Newly Diagnosed Diabetes and Stress Glycaemia and Its’ Association with Acute Coronary Syndrome

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Abstract

BACKGROUND: Diabetes is diagnosed in 10-20% of patients with acute coronary syndrome (ACS) not known to be diabetics. Elevated blood glucose is an independent risk factor for cardiac events, regardless of presence of diabetes.

AIM: Evaluating the prevalence of new-diagnosed DM among patients with ACS, and assessing the relationship between stress glycaemia and new diagnosed DM with in-hospital cardiac events.

METHODS: Prospective observational study, in patients with ACS, in whom we analyzed parameters of glycemic metabolism, clinical data, and in-hospital cardiac events. We comparatively analyzed patients according to the HgbA1C and known DM in five groups: non-DM (< 5.6%), new pre-DM (5.6-6.5%), new DM (≥ 6.5%), controlled (<7%) and uncontrolled (≥7%) known DM.

RESULTS: 150 patients, (93 male and 57 female) were included. Impaired glucose metabolism was detected in 44.5% of patients, 7.9% of whom were newly-diagnosed DM. The highest levels of stress glycaemia were found in new and uncontrolled known DM. The in-hospital event rate was 20.7%, the mortality rate 7.3%, being the highest in new diagnosed and uncontrolled known DM patients.

CONCLUSIONS: The prevalence of unknown DM was high among patients with ACS. Stress glycaemia and failure to achieve glycemic control were independent predictors of in-hospital cardiac events.

Introduction

Diabetes mellitus is increasing on a global level with an estimated prevalence around 12-14%. What is even more important, it is also estimated that one in every four hospitalized patients has known diabetes. Hyperglycemia in hospitalized patients is even more frequent [1].

The Study of Abdullatef et al. performed on Qatar population with acute coronary syndrome (ACS), refers that 45% of hospitalized patients without known DM were either with prediabetes, diabetes or stress hyperglycemia [2]. There are three possible causes for hyperglycemia in hospitalized patients: existing known diabetes, existing but unknown diabetes and stress hyperglycemia. Stress hyperglycemia is defined by ADA (American Diabetes Association) as an elevation of fasting glucose ≥ 7 mmol/L, or 2-hour postprandial glucose ≥ 11 mmol/L, in a patient without evidence of previous diabetes. Glycosylated hemoglobin (HbA1c) value has been recommended to distinguish between patients with stress hyperglycemia and those with previously undiagnosed diabetes. HbA1c value ≥ 6.5% indicates pre-existing unrecognized diabetes, whereas HbA1c value < 6.5% indicates stress-induced hyperglycemia. The prevalence of stress hyperglycemia in critically ill patients varies between 30-40%, 10-15% of which have previously unrecognized diabetes. (3) Gornik et al. reported a prevalence of unrecognized diabetes among critically ill patients of 17% [4]. In the population of elderly patients hospitalized due to heart failure the prevalence of hyperglycemia was 44%, and 41% in patients with acute coronary syndrome [5].

Stress hyperglycemia has several means. Stress conditions such as surgery, trauma and acute illness increase the circulatory level of counter regulatory hormones (glucagon, cortisol, catecholamines) and pro-inflammatory cytokines and...
they alter the effect of insulin on the hep and on the skeletal muscle by increasing of the hepatic production of glucose and decreasing the peripheral utilization of glucose. Pro-inflammatory cytokines also increase the hepatic release of glucose and increase the insulin resistance in the hep and in the skeletal muscle. Stress hyperglycemia in patients with diabetes type 2 involves a combination of insulin resistance and beta cell secretory defect [3].

The impact of hyperglycemia

Patients with stress hyperglycemia have a higher mortality rate and longer hospitalization time in comparison with patients with known diabetes and with normoglycaemia. They have worse outcome in comparison with diabetic patients with a comparable degree of hyperglycemia. It depends on the underlying diagnose, risk of infection etc. Non-diabetic patients with stress hyperglycemia have 3.9 fold higher risk of death after myocardial infarction in comparison with normoglycaemic non-DM patients. The same finding is also evident in patients with stroke. Worse clinical status is evident in non-diabetic patients with stress hyperglycemia in comparison with diabetic patients [3, 6, 7].

The aim of this study was to evaluate the prevalence of new-diagnosed DM among patients with ACS, and assessing the relationship between stress glycaemia and new diagnosed DM with in-hospital cardiac events.

Material and Methods

This was a prospective observational study. Patients admitted to ICCU and treated for acute coronary syndrome-ACS (unstable angina-APNS, NSTEMI-myocardial infarction without ST-segment elevation and STEMI-myocardial infarction with ST-segment elevation), were enrolled. All patients with confirmed ACS during the two month period were included. We analyzed glycemc parameters: blood glycaemia at admission (stress glycaemia), fasting plasma glycaemia the first morning after admission, glycaemia levels during the hospital treatment and HgbA1C.

Demographic, clinical, left ventricular functional and angiographic data were obtained for all 150 patients (pts.). We analyzed: body presence of risk factors and co-morbidities, basic biochemical variables (Hgb, BUN, creatinine, Na, K), lipid profile (Tg, HDL, LDL Hol, Ipa), LV systolic and diastolic function, SINTAX score, TIMI flow before and after PCI procedure, duration of hospitalization (days) and in-hospital morbidity/ mortality: heart failure, malignant arrhythmias, early ischemic events, bleeding complications (CE) and cardiac death (CD).

We used ADA (American Diabetes Association) 2015 Guidelines criteria for the diabetes definition (fasting plasma glucose (FPG) >7 mmol/L, or random plasma glucose (RPG) >11.1 mmol/L, or HgbA1C >6.5%), and HgbA1C >5.6% for the definition of pre-diabetes; for the definition of stress hyperglycemia: an elevation of FPG ≥7 mmol/L, or RPG ≥11 mmol/L in a patient without evidence of previous diabetes. We used glycosylated hemoglobin (HbA1c) value to distinguish between patients with stress hyperglycemia and those with previously undiagnosed diabetes (an HbA1c value ≥ 6.5% indicated pre-existing unrecognized diabetes, whereas HbA1c value < 6.5% indicated stress-induced hyperglycemia). Also, we used the ADA recommendations for controlled diabetes (HgbA1C <7%), to distinguish between diabetic patients with controlled and uncontrolled diabetes. We also used the ADA glycemic target for critically ill patients (6.1-10 mmol/L). If a patient was in this range during hospitalization we defined that as a good glycemic control as opposite to those patients in whom we failed to achieve this target.

We performed several comparative analyses. We compared diabetic versus non diabetic patients. We also compared patients with good glycemic control versus uncontrolled patients. Based on HgbA1C and prediagnosed diabetes we subdivided the patients in five groups: three groups without known diabetes: non-diabetic (<5.6%), pre-diabetic (5.6-6.5%), newly diagnosed diabetic (≥6.5%), and two groups of pts. with known diabetes: controlled (<7%), and uncontrolled (≥7%). Cardiac event rate was analyzed as a function of these glycemic variables.

Statistical analysis

Descriptive and comparative statistics for continuous variables with t-test (and non-parametric test for small samples), Chi square test for categorical variables (Pearson Chi square) and Fisher exact test for 2x2 tables and Odds Ratio (with Mantel-Haenszel common odds ratio), uni and multivariate logistic regression analysis for identifying the predicting variables and obtaining the ROC curves. Significance was determined at 0.05.

Results

The study population consisted of 150 patients, 93 males and 57 females (overall mean age 62.9 ± 12.3 years) According to their HgbA1C level,
glycemic control and known diabetes patients were divided in five groups: Group 0: non-diabetic patients (37.3%); Group 1: newly diagnosed pre-diabetes (24.7%); Group 2: newly diagnosed diabetes (5.3%); Group 3: known diabetes good controlled (14.0%); and Group 4: known diabetes uncontrolled (18.7%).

We found that no significant differences in LV function or in CAD distribution or PCI outcome. The only significant difference in the biochemical parameters between genders was found for Hgb which was significantly lower in females (p = 0.000), but in the normal range.

When we made a comparison of these same variables between patients with and without DM, we found that no DM patients had OR of 2.1 (CI 1.16-2.40; p = 0.002) for smoking. But, there were no significant differences in LV function or in CAD distribution or PCI outcome. DM pts had significantly higher Tg levels (p = 0.041) and variables of glycemic control: HgbA1C and stress glycaemia (p = 0.000 for both variables).

The baseline characteristics of the patients as a function of the glycemic metabolism revealed that newly diagnosed diabetes was far more frequent in females (75%), both groups being significantly older. Based on HbgA1C levels we identified 45 out of 101 patients (44.5%) without known DM to be diabetic (7.9%) or prediabetic (36.6%). Mean HbgA1C levels were high in newly diagnosed DM, but even higher in uncontrolled DM pts. The same was for stress glycaemia levels. The highest levels were in new DM and uncontrolled known DM, as compared with controlled known DM (p = 0.026 and p = 0.001 respectively).

### Table 1: Baseline characteristics of ACS patients, and according to gender

| Variable       | Total (N) | Males (M) | Females (F) | Significance (p) |
|----------------|-----------|-----------|-------------|------------------|
| Gender         |           |           |             |                  |
| Male           | 145       | 86 (59)   | 59 (41)     |                  |
| Female         | 292       | 146 (49)  | 146 (51)    |                  |
| Age            |           |           |             |                  |
| Mean ± SD      | 60.3 ± 13.2 | 62.3 ± 13.9 | 58.9 ± 12.7 |                  |
| Median          | 63 (13)   | 65 (13)   | 61 (13)     |                  |
| HgbA1C (mmol/L) |           |           |             |                  |
| Mean ± SD      | 47.9 ± 6.3 | 47.4 ± 6.2 | 48.2 ± 6.4  |                  |
| Median          | 47 (9)    | 47 (9)    | 48 (9)      |                  |
| Hgb (g/L)      |           |           |             |                  |
| Mean ± SD      | 145 ± 20  | 143 ± 20  | 146 ± 20    |                  |
| Median          | 147 (11)  | 147 (11)  | 147 (11)    |                  |
| LDL Chol (mmol/L) |           |           |             |                  |
| Mean ± SD      | 3.4 ± 1.5 | 3.3 ± 1.5 | 3.5 ± 1.6   |                  |
| Median          | 3.5 (1)   | 3.5 (1)   | 3.5 (1)     |                  |
| Tg (mmol/L)    |           |           |             |                  |
| Mean ± SD      | 1.7 ± 0.8 | 1.7 ± 0.8 | 1.7 ± 0.8   |                  |
| Median          | 1.7 (1)   | 1.7 (1)   | 1.7 (1)     |                  |
| BMI (kg/m²)    |           |           |             |                  |
| Mean ± SD      | 25.0 ± 3.5 | 25.0 ± 3.5 | 25.0 ± 3.5  |                  |
| Median          | 25.0 (3)  | 25.0 (3)  | 25.0 (3)    |                  |
| Blood pressure |           |           |             |                  |
| SBP (mmHg)     |           |           |             |                  |
| Mean ± SD      | 120 ± 12  | 120 ± 12  | 120 ± 12    |                  |
| Median          | 120 (12)  | 120 (12)  | 120 (12)    |                  |
| HR (bpm)       |           |           |             |                  |
| Mean ± SD      | 72 ± 12   | 72 ± 12   | 72 ± 12     |                  |
| Median          | 72 (12)   | 72 (12)   | 72 (12)     |                  |
| Smoking        |           |           |             |                  |
| Never          | 145       | 79 (55)   | 66 (45)     |                  |
| Former         | 149       | 71 (48)   | 78 (52)     |                  |
| Current        | 149       | 73 (50)   | 76 (50)     |                  |
| Diabetes       |           |           |             |                  |
| Yes            | 292       | 148 (50)  | 144 (50)    |                  |
| No             | 145       | 79 (55)   | 66 (45)     |                  |
| Hypertension   |           |           |             |                  |
| Yes            | 292       | 148 (50)  | 144 (50)    |                  |
| No             | 145       | 79 (55)   | 66 (45)     |                  |
| Stressful       |           |           |             |                  |
| Yes            | 292       | 148 (50)  | 144 (50)    |                  |
| No             | 145       | 79 (55)   | 66 (45)     |                  |

Smoking was the only risk factor that significantly differed between groups. The smallest proportion of smokers was among pts with controlled DM. Newly diagnosed DM had the worst biochemical parameters: low Hgb (p < 0.05 in comparison to all groups), high BUN and creatinine as renal function parameters. But there were no significant differences in the Lp fractions. No significant difference was found for LV function, as opposite to CAD distribution, which was found to be the worst in newly diagnosed DM pts who had worst TIMI flow before treatment, but no intergroup differences were found after the PCI.
procedure. The mean hospitalization time was 4.5 ± 3.0 days with the longest duration in pts with newly diagnosed DM (p = 0.035). The event rate was 20.7% during the hospital treatment with in-hospital mortality rate of 7.3%.

Table 2: Baseline characteristics of ACS patients in accordance with the presence of known DM

| Variable | All pts | DM pts | NDM pts | Sig (p) |
|----------|---------|--------|---------|---------|
| Risk factors | | | | |
| HTA | 85 (56.7%) | 32 (65.3%) | 53 (52.5%) | ns (OR for HTA in DM pts: 1.7; CI: 0.84-3.4; p=0.18) |
| HLP | | | | |
| Smoking history | 22 (14.7%) | 17 (34.7%) | 5 (5.0%) | ns |
| Smoking (current smokers) | 89 (59.3%) | 20 (40.4%) | 69 (68.3%) | 0.001 (OR for NDM smokers is 2.1; CI 1.5-3.4; p<0.002) |
| LV function | | | | |
| EF (%C) | 51 ± 61 | 51 ± 67 | 51 ± 48 | 0.826 (ns) |
| LV systolic function | | | | |
| Normal (EF ≥50%) | 60 (40.3%) | 25 (51.0%) | 35 (34.7%) | 0.247 (ns) |
| Reduced (EF <50%) | 59 (39.3%) | 23 (46.9%) | 36 (35.6%) | |
| Severe reduced (<30%) | 8 (5.5%) | 1 (2.1%) | 7 (7.1%) | |
| LV diastolic dysfunction | 60 (43.3%) | 21 (42.8%) | 39 (38.5%) | 0.538 (ns) |
| Syntax score | 16 ± 8.8 | 16 ± 8.5 | 15 ± 7.9 | 0.365 (ns) |
| Syntax score before treatment | | | | |
| 0 | 63 (47.7%) | 23 (55.2%) | 40 (40.4%) | 0.562 (ns) |
| 1 | 7 (5.3%) | 2 (4.4%) | 5 (5.1%) | |
| 2 | 1 (1.4%) | 0 (0%) | 1 (1.1%) | |
| 3 | 50 (41.1%) | 17 (37.4%) | 33 (34.3%) | |
| Syntax score after treatment | | | | |
| 0 | 8 (6.5%) | 5 (11.1%) | 3 (3.4%) | 0.162 (ns) |
| 1 | 3 (2.9%) | 1 (2.1%) | 2 (2.3%) | |
| 2 | 1 (1.4%) | 0 (0%) | 1 (1.1%) | |
| 3 | 120 (90.4%) | 39 (84.4%) | 81 (84.4%) | |
| Blood analyses | | | | |
| Hgb | 14 ± 1.8 | 14 ± 1.6 | 14 ± 1.9 | 0.678 (ns) |
| BUN | 16 ± 4.5 | 7 ± 4.6 | 6 ± 4.5 | 0.455 (ns) |
| Creatinine | 95 ± 37 | 105 ± 38 | 91 ± 38 | 0.280 (ns) |
| Na | 135 ± 4.7 | 135 ± 4.4 | 133 ± 4.6 | 0.000 |
| K | 4 ± 0.6 | 4 ± 1.0 | 4 ± 0.6 | 0.098 (ns) |
| Lipid profile | | | | |
| Total cholesterol | 5 ± 1.8 | 5 ± 1.9 | 5 ± 1.9 | 0.084 |
| HDL cholesterol | 1 ± 0.2 | 1 ± 0.2 | 1 ± 0.2 | 0.488 (ns) |
| LDL cholesterol | 3 ± 1.2 | 3 ± 1.1 | 3 ± 1.2 | 0.615 (ns) |
| Triglycerides (mg/dl) | 3 ± 1.2 | 3 ± 1.1 | 3 ± 1.2 | 0.615 (ns) |
| Syntax score before treatment | | | | |
| 0 | 63 (47.7%) | 23 (55.2%) | 40 (40.4%) | 0.562 (ns) |
| 1 | 7 (5.3%) | 2 (4.4%) | 5 (5.0%) | |
| 2 | 1 (1.5%) | 0 (0%) | 1 (1.0%) | |
| 3 | 50 (41.1%) | 17 (37.4%) | 33 (34.3%) | |
| Syntax score after treatment | | | | |
| 0 | 8 (6.5%) | 5 (11.1%) | 3 (3.4%) | 0.162 (ns) |
| 1 | 3 (2.9%) | 1 (2.1%) | 2 (2.3%) | |
| 2 | 1 (1.4%) | 0 (0%) | 1 (1.1%) | |
| 3 | 120 (90.4%) | 39 (84.4%) | 81 (84.4%) | |

Although the number of patients in each group was small and the groups were unequal, the highest death rate was registered among patients with newly diagnosed and uncontrolled known DM. When we analyzed the duration of hospital treatment as a function of in-hospital morbidity and mortality we registered a significant difference (4.2 ± 2.5 for pts without, versus 5.7 ± 4.2 for pts with, p = 0.009), as shown in Figure 2.

Figure 2: Length of hospitalization as a function of in-hospital morbidity

Table 3: Baseline characteristics of ACS patients according to glycemic metabolism

| Variable | HgbA1C (%) | 1/2 Years of DM | 3-4 Years of DM | 5 Years of DM | DM good controlled | Known DM uncontrolled | Sig (p) |
|----------|------------|----------------|----------------|--------------|-------------------|----------------------|---------|
| HgbA1C1 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C2 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C3 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C4 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C5 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C6 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C7 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C8 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C9 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |
| HgbA1C10 | 50 (0.0) | 70 (0.0) | 80 (0.0) | 90 (0.0) | 50 (0.0) | 70 (0.0) | 90 (0.0) |

Aiming to identify predictors of cardiac events (CE) and cardiac death (CD), and to define the role of glycemic variability among CE predictors, we performed a univariate logistic binary regression analysis using the variables found as predictors in the univariate analysis. Variables included in the prediction model were: age, cigarette smoking, EF as a continuous and a categorical variable (three categories: 0-normal, 1-mildly/moderately reduced; 2-severely reduced), TIMI flow and Syntax score, Hgb, BUN, creatinine, stress glycaemia, glycerolysis, and HDL cholesterol. We

In an attempt to identify independent predictors of cardiac events we performed a multivariate logistic variable selection analysis using the variables found as predictors in the univariate analysis. Variables included in the prediction model were: age, cigarette smoking, EF as a continuous and a categorical variable (three categories: 0-normal, 1-mildly/moderately reduced; 2-severely reduced), TIMI flow and Syntax score, Hgb, BUN, creatinine, stress glycaemia, glycerolysis, and HDL cholesterol. We
applied a backward stepwise conditional model with Chi square 38.675; sig 0.001, correct prediction 88.6%, and at step 10 we identified four independent predictors: smoking (Exp(B) 5.945; CI 1.79-19.66); p = 0.004), EF (%) (beta -0.089; p = 0.007), HDL chl (-3.179; p = 0.016), and glycoregulation (yes) (Exp(B) 0.324 (CI 0.08-1.19; p = 0.060).

Table 4: Univariate predictors of in-hospital cardiac events (binary logistic regression)

| Variable                | Chi square | sig | Wald     | Exp (B) | sig |
|-------------------------|------------|-----|----------|---------|-----|
| Age                     | 6.819      | 0.039 | Beta.045 | 0.012   |
| Smoking                 | 4.825      | 0.028 | 4.742    | 0.409   | 0.029 |
| Stress glycaemia        | 9.574      | 0.002 | Beta.087 | 0.004   |
| HgbA1C                  | 8.596      | 0.072 | 3.114    | 0.330   | 0.078 |
| Group 2                 |            |      |          |         |
| Group 4                 | 7.496      | 0.006 | Beta.1.161 | 0.006  |
| Glycoregulation*        | 6.474      | 0.011 | 6.803    | 0.303   | 0.009 |
| Hgb                     | 14.141     | 0.000 | Beta.425 | 0.000   |
| BUN                     | 25.548     | 0.000 | Beta.242 | 0.000   |
| Creatinine              | 4.962      | 0.026 | Beta.005 | 0.037   |
| HDL cholesterol         | 9.443      | 0.002 | Beta.444 | 0.007   |
| Reduced EF (<50%)       | 14.503     | 0.000 | 7.847    | 0.107   | 0.005 |
| EF (%)                  | 9.597      | 0.002 | Beta.076 | 0.003   |
| TIMI flow before PCI    | 0.113      | 0.028 |         |         |
| TIMI 0                  | 6.486      | 0.017 | 5.417    | 0.011   |
| TIMI 1                  | 3.562      | 0.039 | 6.333    | 0.059   |
| Syntax score            | 5.030      | 0.025 | Beta.065 | 0.026   |

Legend: glycoregulation (Gl range 6-10mmol/L); BUN-blood urea; PCI- percutany coronary intervention

We applied the same model for the prediction of cardiac death only without angiographic variables because 4 cardiac deaths occurred in patients who did not underwent coronary angiography (Chi square 47.419; sig 0.000, correct prediction 94.7%) at step 6 we identified three independent predictors: EF (beta -0.368; p = 0.014), BUN (beta 0.267; p = 0.002), and stress glycaemia (beta 0.146; p = 0.060).

During the analysis of the performance capability of stress glycaemia to identify/predict cardiac events and cardiac deaths we found that both in-hospital morbidity and mortality in ACS patients are highly predictable and defined by the stress glycaemia level.

The classification of performance capability of stress glycaemia on cardiac events presented with a ROC curve found an area under the curve of 0.716 and p = 0.000.

The classification of performance capability of stress glycaemia on cardiac death presented with a ROC curve found an area under the curve of 0.850 and p = 0.000.
Hyperglycemia and newly diagnosed diabetes and in-hospital morbidity/mortality

The association of diabetes with increased risk of complications in patients with acute coronary syndrome is a well-known fact. But, less established are the facts that stress hyperglycemia is caring out an increased risk of complications, not only in diabetic but also in patients with previously unknown diabetes. Simon et co-workers reported a positive linear association between the degree of hyperglycemia and the mortality in patients with acute coronary syndrome, independently of the presence of confirmed diabetes [10].

In our study we found that stress hyperglycemia was more pronounced in newly diagnosed diabetics, as compared to patients with well controlled known diabetes. The occurrence of complications was associated with stress hyperglycemia and failure to achieve good glycemic control during hospitalization, not with the presence of diabetes. Patients with newly diagnosed diabetes had the longest hospitalization time and the highest in-hospital morbidity and mortality rates. Although we had a small sample size of newly diagnosed DM patients, the death rate in these was 25% and 18% in known uncontrolled diabetes.

Hyperglycemia is an even more significant predictor of complications as compared with diabetes per se. Patients with stress hyperglycemia with no previous history of diabetes have worse clinical outcome compared to those with pre-existing diabetes with a comparable degree of hyperglycemia. The impact of hyperglycemia on the clinical outcome depends of several factors such as: the intensity of hyperglycemic response, the underlying disease, the co-morbidities, the caloric intake and the risk of infection. Patients with stress hyperglycemia had a higher mortality rate and longer hospital stay compared to those with known diabetes and those with normoglycaemia [2, 4-7, 11].

Points to be remembered: Routine testing for glycolized Hbg seems reasonable in patients admitted due to ACS in all patients hospitalized because of ACS. It helps us to identify diabetic patients yet unidentified. Second, even more important goal, is to establishes a good glycemic control in both cohorts of patients, newly diagnosed and known DM, in order to decrease in-hospital complications and length of hospitalization and increase survival of patients treated because of acute coronary syndrome.

Limitations: One of the major limitations of the study is the small sample size. For these types of studies a bigger number of participants are needed, and of course long term follows up studies.

In conclusion, we identified high prevalence of previously unknown pre-diabetes and diabetes in ACS patients. Stress hyperglycemia and failure to achieve good glycemic control during the hospital treatment were found to be independent predictors of in-hospital morbidity and mortality.

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