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Original article

Impact of Hyperglycemia on Outcomes of Patients with Acute Myocardial Infarction

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ABSTRACT

Background: Hyperglycemia was found to cause multiple adverse effects on myocardial muscle as it induces oxidative stress, apoptosis, enhances inflammation, and activates coagulation, which deteriorates myocardial damage in the setting of ischemia. **Objective**: Assess the impact of acute and chronic hyperglycemia on In-hospital and short term "6 months" outcomes in patients with acute myocardial infarction. And the effect of hyperglycemia on left ventricular remodeling using speckle-tracking echocardiography. **Subject and methods:** The final study population was 100 patients who were divided according to: A) Admission plasma glucose, they were classified into 2 groups: Non acute group: including 77 patients with RBS (< 200 mg/dl) and Acute group: including 23 patients with RBS (≥ 200 mg/dl). B) Glycated hemoglobin (HbA1c), also cases were classified into another (2) groups: Non chronic group: including 64 patients with normal HbA1c (< 6.5) and Chronic group: including 36 patients with elevated HbA1c (≥ 6.5). **Result:** From the echocardiographic finding in patients with and without acute-HG and in patients with and without chronic-HG, we can say that the increased values of EDV, ESV, E wave, WMSI and E/A ratio, along with decreased values of EF, ś and A wave (parameters of worsening diastolic and myocardial systolic function) in the patients with acute-HG in comparison with the patients without acute-HG, indicates that acute-HG is associated with adverse effects on LV functions, mainly progressive diastolic dysfunction and deterioration of pumping function of the heart (LV remodeling) . Regarding the GLS, our results showed that patients with both acute and chronic HG were having worse GLS in comparison with those

without. **Conclusion:** Both acute and chronic hyperglycemia had been found to be associated with myocardial adverse effects in patients with AMI. And, 2D speckle echocardiography can be used as a good predictor of LV remodeling in those patients.

Keywords: Hyperglycemia; Acute Myocardial infraction; Echocardiography.

Introduction

The increasing incidence and mortality rates of cardiovascular and cerebrovascular diseases over the past 20 years have occurred in parallel with the improvement of living standards worldwide. This increased disease burden poses a serious threat to the health and quality of life, not only to individuals but also to families and society [(1)].

In order to investigate factors affecting outcomes in cardiovascular diseases especially acute myocardial infection (AMI) many studies had investigated the effect of acute (stress) and chronic hyperglycemia on short-term outcomes in patients with AMI.

High admission glycemia "stress hyperglycemia" has been shown to be the marker of high risk in many acute diseases (e.g. apoplexy, sepsis, trauma), including acute myocardial infarction (AMI). SH in AMI is associated with high risk of in-hospital mortality, heart failure, cardiogenic shock, atrial fibrillation, ventricular tachycardia and ventricular fibrillation, bundle branch block, new atrio-ventricular block, no-reflow phenomenon [(2)].

Hyperglycemia is associated with increased levels of inflammatory markers, enhanced expression of cytotoxic T-cells, and reduced expression of T-cells, which are implicated in limiting the immune process. An increased inflammatory immune process seems likely a mechanism linking acute hyperglycemia to poor cardiac outcome in MI patients [(3)].

In the last decades, many noninvasive imaging techniques have demonstrated to be feasible and reproducible for assessment of both systolic and diastolic function in the human heart; among these, echocardiography has gained a predominant role in clinical practice [(4)].

Two dimensional speckle tracking echocardiography is a novel echocardiographic modality that allows analysis of myocardial deformation, including the segmental longitudinal strain, circumferential and radial strain [(5)].

Subjects and methods

This was a single center prospective cross sectional study that was carried out at the cardiology department in Benha university Hospital during the period from December 2017 to August 2018.

Before the start of the study, permission was obtained from Ethical Committee in the faculty of medicine, Benha University. Also, informed written consents from patients included in the study were obtained.

❖ ***Subjects:*** It was conducted on 129 patients who were admitted to CCU with first acute anterior STEMI, treated by thrombolytic reperfusion strategy (streptokinase) according to the following criteria:

* ***Inclusion criteria:***

All patients with acute ST-segment elevation myocardial infarction were selected, It was defined as chest pain of about 30 min duration and electrocardiographic changes with ST segment elevation of > 2 mm in at least 2 precordial leads, and abnormal troponin levels or Creatine Kinase MB at least twice the upper limit of normal.

* ***Exclusion criteria:***

All patients with the following criteria were excluded:

- Patients' refusal.
- Acute myocardial infarction treated by primary percutaneous coronary intervention.
- Any myocardial infarction rather than acute anterior myocardial infarction.
- Patients presented by cardiogenic shock.
- Prior percutaneous coronary intervention or coronary artery bypass graft surgery.
- Any cardiac rhythm other than sinus rhythm.

According to these exclusion criteria, 29 patients were excluded:

The final study population was 100 patients and were classified according to:

A) Admission plasma glucose, they were classified into (2) groups:

- Non acute group: including 77) patients with RBS (< 200 mg/dl).
- Acute group: including 23 patients with RBS (≥ 200 mg/dl).

B) Glycated hemoglobin (HbA1c), also cases were classified into another (2) groups:

- Non chronic group: including 64 patients with normal HbA1c (< 6.5).
- Chronic group: including 36 patients with elevated HbA1c (≥ 6.5).

❖ ***Methods:*** All subjects of the study were subjected to the following:-

A. Informed consents were obtained from all included patients.

B. Full history and thorough clinical examination:

C. Routine investigations:

- Serum CK- MB and Troponin I.
- Kidney and liver Function tests.
- Complete Blood Count.
- Glycated Hb (HbA1c) and random blood sugar on admission.

D. Body Mass Index was calculated.

E. Echocardiographiac evaluation of all patients including:

- Conventional Echo Doppler study.
- Tissue Doppler.
- 2D Speckle Tracking.

❖ ***Statistical Analysis:*** Data were analyzed with SPSS version 15.0 (statistical package for the Social Science, Chicago, IL). Quantitative data were expressed as mean ± standard deviation (SD), data were analyzed by independent sample, unpaired t test, while qualitative data were expressed as number and percentage and were analyzed by Man Whitney test. P-value of <0.05 was considered significant.

Results

According to admission plasma glucose, 23/100 of those patients were having acute-HG. They were 17 (73.9%) males and 6 (26.1%) females with mean age were 55.85 ± 6.71 (42 - 65 years). Their mean body mass index "BMI" was 29.7 ± 2.48 (26 - 34 Kg/m2). On the other hand, there were 77/100 patients without-acute HG. They were 55 (71.4%) males and 22 (28.6%) females with the mean age was 55.26 ± 7.48 (42 - 82 years). Their mean BMI was 29.94 ± 2.97 (23 - 37 Kg/m2). Statistically, there were no statistical significant differences between the two groups among age, BMI, or sex distribution ($p > 0.05$).

Regarding their HbA1c levels, those patients were classified into 2 groups, group for those with chronic-HG, they were 36/100 patients (32 (88.9%) males and 4 (11.1%) females) with mean age was 57.86 ± 8.05 (42 - 82 years) and with mean BMI was 29.08 ± 2.43 (23 - 34 Kg/m^2). The group were for those without chronic-HG, they were 64/100 patients "40 (62.5%) males and 24 (37.5%) females" with mean age was 53.82 ± 6.41 (42 - 67 years). The mean BMI for these patients were 29.43 ± 2.93 (24 - 37 Kg/m2). Statistically, there were statistical significance increase in mean age ($p = .007$)

and frequencies of male gender (p =.005) among chronic cases compared to non-chronic.

There was no significant difference between acute-HG and non-acute HG patients in frequency of HTN, dyslipidemia and smoking, while there was a significant increasing in frequencies of HTN, DM, dyslipidemia and smoking among chronic-HG as compared to non-chronic-HG patients (p < 0.001).

There was a significantly increase in CKMB enzyme among acute-HG patients as compared to patients without acute-HG with p-value: < 0.001; and there was no significant differences in Troponin enzyme between the acute-HG and non-acute-HG patients. While, in patients with the chronic and non-chronic-HG in this study we found a significant increase CKMB and Troponin between both groups with p-value: <0.001 and 0.02 respectively.

Regarding echocardiographic finding in patients with and without acute-HG, our results showed the following

In comparison with patients without acute-HG, patients with acute-HG were having significantly higher EDV pre & post, ESV pre & post, E- wave pre & post, E/A ratio post and E/é ratio pre & post with p-value: 0.007, <0.001, <0.001, <0.001, <0.001, <0.001, 0.001, 0.001 and <0.001 respectively. And also, were insignificant higher as regard E/A ratio pre and á – post with p-value: 0.19 and 0.69.

While in comparison with patients without acute-HG, they were having significant lower values as regard EF pre & post, A-wave pre & post, WMSI pre & post, é wave pre , á post and ś wave pre & post with p-value: <0.001, <0.001, 0.003, <0.001, <0.001, 0.04, 0.04,<0.001 and <0.001 respectively, also they were insignificant lower as regard A-wave pre, é wave post á and wave pre with p-value: 0.08, 0.29 and 0.69 respectively.

Regarding echocardiographic finding in patients with and without chronic-HG, our results showed the following.

Variable		(Non-acute) (n=77)	(Acute) (n=23)	t	p
EDV pre	Mean ± SD	101.90 ± 22.03	115.91 ± 9.39	2.78	0.007
EDV post	Mean ± SD	104.17± 24.95	١٤٦.٥٣ ± ٤٠.٢١	2.41	<0.0٠١
ESV pre	Mean ± SD	٥4.78 ± 6.39	73.48 ± 9.83	5.1	<0.001
ESV post	Mean ± SD	٥7.22 ± 6.20	78.57 ± 17.07	4.89	<0.001
EF pre	Mean ± SD	4٨.95 ± 4.44	36.61 ± 4.99	4	<0.001
EF post	Mean ± SD	4٧.01 ± 4.22	35.13 ± 7.34	4.86	<0.001
E- pre	Mean ± SD	48.60 ± 15.03	63.92 ± 23.21	MW 3.67	<0.001
E- post	Mean ± SD	50.22 ± 17.99	73.82 ± 30.81	4.47	<0.001
A pre	Mean ± SD	102.08 ± 28.65	81.99 ± 37.15	1.76	0.08
A post	Mean ± SD	99.22 ± 28.90	78.55 ± 35.87	2.95	0.003
E/A pre	Mean ± SD	0.71 ±0.48	1.13 ± 0.83	1.32	0.19
E/A post	Mean ± SD	0.73 ±0.56	1.41 ± 1.05	3.41	0.001
WMSI pre	Mean ± SD	١.33 ± 0.44	2.51 ± 0.30	7.26	<0.001
WMSI post	Mean ± SD	١.35 ± 0.51	٢.٨ ± 0.32	8.40	<0.001
é - Pre	Mean ± SD	6.31 ± 0.66	6.01 ± 0.51	2.03	0.04
é - post	Mean ± SD	6.14 ± 0.63	5.97 ± 0.68	1.06	0.29
E / é - Pre	Mean ± SD	7.86 ± 3.11	10.78 ± 4.61	MW 3.44	0.001
E / é - Post	Mean ± SD	8.33 ± 3.77	12.78 ± 6.33	MW 3.92	<0.001
á - pre	Mean ± SD	3.71 ± 0.66	3.41 ± 0.51	2.03	0.04
á - post	Mean ± SD	3.44 ± 0.63	3.51 ± 1.1	0.40	0.69
ś - pre	Mean ± SD	8.19 ± 0.88	7.36 ± 1	3.85	<0.001
ś - post	Mean ± SD	8.21 ± 0.85	7.07 ± 1.48	4.69	<0.001
GLS	Mean ± SD	-14.87 ± 1.73	-11.51 ± 2.83	4.82	<0.001

In comparison with patients without chronic-HG, patients with chronic-HG were having significantly higher findings regarding ESV pre & post, E wave pre & post, E/A ratio pre & post and E/é pre & post findings with p-value: <0.001 for each. And were insignificantly higher as regard EDV pre and post findings with p-value: 0.054 and 0.50 respectively.

Variable		(non-chronic) (n=64)	(chronic) (n=36)	t	p
EDV pre	Mean ± SD	99.92 ± 9.35	109.69 ± 9.16	1.95	0.054
EDV post	Mean ± SD	105.03 ± 9.19	119.63 ± 14.24	0.69	0.040
ESV pre	Mean ± SD	53.67 ± 6.5	66.3 ± 7.8	5.89	<0.001
ESV post	Mean ± SD	56.4 ± 6.95	73.91 ± 13.59	4.63	<0.001
EF pre	Mean ± SD	46.9 ± 4.62	40.47 ± 3.14	6.27	<0.001
EF post	Mean ± SD	47.18 ± 4.28	38.16 ± 4.94	7.44	<0.001
E- pre	Mean ± SD	43.2 ± 6.83	67.96 ± 21.45	8.52	<0.001
E- post	Mean ± SD	44.98 ± 12.68	74.6 ± 26.73	MW 7.51	<0.001
A pre	Mean ± SD	115.15 ± 14.82	65.99 ± 29.44	11.11	<0.001
A post	Mean ± SD	111.96 ± 18.14	63.35 ± 28.5	10.40	<0.001
E/A pre	Mean ± SD	0.52 ± 0.2	1.31 ± 0.72	8.17	<0.001
E/A post	Mean ± SD	0.52 ±0.29	1.52 ± 0.88	8.32	<0.001
WMSI pre	Mean ± SD	1.29 ± 0.42	1.92 ± 0.56	3.74	0.03
WMSI post	Mean ± SD	1.39 ± 0.51	1.83 ± 0.68	3.83	<0.001
é - Pre	Mean ± SD	6.45 ± 0.75	5.85 ± 0.57	5.01	<0.001
é - post	Mean ± SD	6.27 ± 0.57	5.78 ± 0.65	3.91	<0.001
E / é - Pre	Mean ± SD	6.76 ± 1.6	11.66 ± 4.22	MW 8.25	<0.001
E / é - Post	Mean ± SD	7.2 ± 2.78	13.16 ± 5.36	MW 7.33	<0.001
á - pre	Mean ± SD	3.85 ± 0.57	3.25 ± 0.57	5.01	<0.001
á - post	Mean ± SD	3.57 ± 0.57	3.23 ± 0.98	2.20	0.03
ś - pre	Mean ± SD	8.4 ± 0.91	7.29 ± 0.6	6.46	<0.001
ś - post	Mean ± SD	8.44 ± 0.86	7.05 ± 0.99	7.32	<0.001
GLS	Mean ± SD	-15.27 ± 1.87	-13.5 ± 4.8	4.05	<0.009

In comparison with patients without chronic-HG, patients with chronic-HG were having significantly lower findings regarding EF pre & post, A wave pre & post, WMSI pre & post, é wave pre & post, á pre & post and ś wave pre & post findings with p-value: <0.001 for each and 0.003 for WMSI pre only.

Regarding the GLS, the current study results showed that patients with acute HG were having worse GLS value in comparison with patients without acute-HG with (p-value: 0.006). Also, the patients with chronic-HG were having worse GLS value in comparison with those without chronic-HG with (p-value: 0.009).

There was significant correlation between GLS and other Echo parameters in the current study, our results showed that there was a significant positive correlation between GLS and EDV, ESV, E wave, E/A ratio and E/é ratio and a significant negative correlation between GLS and EF, WMSI, A wave, é wave and ś wave.

DISCUSSION

Hyperglycemia (HG) is associated with increased morbidity and mortality in patients with an AMI **(6)**. HG on admission is common in AMI patients regardless of diabetic status, and is known as one of prognostic factors **(7)**. HG upon hospital admission in patients with ST-segment elevation myocardial infarction (STEMI) occurs frequently and is associated with adverse outcomes. It is; however, unsettled as to whether an elevated blood glucose level is the cause or consequence of increased myocardial damage **(8)**.

the current study showed a significantly increase in CKMB enzyme among acute-HG patients as compared to patients without acute-HG with (p-value < 0.001); These results are in agreement with the study **(1)** that measured CK-MB every 3 hours from admission until it reached its peak and found that patients with acute-HG had a significantly higher peak CK MB than those without (p <0.001). Also, other researchers reported in their study that peak creatine kinase was obtained in 1187 (94%) patients. Peak creatine kinase was significantly higher in patients with acute hyperglycemia than in patients without (P = 0.004).(9)

In patients with the chronic and non-chronic-HG in this study we found a significant increase in CK-MB and Troponin between both groups with (p-value <0.001 and 0.02 respectively). This contradicts the findings reported before **(1)** that there was no significant difference in peak CK MB between patients with chronic-HG and those without (p-value= 0.59).

From the echocardiographic finding in patients with and without acute-HG and in patients with and without chronic-HG, we can say that the increased values of EDV, ESV, E wave, WMSI and E/A ratio, along with decreased values of EF, ś and A wave (parameters of worsening diastolic and myocardial systolic function) in the patients with acute-HG in comparison with the patients without acute-HG, indicates that acute-HG is associated with adverse effects on LV functions, mainly progressive diastolic dysfunction and deterioration of pumping function of the heart (LV remodeling).

In the study done ***2013*** [(2)] it was reported that LVEF changed in patients without both known DM and SH from 52.3±11% to 54.5±9% with no significant difference, LVESV changed in patients without both known DM and SH from 72.6±25.0 ml to 63.0±25.0 ml with no significant difference and LVEDV increased significantly in patients with SH without known DM from 126.8±37 ml to 145.7±30.9 ml after one year (t= – 2.835, P=0.009) and concluded that the significant increase of EDV during one year could be marker of LV remodeling and SH is associated with LV remodeling and high in-hospital mortality risk.

It was reported before that LV remodeling was observed in 46% patients in the SH group vs. 19% patients in the no SH group (P = 0.0008). And concluded that SH is a major and independent predictor of LV remodeling after anterior MI in non-diabetic patients. [(10)]

In the study done ***2003*** [(3)] it was concluded that, compared with known diabetic patients, patients with new hyperglycemia presented with higher infarct segment length (P < 0.04), wall motion score (P < 0.05), and MPI (P < 0.04), but lower transmitral Doppler flow (P < 0.05) and pulmonary venous flow analysis (P < 0.05). And concluded that during MI, hyperglycemia is associated with increased levels of inflammatory markers, enhanced expression of cytotoxic T-cells, and reduced expression of T-cells, which are implicated in limiting the immune process. An increased inflammatory immune process seems a likely mechanism linking acute hyperglycemia to poor cardiac outcome in MI patients.

In the retrospective study done previously in 2006***)*** on 543 patients with AMI and reported that age, heart rate on admission and infarct size statistically significantly correlated with hyperglycemia and a trend for statistically significance was found for higher blood glucose values in women. They explain their findings by for connection of stress hyperglycemia and mortality is probably occurs due to larger infarctions result in more pronounced sympathetic nervous system activation and catecholamine secretion. That leads to higher glycemia, on one hand, and higher mortality, on the other. [(11)]

Some other studies were also in agreement with our results in that, the myocardial changes occurring with acute HG were worse than that occurring with chronic-HG. ***Fujino et al. (2014)***[(1)] reported that acute-HG, but not chronic-HG, was associated with adverse short-term outcomes after AMI. Paradoxically, in patients with acute-HG,

chronic-HG might abate the adverse effects of acute-HG.

It was also reported that acute hyperglycemia was associated with higher in-hospital mortality, higher levels of cardiac biomarkers and decreased in left ventricular ejection fraction thereby suggesting larger infarct size as compared to chronic hyperglycemia. [12]

Regarding the GLS, our results showed that patients with both acute and chronic HG were having worse GLS in comparison with those without. These findings were in agreement with the study which reported that, the patients with LV remodeling (-11.2±2.5) were having worse GLS value in comparison with those without LV remodeling (-14.8±3.2) with (p-value: 0.003). And concluded that 2D-STE early in acute STEMI is a valuable predictor of LV remodeling after 6 months. [13]

Recently, it was reported that in LV remodeling group, There was statistically significant higher LV peak systolic GLS values in comparison with the LV non-remodeling group. And concluded that Average peak systolic GLS at echocardiography done early after myocardial infarction are independent predictors of LV remodeling after anterior STEMI and can be used to predict occurrence of LV remodeling after 6 months. [14]

Conclusion

Both Acute and Chronic Hyperglycemia had been found to be associated with myocardial adverse effects in patients with AMI. And, 2D speckle echocardiography can be used as a good predictor of LV remodeling in those patients.

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