
Male	42 (73.7%)	32 (74.4%)	0.934
Female	15 (26.3%)	11 (25.6%)	
Age (years) Mean±SD.	52.88±10.53	59.70±8.16	<0.001*
Diabetes			
No DM	42 (73.7%)	19 (44.2%)	0.003*
Duration of DM (years)			
Mean±SD.	16.27±7.88	25.08±8.91	0.003*
Complicated DM	0 (0.0%)	17 (39.5%)	<0.001*
Hypertension	13 (22.8%)	20 (46.5%)	0.013*
Smoking	39 (68.4%)	21(48.8%)	0.048*
History of dyslipidaemia	9 (15.8%)	15 (34.9%)	0.027*
BMI (Kg/m^2) Mean±SD.	30.23±2.93	31.98±3.41	0.007*
Family History	7 (12.3%)	8(18.6%)	0.381
Previous stroke / TIA	0 (0.0%)	6 (14.0%)	^{FE}p=0.005*
CKD	0 (0.0%)	5 (11.6%)	^{FE}p=0.013*
Past Hx Of IHD	10 (17.5%)	15 (34.9%)	0.047*
Previous MI / ACS	4 (7.0%)	11 (25.6%)	0.010*
Previous PCI	4 (7.0%)	11 (25.6%)	0.010*
Patient Delay (Hours) Mean±SD.	3.74±1.77	6.09±7.18	0.013*
SBP (mmHg) Mean±SD.	133.51±21.09	129.07±24.77	0.336
DBP (mmHg) Mean±SD.	83.86±11.14	82.09±16.41	0.523
Pulse (bpm) Mean±SD.	79.53±16.91	95.70±20.35	<0.001*
Killip Class			
I	55 (96.5%)	25 (58.1%)	^{MC}p <0.001*
II	2 (3.5%)	10 (23.3%)	
III	0 (0.0%)	3 (7.0%)	
IV	0 (0.0%)	5 (11.6%)	
Mechanical complications	0 (0.0%)	0 (0.0%)	–
STEMI Location			
Anterior	23(41.8%)	21(50.0%)	0.587
Inferior	24(43.6%)	14(33.3%)	
Lateral	8(14.5%)	7(16.7%)	
Gensini Score			
Mean±SD.	55.28±18.59	95.88±33.79	<0.001*

The data was presented as mean±SD or frequency and percentage. DM: Diabetes Mellitus; BMI: Body mass index; CKD: Chronic Kidney Disease; ACS: acute coronary syndrome; SBP: systolic blood pressure; DBP: Diastolic Blood Pressureχ^2: Chi square test; t: Student t-test; MC: Monte Carlo; FE: Fisher Exact; p: p value for comparing between different category; *: Statistically significant at $p \leq 0.05$. There was statistically significant positive correlation between Gensini score and RBS and fasting RBS ($p<0.001$). Figure 1.

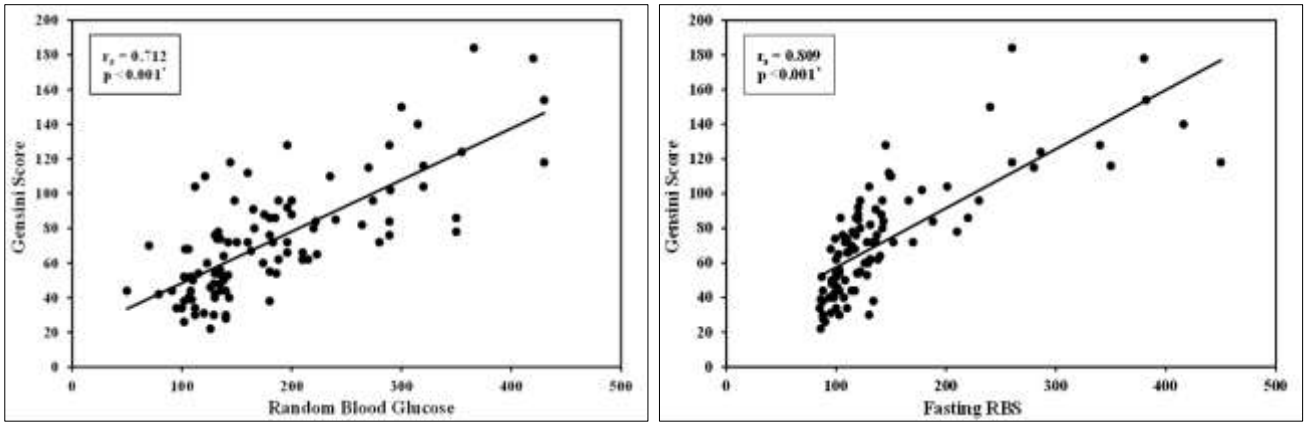

Fig 1: Correlation between Gensini Score with Random Blood Glucose and Fasting RBS

Logistic Regression was performed to evaluate the impact of ABG and FBG on Gensini Score and MACEs where Fasting blood glucose showed stronger association with both Vs Random blood glucose. Table 3.

Table 3: Univariate and multivariate liner regression analysis Random blood glucose and fasting blood glucose as regard their effect on Gensini Score and their correlation with MACE

	Univariate		Multivariate	
	P	**B (95% C.I)**	**P**	**B (95% C.I)**
Gensini Score				
Random Blood Glucose	<0.001*	0.297(0.244 – 0.351)	0.001	0.138(0.059 – 0.217)
Fasting RBS	<0.001*	0.341(0.286 – 0.396)	<0.001*	0.221(0.134 – 0.307)
Mace				
Random Blood Glucose	<0.001*	1.011(1.005 – 1.017)	0.003*	0.970(0.951 – 0.990)
Fasting RBS	<0.001*	1.031(1.016 – 1.045)	<0.001*	1.081(1.037 – 1.128)

χ2: Chi square test; U: Mann Whitney test t: Student t-test; B: Unstandardized Coefficients C.I: Confidence interval LL: Lower limit; UL: Upper Limit; p: p value for comparing between different category; *: Statistically significant at $p \leq 0.05$

There was statistically significant difference between the two groups (P value<0.001) where post-intervention TIMI Flow in hyperglycemic group was worse), There was statistically significant difference with more impairment of EF in group 2 (P value <0.001) according to Echocardiographic parameters (Table 4).

Table 4: Comparison between Non-FBG elevated and FBG elevated according to Management and procedural characteristics, some laboratory data and Echocardiographic parameters

	Non-FBG elevated (<126) (n = 57)	**FBG elevated (>126) (n = 43)**	**P**
	No (%)	**No (%)**	
Reperfusion Strategy			
Medical Treatment	0 (0.0%)	1 (2.3%)	^{MC}p=0.336
Pharmacoinvasive	5(8.8%)	6 (14.0%)	
Primary PCI	52 (91.2%)	36 (83.7%)	
Infarction Related Artery			
Left Main	7 (12.3%)	6 (14.0%)	0.676
LAD	19 (33.3%)	17 (39.5%)	
LCX	7 (12.3%)	7 (16.3%)	
RCA	24 (42.1%)	13 (30.2%)	
Type of Intervention			
None	2 (3.5%)	0 (0.0%)	^{MC}p=0.637
PTCA	2 (3.5%)	1 (2.3%)	
DES	53 (93.0%)	42 (97.7%)	
Surgery Referral	0 (0.0%)	0 (0.0%)	
Final TIMI Flow			
0	4 (7.0%)	12 (27.9%)	^{MC}p= 0.008*
I	4 (7.0%)	7 (16.3%)	
II	6 (10.5%)	4 (9.3%)	
III	43 (75.4%)	20 (46.5%)	
Hemoglobin (gm/dl) Mean±SD.	13.01±1.23	12.37±1.43	0.018*
HDL (mg/dl) Mean±SD.	39.61±7.95	35.95±6.48	0.016*
LDL (mg/dl) Mean±SD.	134.65±28.03	149.33±25.61	0.008*
Triglycerides (mg/dl) Mean±SD.	172.58±33.65	192.74±29.50	0.002*
Creatinine (mg/dl) Mean±SD.	1.17±0.21	1.42±0.43	0.004*
Ejection fraction (%) Mean±SD.	46.79±5.70	40.56±8.72	<0.001*

PCI: Percutaneous coronary intervention; LAD: Left anterior descending coronary artery; LCX; Left circumflex coronary artery; RCA: Right coronary artery; PTCA: Percutaneous transluminal coronary Angioplasty; DES: Drug-eluting stent; HDL: High density lipoprotein; LDL: Low density lipoprotein; χ^2: Chi square test; MC: Monte Carlo; FE: Fisher Exact; U: Mann Whitney test; p: p value for comparing between different category; *: Statistically significant at $p \leq 0.05$

There was statistically significant difference with more events in group II (P value <0.001) & (P value =0.005) respectively (Table 5).

Table 5: Comparison between Non-FBG elevated and FBG elevated according to In-hospital clinical outcomes

	Non-FBG elevated (<126) (n = 57)	FBG elevated (>126) (n = 43)	P
	No (%)	No (%)	
MACE	2 (3.5%)	19 (44.2%)	<0.001*
In Hospital Death	0 (0.0%)	5 (11.6%)	FEp=0.013*
In-Hospital Reinfarction	1 (1.8%)	6 (14.0%)	FEp=0.040*
In-Hospital CHF	1 (1.8%)	12 (27.9%)	<0.001*
Cardiogenic shock	0 (0.0%)	7 (16.3%)	FEp=0.002*
In-Hospital Bleeding Complications	3 (5.3%)	3 (7.0%)	FEp=1.000
Bleeding Requiring blood transfusion	0 (0.0%)	3 (7.0%)	FEp=1.000
Arrythmia (New Onset)			
None	55 (96.5%)	33 (76.7%)	MCp= 0.001*
Atrial fibrillation	2 (3.5%)	1 (2.3%)	
Ventricular Tachycardia	0 (0.0%)	5 (11.6%)	
Ventricular Fibrillation	0 (0.0%)	2 (4.7%)	
High Grade AV Block	0 (0.0%)	2 (4.7%)	
CIN	0 (0.0%)	12(27.9%)	<0.001*
New Requirement for Dialysis	0 (0.0%)	6(14.0%)	FEp=0.005*
Stroke	0 (0.0%)	1(2.3%)	FEp=0.430

MACE: Major adverse cardiac events; CHF: Congestive heart failure; AV: Atrioventricular; χ^2: Chi square test; MC: Monte Carlo; FE: Fisher Exact; p: p value for comparing between different category; CIN: Contrast induced nephropathy; *: Statistically significant at $p \leq 0.05$

Discussion

Diabetes is a standalone risk factor for coronary artery disease (CAD), which is the leading cause of mortality in diabetics. Diabetes is linked to a 2-3-fold increase in the risk of cardiovascular disease (CVD) and mortality from CVD when compared to the non-diabetic population [17].

Acute glycometabolic disturbances powerfully impact outcome in AMI cases as In cases with hyperglycemia and DM, adverse outcomes include congestive heart failure, cardiogenic shock, ventricular arrhythmias, and death are more common after an episode of acute coronary syndrome ACS [4]. High admission plasma glucose (APG) levels are indicators of a bad prognosis and, consequently, of a poor course of disease progression in both diabetics and non-diabetics with ACS [12, 18].

In our research, we assessed the impact of RBG and FBG on the severity of CAD and looked at the potential associations between admission RBG, FBG, and Gensini score and in-hospital clinical outcomes in STEMI cases.

Comparison of the baseline data showed the presence of significant difference where the hyperglycemic and FBG elevated group showed a higher incidence of hypertension, diabetes, previous MI, cerebral infarction, CKD, dyslipidemia, and smoking.

This came in accordance with the work by J.R. Timmer *et al*. [19] and multiple studies for example recent work by Ana T Timóteo *et al*. [20] which showed the higher incidence of acute glycometabolic alteration in the setting of ACS between cases already with several risk factors for developing metabolic syndromes.

In our research cases in both hyperglycemic and FBG elevated groups presented later with mean duration from onset of symptoms to FMC.

This has come with the fact that diabetic cases - who represented the majority of the hyperglycemic and FBG elevated groups - frequently present with silent myocardial ischemia (SMI), and the absence of an imperative clinical "warning symptom" [21, 22]. The presence of diabetes as a recurrent independent predictors of non-pain symptoms in different AMI registries e.g. (NRMI 2) [23] and Arenja *et al*. [24] and Culić *et al*. [25] studies.

More STEMI cases in our study presenting with adverse features belonged to hyperglycemia and FBG elevated groups as 14.3% of the hyperglycemic group presented with Killip IV class only to be accounted for 1.4 of non-hyperglycemic group (P value <0.001) the same was noticed in FBG groups (11.6% Vs 0.0% (P value <0.001)].

These are not merely incidental findings but an important predictor of our primary outcomes as hyperglycemic STEMI cases presenting late with adverse features not receiving timely interventions had worse prognosis as we will find later.

In our study both the hyperglycemic [Mean Gensini Score 102.25±34.71 Vs 61.26±24.12 in non-hyperglycemic group P value < 0.001] and FBG elevated Group [Mean Gensini Score 95.88±33.79 Vs 55.28±18.59 in non- FBG elevated group P value < 0.001] showed more severe, extensive, and diffuse coronary artery lesions.This came in agreement with multiple trials that studied hyperglycemia in ACS as elevated RBS on admission was associated with the higher incidence of multivessel disease in work by Ana T Timóteoe al. [20] also, the work of Peng Wei *et al*. [26] which classified coronary lesions as severe, moderate and slight showed that cases with severe coronary lesion had higher concentration of blood glucose than those in other ACS subgroups or control group (p<0.001). This correlation also wasn't exclusive to diabetic individuals as non-diabetics with hyperglycemia on admissions suffered from more severe CAD in accordance with the findings of N. Fikal *et al*. [24] which showed that in non-diabetic acute coronary syndrome cases, hyperglycemia on admission was predictive of CAD severity assessed by SYNTAX score advocating for the idea of the "glycemic continuum" throughout the range of CVD risk, prediabetes, and Early dysglycemia, a state that is frequently seen in individuals with reduced glucose tolerance, is crucial in initiating the pathological processes that lead to atherosclerotic vascular problems [27].

In our study, MACEs occurred more frequently in cases with impaired glucose metabolism where hyperglycemic

group had (39.3%) Vs (13.9) in non-hyperglycemic group P value =0.005. Also, all in-hospital death in our study occurred in the hyperglycemic group accounting for 17.9% P value < 0.001 confirming the concept a prior study found that for every gramme per litre increase in glucose level, there was a 1.7-fold increase in the probability of death during follow-up in cases with acute infarction, indicating that RBG on admission is directly connected to mortality in AMI cases [28].

The findings of our investigation are given here to support the predictive significance of DM and the plasma glucose status of individuals with an ACS [7, 8]. Although it has also been stated that the presence of hyperglycemia at admission is just as good as or even better than the existence of diabetes as a predictor of death and/or reinfarction [29]. These findings were reproduced in e IABP-SHOCK II study [30] showed that, in AMI cases with cardiogenic shock, RBG [hyperglycemia as ≥11.5 mmol/L] was an independent predictor of 30-day and 1-year mortality after MI (47.7% vs. 36.5% in non-hyperglycemic P value = 0.004.

Otten *et al*. [31] showed that fasting glycemia was an independent predictor of adverse events in cases with ACS. According to Suleiman *et al*. [32], in non-diabetic cases who had experienced an acute myocardial infarction, a substantial correlation between high FPG and APG readings and mortality at 30 days was reported.

It's still not entirely apparent how glycemia is connected to greater mortality. It appears that during ACS, the body's reaction to stress raises the levels of the hormones glucagon, cortisol, and catecholamines with its metabolic consequences leading to increased myocardial damage this came in results of our study which showed that both hyperglycemic and FBG elevated groups had a more infarct size and a more impaired systolic function that agree with the work of Ignacio Cruz-Gonzalez *et al*. [33] which showed that the infarct size measured by SPECT imaging at Day 5 post-MI was larger in the hyperglycemia group.

Also, the hyperglycemic effect on coagulation system plays powerful role, In our study as worse TIMI flow post intervention was noticed more in hyperglycemic group [Final TIMI 3 flow was achieved in 14.3% of cases with hyperglycemia vs 81.9% of cases with non-hyperglycemia (p<0.001)] and FBG elevated group [Final TIMI 3 flow was achieved in 46.4% of cases with FBG elevated vs 75.4% of cases with FBG non-elevated (p<0.001)]. These results in accordance with work by Surya Dharma *et al*. [34] which showed that a higher percentage of cases with hyperglycemia (169 mg/dL) at presentation had final TIMI flows of 0 to 1. Additionally, a prior study found a link between hyperglycemia and a poorer baseline TIMI flow before to primary PCI [35], indicating that hyperglycemia is a significant factor in determining the coronary perfusion prior to and following primary PCI and consequently infarct size and clinical outcomes. This finding is reaffirmed in our finding that in-hospital reinfarction occurred more frequently in hyperglycemic [(17.9%) Vs (2.85) P value =0.017] and FBG elevated group [(14.0%) Vs (1.8%) P value = 0.040]. Our study showed that other surrogates of in-hospital mortality occurred more in the hyperglycemic and FBG elevated group, a recurrent finding in studies of hyperglycemia in ASC. Cases in Hyperglycemic and FBG elevated groups suffered more from cardiogenic shock and congestive heart failure a finding that the latest CardShock Study [36] has found in both ACS and non-ACS forms of cardiogenic shock which proved that Cases with severe hyperglycemia had the greatest in-hospital mortality rates (56%) compared to those with normoglycemia (22%). (P value 0.01) identifying severe hyperglycemia as a unique predictor of in-hospital death.

Limitations: First of all, this was a small-sample single-center study. The associations between RBG, FBG, and Gensini score need to be further examined in a multicenter study with a larger sample size in order to establish their predictive significance for both short-term and long-term unfavourable prognoses. Second, we omitted the precise HbA1c value from our report. Thirdly, the study lacks information on the care given to cases with hyperglycemia while they were hospitalised. Another issue is that during the hospital stay, the glucose level was not routinely checked. Last but not least, this study did not make it clear whether treatments for hyperglycemia can undo its negative effects.

Conclusions

In this study, we highlighted the value of both RBS at admission and FBS in correlation to both severity of CAD and in-hospital outcomes, so in daily clinical practice with this simple method, available in any hospital, it is possible to identify at-risk individuals at admission, allowing for better stratification and channelling the best care for these high-risk cases.

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