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Impact of Admission Hyperglycemia on Postmyocardial Infarction Left Ventricular Remodelling

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ABSTRACT

Objectives*: This study aims to assess left ventricular (LV) remodelling in acute myocardial infarction (AMI) patients presented with admission hyperglycemia (AH) and to determine the association between AH and LV remodelling.*

Patients and Methods: *We conducted a prospective cohort study and we analysed LV remodelling in 122 AMI patients. AH was defined as a glycemia on admission ≥ 140 mg/dl. We divided the patients according to the admission plasma glucose and history of diabetes into three groups: 45 euglycemic patients, 36 diabetic patients and 41 hyperglycemic non diabetic patients. Systematic echocardiographic study was performed at baseline and at 12 months later.*

Results*: The changes in LV end-diastolic volume (LVEDV) from baseline to 12 months follow up was 4.86 ± 13.20 cm3 in euglycemic group, 86.47 ± 65.53cm3 in diabetics and 149.40 ±*

59.55cm3 in hyperglycemic nondiabetic patients (P=0.000 for all). When LV remodelling was defined as a ≥20% increase in LVEDV, it was observed in 17.1% in euglycemic patients, 91.7% in diabetics, and 100% in hyperglycemic nondiabetic patients (P=0.000 between euglycemic patients and both other groups, P=0.248 between diabetics and hyperglycemic nondiabetic patients).By multiple linear regression analysis, AH (P = 0.000), baseline segmental wall motion score (P =0.000), and partial reperfusion (P= 0.035) were independently associated with LV remodelling. By multiple logistic regression analysis, AH was an independent predictor of LV remodelling with adjusted Odd's ratio=2.167(95%CI:2.001 2.333).

Conclusions: *We concluded that patients with admission hyperglycemia had a worse postmyocardial infarction LV remodelling than euglycemic patients, regardless of their diabetic status. Admission hyperglycemia was a major and independent predictor of postmyocardial LV remodelling.*

Keywords*: admission hyperglycemia, postmyocardial infarction remodelling.*

INTRODUCTION

Patients either with or without a prior history of diabetes mellitus may present with hyperglycemia during acute myocardial infarction (AMI). Among patients with no prior history of diabetes, hyperglycemia during AMI may reflect previously undiagnosed diabetes, preexisting carbohydrate intolerance, stress-related hyperglycemia, or a combination of these[1]. Several studies have reported an association between elevated plasma glucose upon admission and subsequent increased adverse events, including congestive heart failure (CHF), cardiogenic shock, and death, but the mechanisms underlying this association are unknown [2].Among the pathophysiological mechanisms that may lead to an adverse prognosis after AMI is left ventricular (LV) remodelling. LV remodelling is a dynamic process that occurs in response to damage to the myocardium after AMI [3].Progressive LV dilation after AMI has been recognized as a strong predictor of heart failure and cardiovascular death [4].Several factors have been shown to influence LV remodelling; these include infarct size, anterior infarct location, or patency of the infarct-related artery [5].

Nian et al.[6]described an active inflammatory infiltrate in the peri-infarct area and in unaffected viable myocardium in patients with AMI.The inflammatory burden in the peri-infarct region is associated with worse short- and mid-term outcomes. This is because the inflammatory response in this region probably amplifies myocardial remodelling. Cytokines such as tumor necrosis factor α are elaborated soon after myocardial ischemic injury and can acutely regulate myocyte survival or

apoptosis, leading to acute cardiac remodelling, paving the way for heart failure. In this context, Marfella et al [7]have shown that hyperglycemic stress during AMI is associated with increased levels of some inflammatory markers, including C-reactive protein and interleukin-18.These results fit with animal studies showing increased levels of oxidative stress indices and proinflammatory cytokines in the ischemic heart tissue of hyperglycemic mice. Both inflammation and oxidative stress correlated strictly with the glucose levels, leading to myocardial apoptosis and greater infarct size. These studies suggest that hyperglycemia amplifying oxidative stress and the inflammatory responses to myocardial ischemia might affect the prognosis of patients presenting with AMI[7].

We hypothesized that patients with AMI presented with admission hyperglycemia may be at greater risk for LV remodelling, which can explain the increase in the short and long term complications in those patients. To test this hypothesis, we conduct this study to assess LV remodeling in patients with AMI presented with admission hyperglycemia by systematic echocardiographic follow-up study and to determine the association between admission hyperglycemia and LV remodelling

PATIENTS AND METHODS

We conducted a prospective cohort study. The study included 122 patients admitted to internal medicine CCU, between January 2010 and March 2014, who presented by the first AMI within 12 hours from symptom onset, treated by thrombolytic agents with at least 50% resolution of ST segment and had infarct zone comprised at least three hypokinetic LV segments by echocardiography.The diagnosis of AMI was based on prolonged chest pain lasting ≥ 30 min, ST segment elevation ≥ 2 mm in at least two contiguous electrocardiographic (ECG) leads of v1 to v3 or ≥ 1 mm in at least two contiguous other leads, and a more than threefold increase in serum creatine kinase (CK) levels. We excluded patients with inadequate echocardiographic image quality, age ≥ 85years, life-limiting non-cardiac disease, significant valvular disease, prior Q-wave MI, presence of left bundle branch block. The study was approved by the ethical committee of college of medicine of Assiut University and written informed consents were obtained from all patients. All patients managed according to 2007 Focused Update of the ACC/AHA 2004 Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction[8].

We divided the patients according to the random plasma glucose level at the admission using the study cutoff value of ≥ 140 mg/dl and the history of diabetes mellitus into three groups: euglycemic patients, hyperglycemic nondiabetic patients and diabetic patients. Diabetes mellitus was considered to be present if patients had a history of diabetes that was managed with diet, or with oral hypoglycemic agents or insulin, regardless of duration. Admission hyperglycemia was defined as

the presence of random plasma glucose ≥ 140 mg/dl (7.7 mmol/l) in nondiabetic patients during the first day of hospitalization, whereas patients with plasma glucose levels <140 mg/dl have been classified as euglycemic patients.

All echocardiograms were performed with an HDI 5000 instrument (Philips Medical Systems, Bothell, Washington, USA) equipped with a broad band harmonic transducer. A standard imaging protocol was used wihin the 3±2 days of admission (i.e.baseline echocardiographic study). All echocardiograms were analyzed at the Assiut University Internal Medicine Echo Laboratory. LV volumes and ejection fraction (EF) were calculated using a modified Simpson's rule. The mean value of three measurements of the technically best cardiac cycles was taken from each examination.To evaluate regional systolic function, the left ventricle was divided according to a 16-segment model as recommended by the American Society of Echocardiography. For each segment, wall motion has been scored from 1 (normal) to 4 (dyskinetic) and a global segmental wall motion score (SWMS) was calculated as the average over 16 segments[9]. Aiming at identifying the actual infarction size with no contractile reserve and excluding the stunned myocardium, low dose dobutamine stress echocardiography (LDSE) was performed to calculate the best SWMS[10]. Twelve months later, a follow up echocardiographic study was performed and dyssynergic segments were classified as recovered, unchanged, or remodeled according to changes in wall motion compared with the baseline echocardiographic study. Recovered wall motion was defined as a decrease in wall motion score of at least one grade. Remodeled wall motion was defined as an increase in wall motion score of at least one grade or a persistent wall motion score of 4(dyskinesis). Unchanged wall motion was defined as a persistent wall motion score of 1–3 with no change. Global LV dilatation was assessed as the change in LV end-diastolic volume (Delta LVEDV) from the baseline level and significant global LV remodelling was defined as a Delta LVEDV≥20% increase from the baseline level [11].

STATISTICAL ANALYSIS

All calculations were performed with the computer program SPSS version 16.Continuous variables were described as mean ± standard; frequencies were expressed as percentages. Continuous variables were compared among the groups of patients with ANOVA test. The Kolmogorov-Smirnov test was used to assess whether continuous variables were normally distributed or not. Linearity assumption for continuous variables was assessed using the graphic method. The Hosmer–Lemeshow goodness-of-fit test was used to check that the model adequately fit the data. A value of $P < 0.05$ was considered statistically significant.

To investigate correlation between glycemic levels and follow up LVEDV and occurrence of LV remodelling, we used pearson's correlation and spearmen's correlation respectively. To

investigate correlation between glycemic levels and follow up LVEDV and occurrence of LV remodelling with isolation of other factors (as SWMS, sum of ST, number of involved leads, peak CK), we used partial correlation. To investigate the independent contribution of glycemic levels to the echocardiographic parameters, a multiple linear regression analysis was performed. The variables entered in the multiple linear regression analysis were SWMS, EF, age and partial reperfusion. Odd's ratio adjusted for SWMS and EF was computed from a multivariable logistic regression model.

RESULTS

The study included 122 patients; classified into 3 groups according to their admission random plasma glucose (PG) levels (with hyperglycemic cutoff value of ≥ 140 mg/dl), and history of diabetes mellitus (DM) as follows: euglycemic group including 45 patients, diabetic group including 36 patients and hyperglycemic nondiabetic group including 41 patients. Patients'characteristics of each group was shown in table 1.The study showed that there were insignificant differences between groups as regard age and gender.The study showed that the diabetic group had a significantly higher percentage of patients with history of hypertension than euglycemic group and hyperglycemic nondiabetic group (p values = 0.000 between all groups), but there were insignificant differences between groups as regard history of previous angina, onset of angina, history of anti-dyslipidemic drugs and current smokers.

The study showed that the diabetic group, followed by hyperglycemic nondiabetic group, had a significantly higher BMI than euglycemic group (p-value = 0.000, p-value = 0.003respectively), but there was insignificant difference between diabetic group and hyperglycemic nondiabetic group as regard BMI.Also the study showed that the diabetic group, followed by hyperglycemic non diabetic group, had a significantly higher heart rate than euglycemic group (p-value = 0.030, p-value = 0.024 respectively), but there was insignificant difference between diabetic and hyperglycemic non diabetic group as regard heart rate.There were insignificant differences between groups as regard both systolic and diastolic blood pressure.The study showed that the hyperglycemic nondiabetic group had a significantly higher percentage of patients with Killip class III (i.e. decompensation) than the diabetic group while euglycemic group had not any patient(p-values =0.027, 0.000, and 0.017respectively).

The study showed that the hyperglycemic nondiabetic group had the significantly largest infarction size, followed by the diabetic group, then the euglycemic group.This result was concluded by the evidence that hyperglycemic nondiabetic group had the significantly highest sum ST, the

highest number of involved ECG leads, the highest best baseline SWMS by low-dose dobutamine stress echocardiography and the highest peak CK, followed by the diabetic group, then the euglycemic group (p-values = 0.000 between all groups). Also, the hyperglycemic nondiabetic group had a significantly lower complete ST resolution than the euglycemic group (p-value = 0.026), but there were insignificant differences in reperfusion between euglycemic group and diabetic group and between diabetic group and hyperglycemic nondiabetic group. In this study, both hyperglycemic group and diabetic group presented with hyperglycemia (table 1).

The study found that the hyperglycemic nondiabetic group, followed by the diabetic group, had a significantly higher baseline LV volumes, lower baseline EF and higher baseline SWMS than the euglycemic group. The follow up echocardiographic study demonstrated that hyperglycemic nondiabetic group has the significantly worst changes in all follow up echoparameters (follow up LVEDV, change in LVEDV (Delta LVEDV), follow up LVESV, Delta LVESV, follow up EF, Delta EF, follow up SWMS, Delta SWMS and ≥ 20% change in LVEDV). When LV remodelling was defined as ≥ 20% increase in LVEDV, the study showed that the incidence of LV remodelling in euglycemic patients was 17.1%, 91.7% in diabetics while in hyperglycemic patients was 100% (P=0.000 between euglycemic and both other group, P=0.248 between diabetic and hyperglycemic nondiabetic group) (table 2,figures 1-2).

In the hyperglycemic nondiabetic group, and also in the diabetic group, the study showed that there was a significant positive correlation between the admission PG and follow-up LVEDV and significant positive correlation between the admission PG and Delta LVEDV, but insignificant correlation between admission PG and baseline LVEDV.In euglycemic group, the study showed that there was a significant positive correlation between the admission PG and Delta LVEDV, but insignificant correlation was found between the admission PG and both baseline LVEDV & follow-up LVEDV (table 3).

When we conducted a partial correlation between admission PG and Delta LVEDV with controlling infarction size parameters that affect remodelling (SWMS, Peak CK, Sum ST, number of involved leads), the study showed that there was a significant positive correlation between the admission PG and Delta LVEDV(r=0.680 and p-value=0.000) (table 4). We concluded that admission hyperglycemia had an isolated and independent effect on postmyocardial infarction LV remodelling that cannot be explained by the admission infarction size only.

By multiple linear regression analysis between Delta LVEDV (as a dependent variable) and admission PG, baseline SWMS, baseline EF, age and partial resolution (all as independent variables), there were significant correlations between Delta LVEDV and admission PG (β

coefficient= 0.696, p-value = 0.000), baseline SWMS(β *co* = 0.335, p-value = 0.000) ,and partial reperfusion(β *co* = 0.060, p-value = 0.035) respectively (table 5). We concluded that admission hyperglycemia, infarction size and partial reperfusion were independent predictors of postmyocardial infarction LV remodelling.

By multivariable logistic regression analysis between the occurrence of LV remodelling (as a binary dependent variable) and admission PG, baseline SWMS, and baseline EF (all as independent variables), we concluded that admission hyperglycemia with Odd's ratio (OR) = 2.167 (95%CI:2.001-2.333) (p-value = 0.049) and baseline SWMS with OR = 8.276 (95%CI:2.588-13.964)(p-value = 0.043) were major and independent predictors of LV remodelling after MI (table 6).

To illustrate the potential interest of combining SWMS with admission hyperglycemia to provide an estimate of the risk of LV remodelling; we calculated the incidence of LV remodelling in patients who had a SWMS ≥ 1.6 (the mean value in our study) and admission PG ≥ 140 mg/dl (the cutoff value in our study) and we found that the incidence was 100% in these patients so we can conclude that the risk of having a LV remodelling with SWMS ≥ 1.6 and admission PG ≥ 140 mg/dl is 100 %.

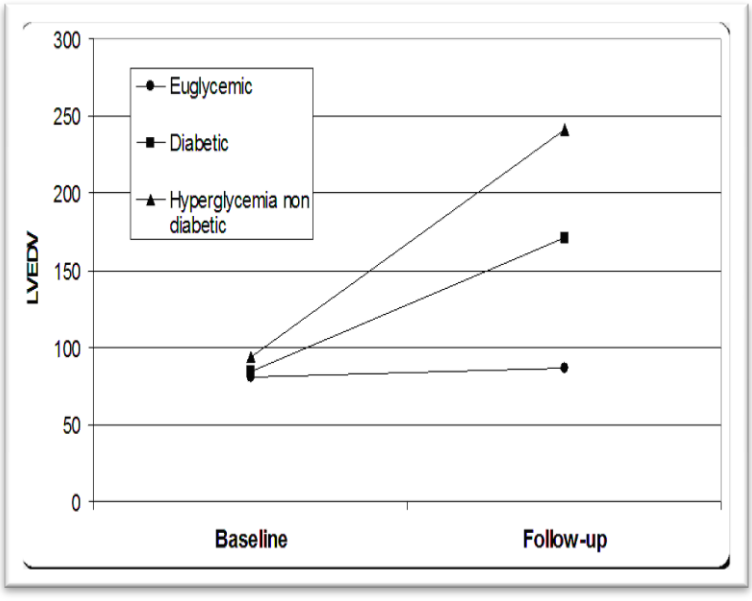

Figure 1 : Changes in LVEDV at baseline and follow up of different studied groups

Table 1: Patients' characteristics of different group

	Euglycemic	Diabetics	Hyperglycemic	P value
Age (years)	58.89 ± 6.97	56.81 ± 5.86	59.27 ± 7.84	0.259
Sex(male %)	80	69.4	82.9	*0.331*
Hx HTN (%)	15.6	41.7	2.4	*0.000*•
Dyslipidemia (%)	0	2.8	0	*0.300*
Smokers (%)	48.9	66.7	51.2	*0.235*
Previous angina (n)	17.8	5.6	9.8	*0.210*
Onset of angina (h)	4.11 ± 1.75	4.61 ± 1.50	4.81 ± 1.87	*0.160*
BMI (kg/m²)	27.29 ± 3.75	31.04 ± 4.42	30.15 ± 4.98	*0.000*,0.003$^{\psi}$,.413$^{\phi}$*
HR (b/m)	86.16 ± 14.71	93.03 ± 12.80	92.95 ± 12.37	*0.030, 0.024, 0.979*
Systolic BP(mmHg)	118.22 ± 14.35	124.17 ± 22.47	113.90 ± 23.12	*0.152, 0.296, 0.053*
Diastolic BP (mmHg)	75.78 ± 9.88	78.33 ± 11.83	72.44 ± 15.62	*0.293, 0.235, 0.069*
Killip class III (%)	0	8.3	19.5	*0.027, 0.000, 0.017*
Admission PG (mg/dl)	113.59 ± 13.48	253.17 ± 49.96	264.99 ± 64.35	*0.000, 0.000, 0.376*
LDL (mg/dl)	121.76 ± 14.22	124.72 ± 16.79	126.88 ± 19.54	*0.087, 0.053, 0.608*
Peak CK (u/l)	3254.56 ± 1416.48	5819.83 ± 1809.11	7703.49 ± 1467.66	*0.000, 0.000, 0.000*
S cr (umol/l)	97.13 ± 23.42	104.66 ± 16.87	112.01 ± 45.69	*0.109, 0.057, 0.364*
Sum ST (mv)	16.16 ± 6.12	27.36 ± 5.93	31.05 ± 2.85	*0.000, 0.000, 0.001*
No of leads (n)	4.11 ± 0.88	5.25 ± 1.79	6.78 ± 1.04	*0.000, 0.000, 0.000*
> 70% STR (%)	38 (84.4%)	28 (77.8%)	26 (63.4%)	*0.443, 0.026, 0.169*

•P value between all groups, * P value between euglycemic and diabetics, $^{\psi}$ P value between euglycemic and hyperglycemic, $^{\phi}$ P value between diabetic and hyperglycemic, BMI; body mass index, Hx HTN; history of hypertension, HR; heart rate, PG; plasma glucose, s cr; serum creatinine, Sum ST; summation of ST segments, No of leads; numbers of involved leads, STR; ST segment resolution.

Table 2: Comparison of echocardiographic data between different groups

	Euglycemic	Diabetics	Hyperglycemic	P¹	P²	P³
LVEDV(cm³)						
Baseline	80.62 ± 21.95	84.78 ± 25.72	93.95 ± 28.63	*0.435,*	*0.017,*	*0.146*
Follow up	86.47 ± 26.62	171.25 ± 75.07	241.63 ± 79.76	*0.000,*	*0.000,*	*0.000*
Changes(Delta)	4.86 ± 13.20	86.47 ± 65.53	149.40 ± 59.55	*0.000,*	*0.000,*	*0.000*
≥ 20%	6 (17.1%)	33 (91.7%)	35 (100.0%)	*0.000,*	*0.000,*	*0.248*
LVESV(cm³)						
Baseline	41.36 ± 15.02	46.49 ± 18.27	57.46 ± 22.64	*0.169,*	*0.000,*	*0.023*
Follow up	42.93 ± 18.43	121.38 ± 63.51	189.63 ± 77.99	*0.000,*	*0.000,*	*0.000*
Changes(Delta)	0.86 ± 9.04	74.89 ± 53.03	135.31 ± 62.31	*0.000,*	*0.000,*	*0.000*
EF(%)						
Baseline	47.89 ± 9.13	44.27 ± 6.69	39.02 ± 9.81	*0.050,*	*0.000,*	*0.008*
Follow up	49.46 ± 11.19	28.91 ± 10.73	22.82 ± 9.92	*0.000,*	*0.000,*	*0.001*
Changes(Delta)	-2.26 ± 5.81	15.35 ± 9.00	18.39 ± 9.77	*0.000,*	*0.000,*	*0.178*
SWMS						
The best baseline	1.45 ± 0.14	1.64 ± 0.18	1.81 ± 0.15	*0.000,*	*0.000,*	*0.000*
Follow up	1.43 ± 0.26	2.09 ± 0.11	2.09 ± 0.18	*0.000,*	*0.000,*	*0.929*
Changes(Delta)	-0.01 ± 0.23	0.45 ± 0.20	0.28 ± 0.22	*0.000,*	*0.000,*	*0.000*

P¹ value between euglycemic and diabetics, P² value between euglycemic and hyperglycemic, P³ value between diabetic and hyperglycemic.

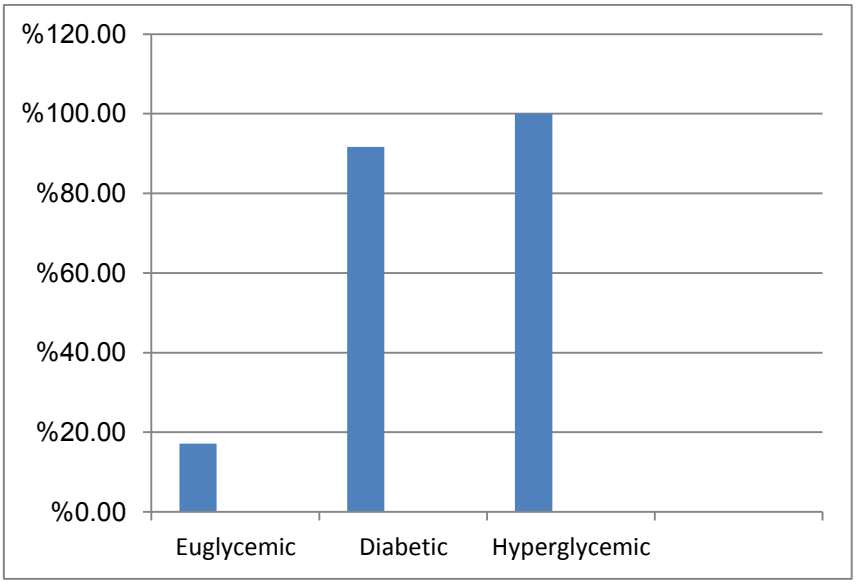

Figure 2 : Incidence rate of LV remodeling in different studied groups

Table 3: Correlation between admission PG and (baseline LVEDV, follow-up LVEDV &Delta LVEDV)

Groups		Admission plasma glucose	
		r-value	P-value
Euglycemic	Baseline LVEDV	0.648	0.061
	Follow-up LVEDV	0.321	0.060
	Delta LVEDV	0.848	0.000
Diabetic	Baseline LVEDV	0.564	0.059
	Follow-up LVEDV	0.735	0.000
	Delta LVEDV	0.868	0.000
Hyperglycemicnon diabetic	Baseline LVEDV	0.487	0.578
	Follow-up LVEDV	0.323	0.047
	Delta LVEDV	0.547	0.001

Table 4: Partial correlation between admission PG and baseline LVEDV, follow-up LVEDV & Delta LVEDV after control of other parameters of infarction size (baseline SWMS, peak CK, sum ST, number of involved leads).

	Admission plasma glucose	
	r-value	**P-value**
Baseline LVEDV	0.311	0.351
Follow-up LVEDV	0.412	0.260
Delta LVEDV	0.680	0.000

Table 5: Multivariate linear regression analysis

	Beta	***Sig.***	**95% Confidence Interval for *B***	
			Lower Bound	Upper Bound
Admission PG	0.696	0.000	0.737	0.941
Baseline SWMS	0.335	0.000	93.239	165.483
Baseline EF	0.021	0.526	-0.438	0.850
Age	-0.004	0.875	-0.602	0.514
Partial reperfusion	0.060	0.035	0.045	1.214

Table 6: Multivariable logistic regression analysis

	Sig.	Exp(B)	95% CI for EXP(B)	
			Lower Bound	Upper Bound
Admission PG	0.049	2.167	2.001	2.333
Baseline SWMS	0.043	8.276	2.588	13.964
Baseline EF	0.217	1.311	0.853	2.017

DISCUSSION

In patients with AMI, several studies have reported an association between elevated blood glucose upon admission and subsequent increased adverse events, including CHF, cardiogenic shock, and death [2].Although there is a large consensus regarding the prognostic impact of admission hyperglycemia on AMI patients, the exact mechanisms underlying this association remain poorly understood. Among the pathophysiological mechanisms that may lead to an adverse prognosis after AMI is LV remodelling. Several factors have been shown to influence LV remodelling; these include infarct size, anterior infarct location, or patency of the infarct-related artery [5]. Our study showed that the hyperglycemic nondiabetic group had the significantly largest infarction size, followed by the diabetic group, then the euglycemic group.This result agreed with several studies which found that admission plasma glucose was associated with larger infarct size and worse LV function [12].

The study found that the hyperglycemic nondiabetic group, followed by the diabetic group, had a significantly higher baseline LV volumes, lower baseline EF and higher baseline SWMS than the euglycemic group. These volumetric differences in the favor of hyperglycemic nondiabetic patients may be explained by the rapid onset of early LV remodelling by infarction expansion during the hospital stay and can be responsible of the acute complications that was noticed by numerous studies as mentioned by Capes et al.[1] meta-analysis in stress hyperglycemia in AMI. Early ventricular dilatation due to infarct expansion has been unequivocally demonstrated in man.Infarct expansion results from the degradation of the intermyocyte collagen struts by serine proteases and the activation of matrix metalloproteinases released from neutrophils [13].Infarct expansion occurs within hours of myocyte injury results in wall thinning and ventricular dilatation [14].

Although parameters measured at hospital admission or discharge are helpful to predict mid- and long-term clinical outcome after MI, they may not be the most accurate prognostic indicators. Indeed, follow-up studies have documented progressive changes in the LV chamber size,

shape, muscle mass, and function during the early months or years following MI (i.e.LV remodelling)[3].Progressive LV dilation after AMI has been recognized as a strong predictor of heart failure and cardiovascular death [4].We therefore designed the present study to analyse the impact of admission hyperglycemia not only on predischarge LV function but also on LV remodelling assessed by echocardiography 1-year later.

The follow up echocardiographic study demonstrated that hyperglycemic nondiabetic group has the significantly worst changes in all follow up echoparameters (follow up LVEDV, change in LVEDV (Delta LVEDV), follow up LVESV, Delta LVESV, follow up EF, Delta EF, follow up SWMS, Delta SWMS and ≥ 20% change in LVEDV).These results were consistent with several animal trials [15]and clinical trials, using either echo [16,17]or SPECT[18] ,which demonstrated that admission hyperglycemia exaggerate LV remodelling.

Djordjevic-Radojkovic et al. [17] enrolled 275 patients who were admitted with first STEMI and reperfused. Patients were divided according to admission glycemia into three groups: (1) with diabetes mellitus (DM); (2) with stress hyperglycemia (SH), without DM and; (3) without both DM and SH. SH was defined as admission blood glucose level ≥ 8 mmol/l (144 mg/dl). LVDV changed in patients with SH without DM from 126 ± 37 to 145 ± 30 ml after one year,($P < 0.05$).They concluded that SH could be marker of LV remodelling (significant increase of LVDV during one year).

We considered ≥ 20% increase in the LVEDV is a significant change and considered as LV remodelling [11].The incidence of LV remodelling in euglycemic patients was17.1%, 91.7% in diabetics while in hyperglycemic nondiabetic patients was 100%. This difference was statistically significant only between euglycemic patients and both diabetic and hyperglycemic patients. We should emphasize that both diabetic and hyperglycemic nondiabetic groups presented with hyperglycemia. Also,we should emphasize that although hyperglycemic nondiabetic patients had the worst in-hospital infarction size (ECG, enzymatic and echo) and the worst follow up echo parameters than patients with DM, the incidence of LV remodelling (by ≥20%) was statistically insignificant between these two groups, so we can conclude that patients with admission hyperglycemia had a worse post MI LV remodelling than euglycemic patients, regardless of their diabetic status.

When we conducted a partial correlation between admission PG and Delta LVEDV after controlling of other parameters affecting the infarction size , we concluded that admission hyperglycemia had an isolated and independent effect on postmyocardial infarction LV remodelling that cannot be explained by the admission infarction size only. By multiple linear regression analysis, we concluded that admission hyperglycemia, infarction size and partial reperfusion were independent predictors of postmyocardial infarction LV remodelling. By multivariable logistic regression analysis, we

concluded that admission hyperglycemia and infarction size were major and independent predictors of LV remodelling after MI with Odd's ratio = 2.167(95%CI: 2.001-2.333) and Odd's ratio = 8.276 (95%CI:2.588-13.964) respectively. These results agreed with those reported by Bauters et al.[16] who analysed LV remodelling in 162 non-diabetic patients with anterior MI. Admission hyperglycemia in their study was defined as a glycemia on admission ≥ 7 mmol/L(126mg/dl). Systematic echocardiographic follow-up was performed at 3 months and 1 year after MI. When LV remodelling was defined as a > 20% increase in EDV, it was observed in 46% patients in the stress hyperglycemia (SH) group vs. 19% patients in the no SH group (P < 0.0008). By multivariable analysis, baseline SWMS(P < 0.001) and SH (P < 0.009) were independently associated with changes in EDV. SH was an independent predictor of LV remodelling [adjusted OR: 3.22 (1.31 7.94)]. They concluded that SH is a major and independent predictor of LV remodelling after anterior MI in non-diabetic patients.

The main finding of our study is that admission hyperglycemia was a major and independent predictor of postmyocardial infarction LV remodelling with Odd's ratio = 2.167[95%CI: 2.001-2.333]. At least two possible explanations may account for this finding. Admission hyperglycemia can either be an indicator of concomitant metabolic abnormalities which may themselves play a role in the remodelling process, or, alternatively, can simply be a marker of more extensive myocardial damage that would not be entirely taken into account by relatively crude measurements of infarct size (peak CK, ECG, or SWMS), but would, nevertheless, lead to an increased remodelling.

CONCLUSIONS

We concluded that patients with admission hyperglycemia had a worse postmyocardial infarction LV remodelling than euglycemic patients, regardless of their diabetic status. Admission hyperglycemia was a major and independent predictor of postmyocardial LV remodelling with Odd's ratio= 2.167[95%CI: 2.001-2.333].

Study limitations

A larger sample size may have a more statistical power. We recommend the use of other objective methods as; SPECT and CMR in estimating LV volumes, function and infarction size that may decrease the observer dependency of echocardiography. A worldwide consensus on the defining level of stress hyperglycemia may help more accurate comparisons between studies.Financial support to document the patency of infarct related artery (IRA) and other coronary arteries with managing any obstructive lesion would minimize the confounding effect of IRA reocclusion and other distant obstructive CAD.

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Conflict of Interests

The authors declare that there was no conflict of interests as regard the publication of this paper.

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