Prognostic Value of Stress Hyperglycemia for In-Hospital Outcome in Acute Coronary Artery Disease

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Abstract

Background: In acute coronary syndrome (ACS), admission hyperglycemia is associated with adverse cardiovascular events in diabetic and nondiabetic patients.

Objective: To assess the prognostic value of stress hyperglycemia for the in-hospital outcome of patients admitted due to ACS.

Methods: This study included 152 patients admitted to the chest pain unit of a tertiary hospital diagnosed with ACS, who had admission blood glucose data, from September 2005 to February 2010. Group I comprised patients with stress hyperglycemia, defined as admission blood glucose concentration ≥ 126 mg/dL for nondiabetic individuals and admission blood glucose concentration ≥ 200 mg/dL for diabetic individuals. Group II was formed by patients with admission blood glucose concentration lower than those established. The association of hyperglycemia and in-hospital outcome was assessed.

Results: Stress hyperglycemia associated with in-hospital complications, age increase and female sex. On multivariate analysis, only female sex (OR = 2.04; 95% CI: 1.03 – 4.06; p = 0.007) and in-hospital complications (OR = 3.65; 95% CI: 1.62 – 8.19; p = 0.002) associated independently with admission hyperglycemia.

Conclusion: Stress hyperglycemia is an independent predictive factor for in-hospital complications after ACS in diabetic and nondiabetic patients. The results highlight the need to assess admission blood glucose concentration in all patients admitted due to ACS, including nondiabetic ones, aiming at identifying those at higher risk for complications. (Arq Bras Cardiol. 2013;100(2):127-134)

Keywords: Hyperglycemia / complications; length of stay; prognosis.

Introduction

In acute coronary syndrome (ACS), stress hyperglycemia, defined as high blood glucose concentration on hospital admission, is a frequent condition[1], present in 25% to 50% of patients admitted due to ACS[2]. Stress hyperglycemia in coronary artery disease (CAD) is associated with the presence of cardiovascular adverse events and increased mortality in patients with or without diabetes mellitus (DM)[3-6].

High blood glucose concentration is caused by an inflammatory and adrenergic response to ischemic stress, when catecholamines are released and glycogenolysis induced[1,7]. Hyperglycemia associates with an increase in free fat acids, which induces cardiac arrhythmias and insulin resistance, causes chemical inactivation of nitric oxide and the production of oxygen reactive species, generating oxidative stress, which produces microvascular and endothelial dysfunction, a prothrombotic state, and vascular inflammation[8-10]. It is related to myocardial metabolic disorders, leading to thrombosis, extension of the damaged area, reduced collateral circulation, and ischemic preconditioning[1,7]. Its exact pathophysiological mechanism, however, has not been well established[2].

A consensus on the minimum blood glucose concentration that carries a risk, however, still lacks[11]. In their studies, Gois[12] and Capes et al[13] have reported that blood glucose concentrations greater than 110 mg/dL in nondiabetic patients and equal to or greater than 180 mg/dL in diabetic ones are a risk for hospital complications in ACS. Timmer et al[14] have considered values over 140 mg/dL for nondiabetic patients. In the HI-5 study, six-month mortality was considerably greater in patients with acute myocardial infarction (AMI), whose mean blood glucose concentration was over 144 mg/dL[15].

This study aimed at assessing the prognostic value of stress hyperglycemia in the in-hospital outcome of patients admitted due to ACS.

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Methods

Study population

From September 2005 to February 2010, 549 patients were admitted to the Registry of Acute Coronary Syndrome of the Hospital São Lucas (Solar). That registry is an in-hospital cohort of patients diagnosed with ACS and admitted to the Chest Pain Unit of the Hospital São Lucas, in the city of Aracaju, Sergipe state, for investigation and treatment. The following patients were excluded from that registry: those not meeting the ACS criteria; those refusing to make their data available for the study; and those transferred to other health care units.

The diagnosis of AMI was established in the presence of an increase and gradual reduction in troponin or the MB isoenzyme of creatine phosphokinase (CK-MB) in association with at least one of the following criteria: ischemic symptoms; development of pathological Q waves; and electrocardiographic alterations indicating ischemia (ST-segment elevation or depression). The diagnosis of unstable angina was based on compatible clinical findings, no elevation of cardiac enzymes, and at least one of the following criteria: electrocardiographic ischemic changes, except for ST-segment elevation; ischemic changes on stress echocardiography or exercise test, or occasionally on echocardiography at rest, associated with coronary artery lesion on coronary angiography[16].

Of the patients admitted to the registry in the period studied, 397 were excluded from the study because either they lacked admission blood glucose concentration data, or their admission blood glucose levels had not been measured at the institution laboratory (capillary blood glucose test). Thus, this study sample comprised 152 patients divided into two groups: Group I, formed by patients with stress hyperglycemia, defined as blood glucose level on admission equal to or over 126 mg/dL in nondiabetic patients, or equal to or over 200 mg/dL in diabetic patients; and Group II, formed by patients with admission blood glucose concentration lower than those values.

Study protocol

The following characteristics of the groups were compared: clinical characteristics (age, sex, cardiovascular risk factors, pathological and therapeutic antecedents); clinical findings on admission; clinical presentation of ACS; laboratory tests; electrocardiographic and echocardiographic changes; treatment during hospitalization; and in-hospital outcome.

Regarding cardiovascular risk factors, the following were assessed: family history of CAD; smoking habit; sedentary lifestyle; systemic arterial hypertension (SAH); DM; and dyslipidemia (DLP). All individuals reporting having DM, SAH and DLP, or undergoing treatment with a specific agent, were considered as having those pathologies. A family history was considered positive for all individuals reporting a first-degree relative of the male sex under the age of 55 years or of the female sex under the age of 65 years with a history of AMI. Smoking habit was considered for those smoking regularly at least one cigarette per day, for at least 30 days before hospital admission. Patients reporting previous regular use of tobacco at any time in their lives, except for the 30 days preceding hospital admission, were considered ex-smokers. Sedentary lifestyle was considered for all patients reporting practicing no physical activity, or doing it at a frequency lower than three days per week, or with a duration lower than 90 minutes per week[17]. In addition, history of the following was also assessed: deep venous thrombosis (DVT); cerebrovascular accident (CVA); congestive heart failure (CHF); arrhythmias; and previous use of drugs (anti-hypertensive agents, antidiabetic drugs, nitrates, digitalis, antiplatelet drugs, lipid-lowering drugs, and benzodiazepines).

On admission, the following parameters were assessed: heart rate (HR); systolic blood pressure (SBP); diastolic blood pressure (DBP); renal function (urea and creatinine); and Killip classification.

The first echocardiography performed after hospital admission was selected for analysis, and left ventricular ejection fraction was the variable compared.

Coronary angiography was assessed according to the presence of coronary lesions. When present, the lesions were classified as single-, two-, or thee-vessel disease, according to the number of major coronary arteries with stenosis $\geq$ 50% and the presence of left main coronary artery lesion[18].

Follow-up

The patients admitted due to ACS at the Hospital São Lucas were followed up during their hospital stay with daily visits. The indicators used to assess their in-hospital outcome were the length of hospital stay, in days, and the presence of the following in-hospital complications: 1) cardiovascular: acute pulmonary edema (APE); in-hospital CVA; shock; post-infarction arrhythmia; cardiopulmonary arrest (CPA); death; ischemic complications (angina, infarction, reinfarction, and need for new invasive intervention); and 2) noncardiac: infection and respiratory failure of noncardiovascular origin.

Statistical analysis

Categorical variables were described as number of cases and percentages, and the groups were compared by use of the following nonparametric tests: chi-square test; two-tailed test; and Fisher exact test. Quantitative variables were characterized as mean $\pm$ standard deviation, and comparisons performed by use of non-paired *t* test. The factors independently associated with stress hyperglycemia were identified by use of univariate logistic regression, followed by multivariate logistic regression, which included the variables with $p \leq 0.20$ identified in the former. The variables were adjusted by use of backward logistic regression. The significance level adopted was $p < 0.05$. Statistical analyses were performed by using the Statistical Package for the Social Sciences software, version 17.0 (SPSS Inc., Chicago, Illinois, USA).

Ethical aspects

The ethical principles for human experimentation were carefully followed. All patients involved in this study were instructed about the objectives of the Registry of Acute Coronary Syndrome of the Hospital São Lucas (Solar) and provided written informed consent. The extension of the research project "*Registro de Síndrome Coronariana*

Aguda SOLAR", previously approved, was submitted to the Committee on Ethics and Research of the Universidade Federal de Sergipe.

Results

The study included 152 patients [90 (59.2%) men; mean age, 63.3 ± 13.8 years]. The group with admission hyperglycemia comprised 67 patients (44.1%).

Table 1 shows the characteristics of the population studied. The groups differed significantly only regarding age and sex. Group I had a higher mean age (65.9 ± 13.5 years *vs.* 61.2 ± 13.8 years, p = 0.04) and fewer male patients (47.8% *vs.* 68.2%, p = 0.011). Both groups were similar regarding the distribution of risk factors for ACS, smoking habit, family history of CAD, sedentary lifestyle, DLP, DM, SAH, CHF, arrhythmia, DVT, and CVA. The same occurred regarding the previous use of medication, such as anti-hypertensive drugs, antidiabetic drugs, nitrates, digitalis, lipid-lowering drugs, and benzodiazepines.

The parameters assessed on admission, such as HR, SBP, DBP, urea, creatinine and Killip classification were similar between the groups.

After diagnostic confirmation, the groups were similar regarding the clinical presentation of ACS.

Regarding mean ejection fraction calculated on echocardiography, both groups showed no difference. Mean ejection fractions were 57.4 ± 12.9% and 57.1 ± 13.5% for groups I and II, respectively (p = 0.881).

On coronary angiography, the arterial pattern did not significantly differ between groups (p = 0.161).

The presence of admission hyperglycemia did not interfere with the type of treatment performed in patients with ACS. Clinical treatment was performed in 27 (40.3%) group I patients and 23 (27.1%) group II patients. Thrombolytic agents were not used. Angioplasty was chosen for 38 (56.7%) group I patients and 54 (63.5%) group II patients. Coronary artery bypass surgery was performed in eight (9.4%) group II patients and in three (4.5%) group I patients (p = 0.098). [rever com autor se é isso mesmo: mais pacientes operados no grupo sem hiperglicemia?]

In-hospital complications were observed in 35 (23%) patients (table 2) as follows: 24 group I patients (35.8%) as compared to only 11 group II patients (12.9%) (p = 0.001). Patients with hyperglycemia had more cardiac complications [25.4% *vs.* 11.8%, p = 0.029; CPA (11.9% *vs.* 2.4%, p = 0.018)], and noncardiac complications [22.4% *vs.* 2.4%, p < 0.001; infectious processes (17.9% *vs.* 2.4%, p = 0.001)].

Mortality was similar in both groups, and total mortality in the study was 4.6%. The following variables were also similar in both groups: in-hospital APE; CVA; shock; post-infarction arrhythmia; ischemic complications; and respiratory failure of noncardiac origin.

The mean length of hospital stay in group I patients was 8.3 ± 10.2 days, and in group II patients, 7.2 ± 5.7 days (p = 0.403).

On univariate analysis (table 3), the variables associated with stress hyperglycemia were in-hospital complications, age, and female sex. There was no association with the ACS clinical presentation (ACS with and without ST-segment elevation). On multivariate analysis, only female sex (OR = 2.04; 95% CI: 1.03 – 4.06; p = 0.007) and in-hospital complications (OR = 3.65; 95% CI: 1.62 – 8.19; p = 0.002) associated independently with stress hyperglycemia on admission.

The mean blood glucose concentration of patients with complications was 220 ± 99.3 mg/dL, while that of the others was 151 ± 64.9 mg/dL (p < 0.01). Chart 1 shows the mean and confidence interval of admission blood glucose concentration of diabetic and nondiabetic patients with and without in-hospital complications. Among diabetic patients, the mean blood glucose level of those with in-hospital complications was 277 ± 86.1 mg/dL, while that of patients with no in-hospital complication was 199 ± 72.3 mg/dL (p < 0.01). However, among nondiabetic patients, the mean blood glucose level of those with in-hospital complications was 152 ± 85.4 mg/dL, while that of those with no in-hospital complication was 124 ± 40.9 mg/dL (p = 0.12).

Discussion

Stress hyperglycemia as a factor of worse outcome has been discussed on different studies, which have shown a significant increase in mortality during the hospitalization and after the hospital discharge of patients with acute cardiovascular events; however, no minimum blood glucose concentration that could carry that risk has been established[19-21]. In our study, approximately 44% of the sample had admission hyperglycemia. According to Gois[12], in the literature, its prevalence ranges from 25% to 50%. Such large variation might be due to different cutoff points for admission hyperglycemia in different studies, since a consensus about that value has not been agreed upon. Chart 1 shows that, for both nondiabetic and diabetic patients, the mean blood glucose concentration of those with complications was higher than that of patients with an uneventful evolution. This allows us to assume that blood glucose concentration might be related to poor prognosis. Similarly, Duarte et al[22], studying patients with ACS and complications, have found higher mean blood glucose levels, which were significantly associated with in-hospital events.

Regarding blood glucose concentrations, Capes et al[13] have reported that nondiabetic patients with ACS are at risk for in-hospital complications for blood glucose levels over 110 mg / dL, while diabetic patients with ACS are at risk for in-hospital complications for admission blood glucose concentrations equal to or over 180 mg/dL. Timmer et al.[14] have considered blood glucose levels over 140 mg / mL as stress hyperglycemia for nondiabetic patients, and have shown that the increase in mortality was not limited to patients with preexisting diabetes. In the HI-5 study, six-month mortality was higher among patients with AMI who maintained mean blood glucose concentrations over 144 mg/dL[15].

Unlike other studies, an association between mortality and stress hyperglycemia was not observed in ours, and that might have been due to the small number of events. Cardiopulmonary arrest, however, showed an association with elevated blood glucose concentrations, probably because of its higher number of events (p = 0.018). Rocha et al[23] have suggested that an elevated admission blood glucose level is

Table 1 – Characteristics of the patients

Variables	Group I n=67 (44,1%)	Group II n=85 (55,9%)	Total n=152	p
Age: (years)	65.9 ± 13.5	61.2 ± 13.8	63.3 ± 13.8	0.04
Sex:				
Male	32 (47.8%)	58 (68.2%)	90 (59.2%)	
Female	35 (52.2%)	27 (31.8%)	62 (40.8%)	0.011
Antecedents:				
Smoking habit: Current smoker	9 (13.6%)	15 (17.6%)	24 (15.9%)	
Ex-smoker	17 (25.8%)	25 (29.4%)	42 (27.8%)	0.624
Non-smoker	40 (60.6%)	45 (52.9%)	85 (56.3%)	
Family history of CAD	19 (28.8%)	29 (34.1%)	48 (31.8%)	0.485
Previous CAD	24 (36.4%)	36 (42.4%)	60 (39.7%)	0.456
Sedentary lifestyle	52 (78.8%)	63 (74.1%)	115 (76.2%)	0.504
Dyslipidemia	43 (65.2%)	51 (60.0%)	94 (62.3%)	0.517
Diabetes mellitus	32 (47.8%)	29 (34.1%)	61 (40.1%)	0.088
SAH	49 (73.1%)	58 (68.2%)	107 (70.4%)	0.511
Previous CHF	7 (10.6%)	14 (4.7%)	21 (7.3%)	0.166
Previous arrhythmia	5 (7.6%)	9 (10.6%)	14 (10.6%)	0.527
Previous DVT	3 (4.5%)	3 (3.5%)	6 (4.0%)	0.751
Previous CVA	8 (12.1%)	6 (7.1%)	14 (9.3%)	0.287
Medications used:				
Antidiabetic drugs	32 (47.8%)	29 (34.1%)	61 (40.1%)	0.088
Antiplatelet drugs	24 (36.4%)	35 (41.7%)	59 (39.3%)	0.509
Anti-hypertensive drugs	42 (64.6%)	58 (68.2%)	100 (66.7%)	0.641
Beta-blocker	16 (24.2%)	23 (27.1%)	39 (25.8%)	0.695
ACEI	26 (24.2%)	14 (16.5%)	30 (19.9%)	0.235
ARB	13 (19.7%)	15 (17.9%)	28 (18.7%)	0.774
CCB	7 (10.6%)	8 (9.4%)	15(9.9%)	0.808
Diuretics	7 (10.6%)	11 (13.1%)	18 (12.0%)	0.641
Alpha-blocker	2 (1.3%)	2 (1.3%)	4 (2.7%)	0.806
Nitrate	12 (18.2%)	9 (10.7%)	21 (14.0%)	0.191
Lipid-lowering drugs	21 (31.8%)	22 (26.2%)	43 (28.7%)	0.449
Digitalis	2 (3.0%)	2 (2.4%)	4 (2.7%)	0.806
Benzodiazepines	6 (9.1%)	15 (17.9%)	21 (14.0%)	0.125
HR (bpm):	81.6 ± 18	78.6 ± 19.5	79.9 ±18.9	0.378
SBP (mm Hg):	162 ± 34.4	150 ± 32	154.8 ± 33.4	0.057
DBP (mm Hg):	93.3 ± 17.3	91.7 ± 19.6	92.3 ± 18.6	0.654
Urea: (mg/dL)	40.2 ±20.3	38.96 ± 25	39.5 ± 23	0.743
Creatinine: (mg/dL)	1.04 ± 0.59	1.15 ± 1.24	1.1 ± 1.01	0.512
Killip:				
I - II (low)	61 (91.0%)	82 (96.5%)	143(94%)	
III - IV (high)	6 (9.0%)	3 (3.5%)	9 (5.9%)	0.159
Clinical presentation:				
ACS with STE	19 (28.4%)	21 (25.%)	40 (26.5%)	
ACS without STE	48 (71.6%)	63 (75.0%)	111 (73.5%)	0.642
Ejection fraction	57.4 ± 12.9	57.1 ± 13.5		0.881
Classification on coronary angiography:				
No lesion or < 50%	8 (12.5%)	8 (10.3%)	16 (11.3%)	
Single-vessel disease	19 (29.7%)	26 (33.3%)	45 (31.7%)	
Two-vessel disease	18 (28.1%)	20 (25.6%)	38 (26.8%)	0.161
Three-vessel disease	18 (28.1%)	15 (19.2%)	33 (23.2%)	
Left main coronary artery lesion	1 (1.6%)	9 (11.5%)	7 (7.0%)	
Treatment:				
Only medication	27 (40.3%)	23 (27.1%)	50 (32.9%)	
Thrombolytic drug	0 (0%)	0 (0%)	0 (0%)	
Angioplasty	38 (56.7%)	54 (63.5%)	92 (60.5%)	0.098
CABG	3 (4.5%)	8 (9.4%)	10 (6.6%)	

CAD: coronary artery disease; SAH: systemic arterial hypertension; CHF: congestive heart failure; DVT: deep venous thrombosis; CVA: cerebrovascular accident; ACEI: angiotensin-converting-enzyme inhibitor; ARB: angiotensin II receptor blocker; CCB: calcium-channel blocker; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; ACS: acute coronary syndrome; STE: ST-segment elevation; CABG: coronary artery bypass grafting.

Table 2 – In-hospital outcome

Variables	Group I n = 67 (44.1%)	Group Ii n = 85 (55.9%)	Total n = 152	p
In-hospital complications	24 (35.8%)	11 (12.9%)	35 (23%)	0.001
Cardiac complications:	17 (25.4%)	10 (11.8%)	27 (17.8%)	0.029
APE	7 (10.4%)	3 (3.5%)	10 (6.6%)	0.09
In-hospital CVA	2 (3.0%)	0 (0%)	2(1.3%)	0.109
Shock	5 (7.5%)	2 (2.4%)	7 (4.6%)	0.136
Post-infarction arrhythmia	4 (6.0%)	4 (4.7%)	8 (5.3%)	0.728
CPA	8 (11.9%)	2 (2.4%)	10 (6.6%)	0.018
Death	5(7.5 %)	2(2.4 %)	7 (4.6%)	0.136
Ischemic complications:	5 (7.5%)	5 (5.9%)	10 (6.6%)	0.696
Angina	3 (4.5%)	4 (4.7%)	7 (4.6%)	0.947
Reinfarction	1 (1.5%)	0 (0%)	1 (0.7%)	0.258
Infarction	1 (1.5%)	0 (0%)	1 (0.7%)	0.258
Reintervention	2 (3.0%)	2 (2.4%)	4 (2.6%)	0.809
Noncardiac complications:	15 (22.4%)	2 (2.4%)	17 (11.2%)	0.0001
Infection	12 (17.9%)	2 (2.4%)	14 (9.2%)	0.001
Respiratory failure	6 (9.0%)	2 (2.4%)	8 (5.3%)	0.07
Length of hospital stay (days)	8.3 ± 10.2	7.2 ± 5.7	7.7 ± 8	0.403

APE: acute pulmonary edema; CVA: cerebrovascular accident; CPA: cardiopulmonar arrest.

Table 3 – Odds ratio for the factors associated with stress hyperglycemia

Variables	Rough odds	95% CI	p	Adjusted odds	95% CI	p
In-hospital complications	3.75	1.67-8.41	0.001	3.65	1.62-8.19	0.002
Age	1.03	1.001-1.05	0.04	-	-	-
Sex						
Male	Reference	-	-	Reference	-	-
Female	2.3	1.21-4.55	0.01	2.04	1.03-4.06	0.007
Clinical presentation						
ACS without STE	1.19	0.58-2.46	0.64	-	-	-
ACS with STE	Reference	-	-	-	-	-

**Odds ratio adjusted for in-hospital complications, age, sex, and clinical presentation; ACS: acute coronary syndrome; STE: ST-segment elevation.*

an independent predictive factor of in-hospital death among nondiabetic patients admitted due to ACS.

On multivariate analysis, stress hyperglycemia showed an independent association with female sex and in-hospital complications. Such results are in accordance with those of the literature, indicating an association of hyperglycemia with female sex and in-hospital mortality[11,23].

Barsheshet et al[24] have shown an association of admission hyperglycemia with in-hospital mortality and mortality up to 60 days after hospital discharge among nondiabetic patients after ACS. In the study by Stranders et al[25], admission hyperglycemia was an independent predictor of death within two and half years among diabetic and nondiabetic patients after a coronary artery event.

An indirect evidence of the deleterious effect of stress hyperglycemia on AMI outcomes has been reported in studies finding a reduction in the mortality of diabetic[26] or critically ill[27] patients by use of strict glycemic control with insulin administration during hospitalization[28-30]. Despite the recommendations for strict glycemic control in all hospitalized patients[31], blood glucose concentrations are not included in AMI risk scores[32], and the consensuses on ACS have suggested neither diagnostic nor therapeutic strategies for that purpose. That might also occur in nondiabetic patients; however, randomized studies are necessary to establish the usefulness of intensive care directed to glycemic control in nondiabetic patients[23].

The exact mechanism through which hyperglycemia worsens the prognosis of ischemic patients has not been

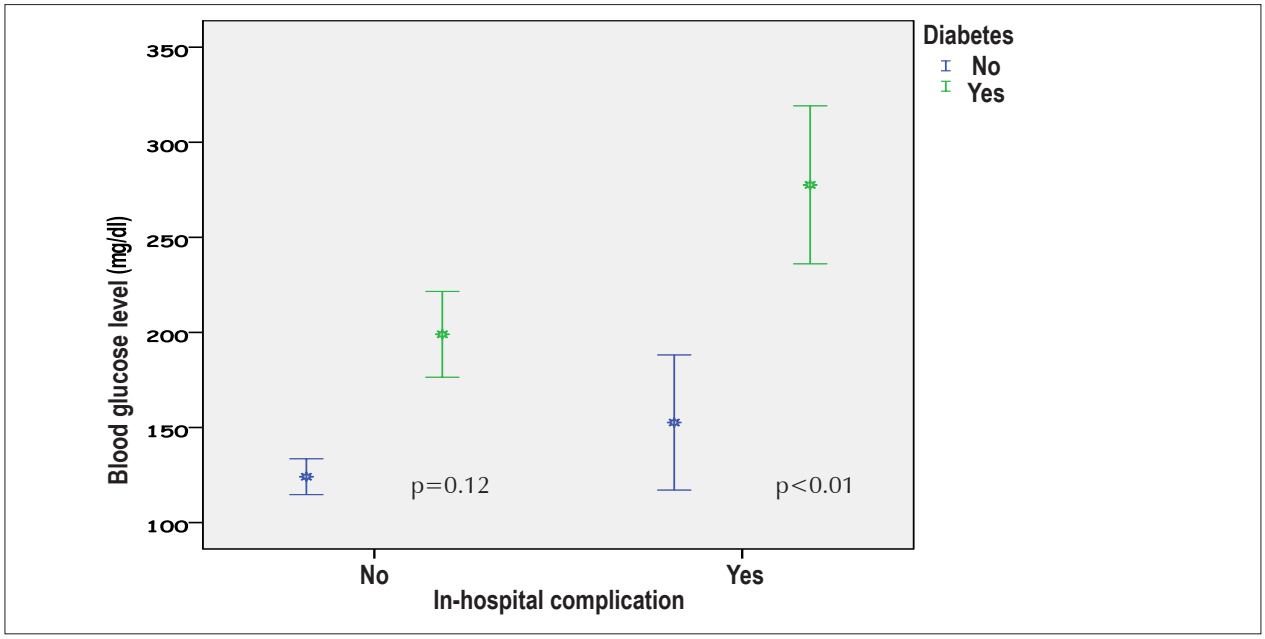

Chart 1 - *Mean blood glucose level in diabetic and nondiabetic patients with and without in-hospital complication.*

well established. Its pathophysiology is believed to be based on endothelial and microvascular dysfunction, causing a prothrombotic state produced by vascular inflammation. The endothelial dysfunction inactivates nitric oxide and increases oxidative stress, responsible for the production of oxygen reactive species[10]. The production of those radicals activates transcription and growth factors and secondary mediators. Through direct tissue lesion or activation of those secondary mediators, hyperglycemia-induced oxidative stress causes additional lesion to myocytes[10,33]. There is evidence that the prothrombotic state generated by hyperglycemia originates from reduced plasma fibrinolytic activity and action of tissue plasminogen activator[34].

Several studies, however, have suggested the following secondary mechanisms that lead to an increase in oxygen consumption and aggravation of ischemia: decreased collateral circulation and consequent increase in the infarcted area; ischemic preconditioning and apoptosis promotion; elevation in catecholamines[11]; platelet dysfunction; blood pressure elevation and QT interval prolongation; and increase in free fat acids, potential inducers of cardiac arrhythmias and insulin resistance[10,12].

Our study has some limitations. One is its retrospective nature and the inherent impossibility of eliminating confounding factors. However, the association of hyperglycemia and in-hospital complications persists after adjusting for several baseline characteristics of the sample. Another limitation is the fact that the patients' admission blood glucose concentration was measured without knowing the patients' prandial status. In addition, because our study excluded capillary blood glucose testing, a measurement usually performed in more severely ill patients, our sample might be comprised by less severely ill patients. This might have weakened the association found between hyperglycemia and complications.

Conclusion

In conclusion, in our study, admission hyperglycemia is an independent predictive factor of in-hospital complications after ACS in both diabetic and nondiabetic patients. Those results emphasize the need to assess admission blood glucose concentration in all patients, including nondiabetic ones, admitted due to ACS, aiming at identifying individuals at greater risk for complications. Further studies are required to clarify whether elevated admission blood glucose levels are only markers of poor prognosis or whether they contribute to worsen ACS.

Author contributions

Conception and design of the research: Pinheiro CP, Barreto-Filho JAS, Oliveira JLM, Sousa ACS; Acquisition of data: Pinheiro CP, Oliveira MDP, Faro GBA, Silva EC, Rocha EAA; Analysis and interpretation of the data: Pinheiro CP, Oliveira MDP, Faro GBA, Silva EC, Barreto-Filho JAS, Oliveira JLM, Sousa ACS; Statistical analysis: Pinheiro CP, Oliveira MDP, Faro GBA; Writing of the manuscript: Pinheiro CP, Faro GBA, Silva EC, Sousa ACS; Critical revision of the manuscript for intellectual content: Pinheiro CP, Faro GBA, Silva EC, Rocha EAA, Barreto-Filho JAS, Oliveira JLM, Sousa ACS

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any post-graduation program.

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