Impact of acute hyperglycemia after primary angioplasty for acute myocardial infarction : GLAMI Study

Impact de l'hyperglycémie aigue après angioplastie primaire à la phase aigüe d'un infarctus du myocarde: Etude GLAMI

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Summary

Background: Hyperglycemia has been shown to be a powerful predictor of worse outcome after ST segment-elevation myocardial infarction (STEMI).

The aim of this study was to investigate the relation between acute hyperglycemia and angiographic and clinical outcome after primary or rescue angioplasty for STEMI.

Methods: We prospectively included 383 patients who underwent revascularization for STEMI. Hyperglycemia was defined as plasma glucose of 11mmol/l (198mg/dl).

Results: Among the 383 patients with STEMI included in the study, 158 (41.2%) patients had hyperglycemia. Angioplasty success, TIMI 3 flow and ST segment resolution were significantly lower in acute hyperglycemia group. On multivariate regression, hyperglycemia wasn't found to be an independant predictor of angioplasty success (p=0.08) or of ST resolution, however diabetes was independently associated with non ST segment resolution after achieving TIMI 3 flow (p=0.014). The in hospital mortality rate was significantly higher in patients with hyperglycemia (18.4% versus 5%, p<0.003). In multivariate analysis, independent inhospital mortality predictors were: Heart failure (OR: 8.9; 95% IC [3.4 -23]; p<0.0001), acute hyperglycemia (OR: 3.8; 95% IC [1.4 – 9.8]; p=0.005), renal insufficiency (OR: 6.5; 95% IC [2.3-18]; p<0.001), and anemia (OR=4.7, 95% CI [1.9 – 11.6], p=0.001). Among the hyperglycemia patients, mortality predictors were: glycemia level (p=0.005), Killip class (p=0.001), blood hemoglobin level (p=0.007), and angioplasty success (p=0.022).

Conclusion: Hyperglycemia in patients with STEMI is an important predictor of mortality with an increasing mortality risk even beyond 11mmol/l but diabetes is a better predictor of non ST resolution after TIMI 3 restoring.

Keywords

Myocardial infarction, Diabetes, Angioplasty, Mortality

Résumé

IIntroduction: L'hyperglycémie est un facteur prédictif de mauvais pronostic après un syndrome coronarien aigu avec élévation du segment ST (SCA ST(+)).

L'objectif de cette étude est d'étudier la relation entre l'hyperglycémie et la survenue d'évènements cliniques et angiographiques après angioplastie primaire ou de sauvetage.

Méthodes: Nous avons inclus 383 patients ayant subi une revascularisation pour SCA ST(+). L'hyperglycémie aiguë est définie comme une glycémie supérieure à 11 mmol/l.

Résultats: 158 (41,2%) patients avaient une hyperglycémie. Succès d'angioplastie, obtention d'un flux TIMI 3 et résolution du segment ST étaient significativement plus faible dans le groupe hyperglycémie. L'hyperglycémie n'a pas été isolée comme facteur prédictif de succès d'angioplastie (p = 0,08) ou de résolution du segment ST, mais le diabète était indépendamment associée à la non résolution du segment ST après obtention d'un flux TIMI 3 (p = 0,014). La mortalité hospitalière était plus élevée chez les patients avec hyperglycémie (18,4% versus 5%, p <0,003). Les facteurs prédictifs de mortalité étaient: l'insuffisance cardiaque (OR: 8,9; IC 95% [3.4 -23], p <0,0001), l'hyperglycémie (OR: 3,8; IC 95% [1,4-9,8], p = 0,005), l'insuffisance rénale (OR: 6,5; IC 95% [2,3-18], p <0,001), et l'anémie (OR = 4,7, IC 95% [1.9-11.6], p = 0,001). Dans le groupe hyperglycémie, les facteurs prédictifs de mortalité étaient: taux de glycémie (p = 0,005), classe Killip (p = 0,001), taux d'hémoglobine (p = 0,007), et le succès d'angioplastie (p = 0,022).

Conclusion: L'hyperglycémie chez les patients atteints de SCA ST (+) représente un facteur prédictif indépendant de mortalité. Le diabète était indépendamment associé à la non résolution du segment ST après obtention d'un flux TIMI 3

Mots-clés

Infarctus du myocarde, Diabète, Angioplastie, Mortalité

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INTRODUCTION

Hyperglycemia (HG) has been shown to be a powerful predictor of worse outcome after ST segment-elevation myocardial infarction (STEMI) [1].

An increase of plasma glucose concentration is often observed during early hours after the onset of acute myocardial infarction (AMI) not only in patients with diabetes mellitus but also in patients without diabetes mellitus [2]. It has been reported that both acute hyperglycemia and diabetes mellitus are independently associated with adverse outcomes after AMI in the prereperfusion era and in the thrombolytic era [3-5]. Primary percutaneous coronary intervention (PCI) has been shown to be more effective than thrombolytic therapy for the treatment of AMI [5]. Recent progress in treatment of AMI might have changed the association between acute hyperglycemia and outcome after AMI [6].

OBJECTIVE

he aim of this study was to investigate the relation between acute hyperglycemia and in hospital outcome after primary angioplasty for STEMI.

PATIENTS AND METHODS

Patients

The GLAMI study is a retrospective observational monocenter study conducted between January 2005 and September 2015. A total of 383 consecutive patients who were admitted to the participating institutions within 48 hours after the onset of AMI were enrolled in the GLAMI.

Acute myocardial infarction was defined by a combination of 2 of the following 3 characteristics: chest pain consistent with ongoing myocardial ischemia persisting longer than 30 minutes.

Plasma glucose was measured at hospital admission.

Acute hyperglycemia was defined as plasma glucose of 11 mmol/L (198 mg/dL), regardless of the diabetic status.

End points

The primary end point was all-cause inhospital mortality.

Other important clinical outcomes, including cardiac death, reinfarction, unstable angina, heart failure, and stroke, and angiographic result (TIMI 3 flow restoring, angioplasty success defined as TIMI 3 flow with residual stenosis less than 20%, and electrocardiographic ST elevation regression more than 50%) were also assessed during hospitalization.

STATISTICAL ANALYSIS

Differences between group means at baseline were assessed with the two-tailed Student t test.

Chi-square analysis was used to test differences between proportions. When the expected frequency of a variable was <5, the Fisher exact test was used.

Independent contribution of parameters was assessed by multivariate logistic regression analysis. A P-value <0.05 was considered significant.

The Statistical Package for the Social Sciences (SPSS Inc.) version 24.0 was used for all statistical analysis

RESULTS

Patient characteristics

Acute hyperglycemia was associated with more women and more diabetes mellitus (Table 1). There was no significant difference in medications before AMI between patients with acute hyperglycemia and patients without, except for more use of oral hypoglycemic drugs and insulin in patients with acute hyperglycemia.

Table 1. Baseline characteristics

	Acute HG (-)	Acute HG (+)	p Value
Age (years)			
Male n(%)	176 (86,7%)	108 (76.1%)	0.011
Hypertension n(%)	54 (26.6%)	47 (33.1%)	0.19
Smoking n(%)	143 (70.4%)	87 (61.7%)	0.09
Diabetes Mellitus n(%)	26 (12,8%)	91 (64,1%)	<0.0001
Admission before H4 n(%)	79 (39.7%)	52 (37.4%)	0.67
Anterior wall infarction n(%)	96 (48%)	66 (47.1%)	0.87
Creatin kinase peak (IU/l)	2445 + 2234	2322 + 1951	0.59
Heart failure at admission n(%)	55 (27.1%)	42 (29.6%)	0.614
TIMI flow 0 or 1 n (%)	149 (77.6%)	102 (75.6%)	0.66
Stenting n(%)	160 (82.1%)	105 (79.5%)	0.57

In hospital outcomes

The in hospital mortality rate was significantly higher in patients with acute hyperglycemia than in patients without (Table 2). Major adverse cardiovascular events, including cardiac death, reinfarction, unstable angina, heart failure and stroke, occurred more frequently in patients with acute hyperglycemia. The inhospital mortality increased as plasma glucose increased (Figure 1). An increase of 1 mmol/L (18 mg/dL) in plasma glucose was associated with an increase in mortality risk of 13% in univariate analysis (OR 1.13, 95% CI [1.08-1.19], P < .001) and 12% in multivariate analysis (OR 1.12, 95% CI [1.06-1.19], P <.001).

Table 2. In hospital Outcome

	Acute HG (-)	Acute HG (+)	P value
Angiographic success n(%)	176 (86,7%)	107 (75,4%)	0.007
TIMI flow 3 n(%)	179 (88.2%)	113 (79.6%)	0.029
Non ST resolution n(%)	19 (9.6%)	24 (17.9%)	0.027
Renal Insuff creat>150µmol/l n(%)	12 (6%)	20 (14,2%)	0.01
Severe arrythmias n(%)	13 (6.4%)	23 (16.2%)	0.004
Atrial fibrillation n(%)	10 (4.9%)	13 (9.2%)	0.12
Auriculo ventricular block, n(%)	22 (10.8%)	19 (13.4%)	0.47
Delayed heart failure n (%)	35 (17.2%)	44 (31%)	0.03
Pericardites en (%)	5 (2.5%)	13 (9.2%)	0.006
Death n(%)	10 (5%)	26 (18,4%)	<0.001

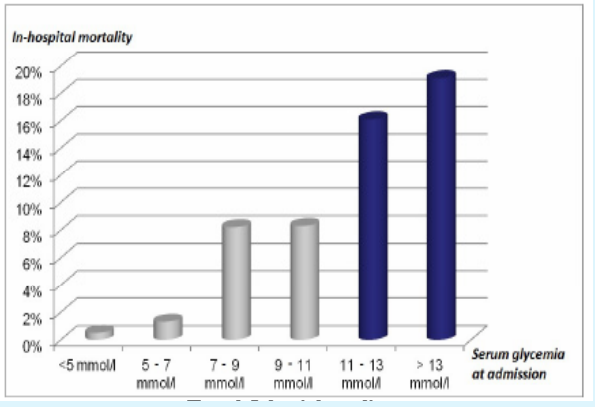

Figure 1. In hospital mortality rate

Among the 383 patients with STEMI included in the study, 158 (41.2%) patients had acute hyperglycemia. There was no difference among the two groups with regard to clinical characteristics and cardiovascular risk factors. Hemodynamic parameters (heart rate and systolic and diastolic blood pressure, Killip class) were overall similar for the two groups. The incidence of anterior wall location of the infarction was similar (47.1 % versus 48%, p=0.68).

Angioplasty success (75.4% vs 86.7%, p=0.007), TIMI 3 flow restoring (79.6% vs 88.2%, p=0.029) and ST segment resolution (82.1% vs 90.4%, p= 0.027) were significantly lower in acute HG group. On multivariate regression, HG wasn't found to be an independant predictor of angioplasty success (p=0.08, OR=0.9, 95%IC [0.92-1]) or of ST resolution (Table 3). Diabetes but not hyperglycemia was independently associated with non ST segment resolution after achieving TIMI 3 flow (p=0.014, OR= 3.2, 95% CI [1.02 – 8.1] (Table 4).

Table 3. Multivariate analysis of angioplasty success

	P value	OR	95% CI
Sex (male)	1391	1370	4318
Serum creatinine > 150 µmol/l	156 (11.2%)	295 (21.5%)	462 (10.7%)

Table 4. Mutltivariate analysis of non ST regression after TIMI 3 flow achieving.

	P value	OR	95% CI	P
Time sypmtom onset to balloon inflation > 6h	<0.001	5.61	2.2 – 14	
Diabetes	0.014	3.2	1.02 – 8.1	<0.001
Heart failure at admission	0.032	2.76	1.15 – 7.02	<0.001

The in hospital mortality rate was significantly higher in patients with acute hyperglycemia than in patients without (18.4% versus 5%, p <0.001).

In multivariate analysis (Table 5), independently of other determinants of death (age, risk factors, location of STEMI, infarct size, incomplete resolution of ST-segment and angiographic success), the predictor of in hospital mortality were: Heart failure (OR: 8.9; 95% IC, 3.4 -23; p<0.0001), acute hyperglycemia (OR: 3.8; 95% IC, 1.4 – 9.8; p=0.005), renal insufficiency (OR: 6.5 ; 95% IC, 2.3-18; p<0.001), and anemia (OR=4.7, 95% IC : 1.9 – 11.6, p=0.001).

Table 5. Multivariate analysis of mortality

	Odds Ratio	95% CI	p
Congestive heart failure	2.6	3.8 -46	0.0001
Acute hyper glycemia	1.49	1.37- 22.1	0.0016
renal insufficiency	2.6	3.8 – 46	0.0001

On multivariate regression, independent mortality predictors among HG group were: Killip class, blood hemoglobin and glycemia levels, and PCI result. Mortality increases when killip class is higher (OR=2.14 when moving from a class to the next one) and glycemia is higher (OR=1.13 when glycemia increases by 1 mmol/l more than 11 mmol/). It decreases when blood hemoglobin is higher (OR=0.69 when blood hemoglobin is 1 g/dl higher) and when there is a success of PCI (OR=0.25) (Table 6).

Table 6. Multivariate regression for mortality among hyperglycemic patients

	Odds Ratio	95% CI	P
Killip class	2.14	1.36 - 3.35	0.001
Blood Hb	0.69	0.53 – 0.90	0.007
Glycemia	1.13	1.03 – 1.23	0.005
PCI success	0.25	0.08 – 0.82	0.022

PCI: percutaneous coronary intervention

DISCUSSION

In this retrospective study, among the parameters examined, hyperglycemia was a consistent predictor for mortality and morbidity in patients following STEMI mechanically treated.

The association between glucose levels and outcomes appeared independent of the diabetes status of patients, although some of the hyperglycemic patients could have had undiagnosed pre-existing diabetes.

In the early phase of STEMI, the acute glycometabolic response to stress is heterogeneous (ranging from no insulin resistance to very high glucose levels and, finally, to the combination of increased glucose values and insulin resistance). Increased glucose values are stronger prognostic factors since they are independently associated with in-coronary care units mortality and complications [7].

Carmen Wong et al.[8] studied plasma cortisol concentration with the degree of hyperglycemia in subjects experiencing STEMI, and their later glucose metabolic status. They found that as many as 38.5% of subjects were found to have newly diagnosed glucose intolerance at follow up. HG in patients who are more unwell (i.e. higher cortisol) reflects the stressed state rather than underlying glucose intolerance. Conversely, if the patient is less sick (i.e. lower cortisol), HG is more likely to reflect underlying glucose intolerance.

The interaction between HG and diabetes has been studied by Gasior [9] who found that elevated blood glucose levels in STEMI affect the prognosis of patients without DM; however, it is not an independent death risk factor of patients with DM treated with PCI.

Ergelen et al. [10] divided STEMI patients to 4 groups (diabetic/not cross HG/not). His study shows that after adjustment for potentially confounding factors, non diabetic HG and diabetic HG, but not diabetic non HG status remained independent predictors of long-term cardiovascular mortality. STEMI patients with non diabetic HG represent the highest risk population for in-hospital mortality.

It has been demonstrated that increased plasma glucose at admission is associated with adverse outcome after AMI in the reperfusion era [11]. Wahab et al. [4] have reported that plasma glucose is an independent predictor of mortality after AMI in the thrombolytic era. However, only 34% of the study patients underwent thrombolytic therapy, and PCI was performed in <10% of the patients. Recent studies show similar results in hard endpoints (in-hospital mortality and complications) when patients are treated with primary PCI [12,13].

GLAMI Study is a study in which all of the patients underwent PCI as reperfusion therapy. Our results are in agreement with other studies.

Greig et al. [14] found in a registry of 1,634 consecutive patients that HG at entry was associated with a greater hospital and long term mortality, independently of the reperfusion strategy utilized but primary PCI was associated to a greater benefit, compared to thrombolysis among hyperglycemic, independently of a previous history of diabetes mellitus and TIMI risk score.

Kruk et al. [12] found that HG, anemia, and impaired renal function are independently of each other related to in-hospital death in patients with STEMI treated

with primary PCI. The triad risk factors cluster and accumulation of these risk factors is related to stepwise, additive increase of risk of in-hospital mortality. In our study, HG and renal insufficiency have been found to be a strong predictor of mortality, anemia is a mortality predictor among HG patients.

A more particular complication of PCI that is contrast induced nephropathy is also boosted by HG. Marenzi et al. [15] showed that acute HG was an independent predictor of contrast induced nephropathy and in-hospital mortality and that those with acute hyperglycemia that developed contrast induced nephropathy had the highest mortality rate (38%).

It remains controversial whether acute hyperglycemia predisposes to adverse outcome or is simply a consequence of large infarct size. A higher incidence of Killip class ≥ 2 suggests that acute HG may reflect extensive myocardial damage. However, experimental studies have suggested that hyperglycemia per se, via microvascular impairement, exacerbates myocardial damage in AMI [16]. This has been more detailed using cardiac magnetic imaging (CMR): Cochet et al. [17] performed CMR to 113 patients with STEMI treated with successful primary PCI to evaluate left ventricular function and perfusion data after injection of gadolinium-DTPA. Their results showed the existence of a strong relationship between glucose metabolism impairment and myocardial damage in patients with STEMI. Jensen CJ et al. [18] also performed CMR to 107 STEMI patients treated with primary PCI and found that HG at admission was independently associated with the presence and extent of microvascular obstruction on contrast-enhanced CMR. Microvascular obstruction as assessed by CMR may be a mechanism that relates admission HG in acute STEMI to worse outcome. Cruz-Gonzalez I et al. studied Infarct size using single-photon emission computed tomography (SPECT) in 347 STEMI patients who underwent primary PCI. No significant difference was found in terms of final TIMI 3 flow achievement but the infarct size was larger in the hyperglycemia group. HG on admission was an independent predictor of infarct size at post-MI [19].

Several studies used the TIMI frame count to reflect coronary flow [20,21]. Patients with acute HG, corrected TIMI frame counts were significantly higher compared with those in patients without acute HG and corrected TIMI frame count was independently associated with plasma glucose level. They conclude that acute HG was associated with the impairment of epicardial coronary flow after primary stent implantation and this mechanism might be responsible for the increased infarct size.

HG increases interstitial fibrosis and myocyte apoptosis that exaggerate left ventricular remodeling. Also, hyperglycemia abolishes the cardioprotective effect of ischemic preconditioning by closing K ATP channels [22].

Another potential mechanism for the association between acute hyperglycemia and adverse outcome is microvascular dysfunction [23], as shown by CMR data [17-19].

Chi HJ et al. found on 267 patients who received successful PCI that HG was an independent predictor of incomplete STR in relation with abnormal coronary microvascular reperfusion, which may contribute, at least in part, to the poor outcomes in these patients [24]. In our study, HG was not, but diabetes was. We think that microvascular impairement and non refmow phenomenon may be related to a more chronic HG status, wheareas in non diabetic patients, HG may reflect a more severe stress situation. In both cases, HG is a pejorative marker of prognosis.

Hyperglycemia may augment thrombus formation. A clinical study suggests that a microthrombus in the capillaries plays a crucial role in the no-reflow phenomenon after AMI [25].

Blood glucose is an independent predictor of platelet-dependent thrombosis, even in the normal range. Hyperglycemia may also attenuate the impact of ischemic preconditioning, which is an independent predictor of the no-reflow phenomenon.

These findings are emphasized by similar result in ACS (not only STEMI) [26,27].

Worse outcome including death and MACE associated to HG remain at mid and long term follow-up [10,28].

CONCLUSION

Acute hyperglycemia in patients with STEMI is an important predictor of mortality with an increasing mortality risk even beyond 11mmol/l but diabetes is a better predictor of non ST resolution after TIMI 3 restoring. This suggests the usefulness of assessment of glycemic metabolism in the setting of reperfusion for acute myocardial infarction and the beneficial effect of strict glycemic control.

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