Impact of Stress Hyperglycemia on Myocardial Salvage Following Successfully Recanalized Primary Acute Myocardial Infarction

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Background: Elevated blood glucose on admission may worsen outcome after acute myocardial infarction (AMI). No relationship has been identified between admission blood glucose level and myocardial salvage in patients with AMI.

Methods and Results: This study assessed 150 consecutive patients with a first AMI who underwent percutaneous coronary intervention within 24 h from onset of symptoms. Plasma blood glucose was measured on admission. Stress hyperglycemia was defined as blood glucose ≥10 mmol/L (180 mg/dl). The extent of myocardial salvage 7 days after AMI was evaluated on cardiovascular magnetic resonance imaging (CMRI) as the difference between areas of myocardium at risk (T2-weighted hyperintense lesion) and areas of late gadolinium enhancement. The association between stress hyperglycemia and myocardial salvage index (MSI) was investigated in patients with and without diabetes. Among non-diabetic patients, MSI was lower in those with stress hyperglycemia than in those without. No significant difference in MSI was noted between diabetes patients with or without stress hyperglycemia. On multivariate analysis, stress hyperglycemia in patients without diabetes was an independent predictor of MSI.

Conclusions: Stress hyperglycemia affects MSI, indicating that the manipulation of glucose levels could be a potential therapeutic target for salvaging ischemic damage. (Circ J 2012; 76: 2690–2696)

Key Words: Acute myocardial infarction; Myocardial salvage; Stress hyperglycemia
Table 1. Non-Diabetic Patient Characteristics

<table>
<thead>
<tr>
<th>Clinical Factors</th>
<th>&lt;180 mg/dl (10 mmol/L) (n=81)</th>
<th>≥180 mg/dl (10 mmol/L) (n=20)</th>
<th>P-value</th>
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<tr>
<td>Age (years)</td>
<td>64.2±11.3</td>
<td>65.7±14.0</td>
<td>0.24</td>
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<tr>
<td>Male</td>
<td>65 (80)</td>
<td>16 (80)</td>
<td>0.75</td>
</tr>
<tr>
<td>Culprit vessel</td>
<td></td>
<td></td>
<td>0.24</td>
</tr>
<tr>
<td>LAD</td>
<td>36 (44)</td>
<td>8 (40)</td>
<td></td>
</tr>
<tr>
<td>LCX</td>
<td>10 (13)</td>
<td>5 (25)</td>
<td></td>
</tr>
<tr>
<td>RCA</td>
<td>35 (43)</td>
<td>7 (35)</td>
<td></td>
</tr>
<tr>
<td>Coronary risk factor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>42 (52)</td>
<td>13 (65)</td>
<td>0.95</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>39 (48)</td>
<td>7 (35)</td>
<td>0.34</td>
</tr>
<tr>
<td>Current smoking</td>
<td>42 (52)</td>
<td>6 (30)</td>
<td>0.46</td>
</tr>
<tr>
<td>Family history</td>
<td>13 (16)</td>
<td>2 (10)</td>
<td>0.47</td>
</tr>
<tr>
<td>Obesity</td>
<td>25 (31)</td>
<td>5 (25)</td>
<td>0.26</td>
</tr>
<tr>
<td>Medication on admission</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACEI or ARB</td>
<td>8 (10)</td>
<td>0 (0)</td>
<td>0.70</td>
</tr>
<tr>
<td>β-blocker</td>
<td>1 (1)</td>
<td>0 (0)</td>
<td>0.20</td>
</tr>
<tr>
<td>CCB</td>
<td>7 (9)</td>
<td>2 (10)</td>
<td>0.49</td>
</tr>
<tr>
<td>Statin</td>
<td>6 (7)</td>
<td>1 (5)</td>
<td>0.62</td>
</tr>
<tr>
<td>Aspirin</td>
<td>1 (1)</td>
<td>1 (5)</td>
<td>0.36</td>
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<tr>
<td>Reperfusion time (min)</td>
<td>422±407</td>
<td>342±337</td>
<td>0.61</td>
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</table>

Data given as mean±SD or n (%).

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

Ingestion to participate. All patients underwent coronary angiography on admission and then underwent percutaneous coronary intervention (PCI) using coronary stents. All patients were routinely treated with heparin, isosorbide dinitrate, ticlopidine, aspirin, and an angiotensin-converting enzyme inhibitor or an angiotensin II receptor blocker. Stress hyperglycemia was defined as blood glucose ≥10 mmol/L (180 mg/dl) on admission, as described previously.10–11 Patients with a previous or current diagnosis of diabetes or an abnormal oral glucose tolerance test (OGTT, 75-g) 5 days after admission were defined as having diabetes.

Clinical Parameters

The assessed clinical parameters were age, gender, and coronary risk factors (smoking, hypertension, diabetes mellitus, hyperlipidemia, and obesity). The diagnostic criteria for coronary risk factors were as follows. Hypertension: blood pressure ≥140/90 mmHg and/or a history of antihypertensive medication; diabetes mellitus: fasting plasma glucose ≥126 mg/dl, casual plasma glucose ≥200 mg/dl, or a diabetic pattern based on the 75-g OGTT (0 min) ≥126 mg/dl or OGTT (2h) ≥200 mg/dl; hyperlipidemia: serum total cholesterol ≥220 mg/dl or serum triglyceride >150 mg/dl; obesity: body mass index ≥25 kg/m².

Blood Sampling and Analysis

Venous blood was collected routinely and immediately after admission for plasma glucose determination, before any i.v. medication. Samples were analyzed in the hospital’s central laboratory.

Of the 195 study participants, 133 who did not have diabetes were divided on the basis of their admission plasma glucose level into either the normoglycemia group (plasma glucose <180 mg/dl [10 mmol/L]) or the stress hyperglycemia group (plasma glucose ≥180 mg/dl). Similarly, the remaining 62 subjects with diabetes were divided on the basis of their admission plasma glucose level into the same groups (normoglycemia, plasma glucose <180 mg/dl; stress hyperglycemia, plasma glucose ≥180 mg/dl). The 4 groups were compared with regard to baseline and admission characteristics.

Non-Invasive CMRI Protocol

The CMRI was performed using a 1.5-T clinical scanner (Intera Achieva; Philips Medical Systems, Best, The Netherlands) equipped with a 5-element cardiac phased-array coil for signal reception 7 days after the onset of AMI, as previously described.12-14 During the examination, patients were continuously monitored on single-lead ECG, repeated blood pressure measurements, and pulse oximetry. With the patient in the supine position, contiguous short-axis cine images covering the left ventricle (LV) from base to apex were acquired using a standard steady-state free-precession sequence. We then applied a breath-hold T2 W sequence with short-TI inversion recovery for fat saturation. Imaging parameters were repetition time (TR), 2 R-to-R intervals; echo time (TE), 90 ms; slice thickness, 8 mm; field of view (FOV), 35 μm; matrix, 256x512 in 3 short-axis slices (basal, midventricular, and apical). Each slice was obtained during an end-expiratory breath-hold of 12–15 s depending on the patient’s heart rate.

LGE imaging covering the whole ventricle was required 10–15 min after i.v. injection of 0.1 mmol/kg gadolinium diethylenetriamine penta-acetic acid (Gd-DTPA; Magnevist, Schering, Berlin, Germany). We used a 3-D inversion-recovery turbo gradient echo sequence, and images were obtained during an end-expiratory breath-hold. Scan parameters were as follows: TR, 4.1 ms; TE, 1.25 ms; flip angle, 15°; FOV, 350x350 mm; partial echo; matrix, 224x256; and spatial resolution, 1.56x2.24x10 mm³ reconstructed to 0.68x20.68x5 mm³. We optimized the inversion time (200–300 ms) to null the nor-
mal myocardium. The slice positions for both T2 W and LGE acquisitions matched those of the cine images.

CMRI Data Analysis

All analyses were performed by consensus of 2 blinded observers (Y.O. and A.S.) on an off-line workstation (View Forum, Philips Medical Systems). The extent of the AAR (T2 W hyperintense lesion) and the extent of the area of LGE were quantified on the same slice location with the maximum extent of T2 signal abnormality using formula as previously reported: (area of high signal/total slice area) × 100. The extent of myocardial salvage 7 days after AMI on CMRI was then defined as the proportion of maximum extent of T2 signal abnormality minus the proportion of LGE in the corresponding section. Myocardial salvage index (MSI) was obtained as follows: extent of myocardial salvage/extent of myocardial AAR × 100. Regional LV function was assessed by determining systolic wall motion in the infarct region. The change of regional wall motion was defined as percent increase of LV wall motion during systole compared with diastole. Myocardial segments showing LGE at day 7 were defined as the infarct region. For an assessment of infarct size, LV myocardium with LGE volumes were quantified. For analysis of the change of regional wall motion, the 2 most basal and 2 most distal slices were excluded, because short-axis images at these levels preclude a reliable segmental evaluation owing to the presence of the LV outflow tract and small diameter, respectively.

Statistical Analysis

All data are expressed as mean±SD unless stated otherwise. Data were analyzed by comparing a non-diabetic group with a diabetic group. Univariate analysis of differences between the groups was performed using the 2-tailed unpaired t-test and chi-squared or Fisher’s exact tests for myocardial salvage variables for the diabetic and non-diabetic groups. P<0.05 were considered significant. A multivariate regression model was used to determine predictors of MSI. Those variables that had P<0.1 on univariate analysis (admission plasma glucose level and reperfusion time) and clinically meaningful factors for MSI (age, sex, hypertension, hyperlipidemia, smoking and obesity) were included in the multivariate logistic analysis. All statistical analysis was done using SPSS version 11.0 (SPSS, Chicago, IL, USA). The authors had full access to the data and take responsibility for its integrity.

Results

Patient Characteristics

Of 195 eligible ST-segment elevation myocardial infarction patients, this study enrolled 150 consecutive patients. The main reasons for exclusion were the lack of CMRI (n=19) and previous myocardial infarction (n=26). Nineteen patients were unable to complete cardiac MRI. Reasons for not undergoing CMRI were difficulties of breath-holding (n=2); arrhythmia (n=2); contrast media allergy (n=1); renal dysfunction (serum creatinine ≥ 1.5 mg/dl, n=9); and long-term intensive care over 7 days (congestive heart failure, n=2; myocardial rupture, n=2; subacute stent thrombosis, n=1). In all remaining 150 patients, clinical outcome dates were available and CMRI quality was suitable to assess myocardial salvage.

On the basis of admission blood glucose level, 81 of the 101 non-diabetic subjects were assigned to the normoglycemia group (<180 mg/dl), and the remaining 20 subjects were assigned to the stress hyperglycemia group (≥180 mg/dl). Of the 49 diabetic subjects, 16 were assigned to the normoglycemia group and 33 to the stress hyperglycemia group.

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ified by blood glucose level are listed in Tables 1, 2, respectively. There were no statistically significant differences in characteristics between the 2 groups without diabetes. Compared with the groups without diabetes, those with diabetes were significantly older and more likely to have hypertension.

Effects of Admission Blood Glucose Level on Acute-Phase MSI

We investigated the relationship between admission blood glucose level and MSI 7 days after AMI. MSI 7 days after AMI was evaluated on CMRI as the difference between the areas of myocardium at risk (T2W hyperintense lesion) and the areas of LGE (Figure 1). Evaluation of MSI in a representative non-diabetic patient without stress hyperglycemia and in a representative non-diabetic patient with stress hyperglycemia is shown in Figure 1. MSI was lower in non-diabetic subjects with stress hyperglycemia than in those without stress hyperglycemia (55.4±13.3% vs. 49.1±10.0%, P=0.047). In subjects with diabetes, no significant association was evident between MSI and glucose level on admission (P=0.422; Figure 2).

On multiple regression analysis, including all variables, stress hyperglycemia appeared to be an independent predictor of MSI in subjects without diabetes (adjusted R²=0.053, P=0.046) (Table 3). Reperfusion time tended to correlate with MSI, but was not significant (adjusted R²=0.61, P=0.084). No other clinical factors affected MSI in subjects without diabetes. In contrast, in subjects with diabetes, stress hyperglycemia did not appear to be an independent predictor of MSI (adjusted R²=0.76, P=0.442).

CMRI was performed in 73 patients (non-diabetes, n=54; diabetes, n=19) during the chronic phase 6 months after the onset of AMI. The change of regional wall motion in the chronic phase was correlated with MSI of acute phase (r=0.49, P<0.001; non-diabetes, r=0.45, P=0.001; diabetes, r=0.64, P=0.004; Figure 3).

Discussion

We describe herein for the first time the association between impaired MSI and stress hyperglycemia in patients without diabetes on admission for AMI. Although the detailed mechanisms of the association between stress hyperglycemia and the impairment of MSI recovery remain unclear, glycemic status on admission provides important clues for treatment.

Stress hyperglycemia with AMI is associated with an increased risk of in-hospital mortality in patients with and without diabetes. In addition, it has been reported that there is a linear positive relationship between stress hyperglycemia and

Figure 1. Measurement of myocardial salvage index evaluated on short-axis cardiac magnetic resonance imaging obtained 7 days after acute myocardial infarction. (A) Non-diabetic patient without stress hyperglycemia; (B) non-diabetic patient with stress hyperglycemia. (A-1, B-1) Extent of the area at risk (hyperintense areas on T2-weighted, fast spin-echo). (A-2, B-2) Infarcted areas (late gadolinium enhancement-bright images). Percent salvaged myocardium was determined as follows: (area of high signal/total slice area)×100.
Myocardial salvage index (MSI) was lower in non-diabetic subjects with stress hyperglycemia (admission blood glucose ≥180 mg/dl [10 mmol/L]) than in those without stress hyperglycemia (<180 mg/dl; P=0.047). In contrast, MSI was not significantly associated with glycemia in diabetic subjects, irrespective of glucose level on admission (P=0.422). *P<0.05 vs. non-diabetic <180 mg/dl.

Table 3. Admission Blood Glucose and Myocardial Salvage

<table>
<thead>
<tr>
<th></th>
<th>Standardized regression coefficient</th>
<th>Partial correlation coefficient</th>
<th>P-value</th>
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<tbody>
<tr>
<td>Non-diabetic group</td>
<td>-0.211</td>
<td>-0.209</td>
<td>0.046</td>
</tr>
<tr>
<td>Diabetic group</td>
<td>0.076</td>
<td>0.073</td>
<td>0.626</td>
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</table>

*Multivariate analysis.

Figure 2. Myocardial salvage index was correlated with the change of regional wall motion in the chronic phase.
Stress Hyperglycemia and Myocardial Salvage

Several study limitations should be considered in the interpretation of the results. First, the results were prospective in terms of patient enrollment but observational in nature. Thus, the present study does not provide a mechanistic explanation for the improvement in MSI 7 days after AMI that was associated with stress hyperglycemia on admission. Second, we performed CMRI assessment of MSI 7 days after reperfusion. The time point at which an area of myocardial edema is determined is very important, and it may have an influence on the results. Wright et al evaluated the influence of the time delay between PCI and AAR assessment in patients with reperfused AMI with T2 W CMRI. In their study CMRI was performed between 1 and 20 days after PCI (mean and median 4 days). There was no correlation between T2 W AAR and the delay between PCI and CMRI (r=0.11, P=0.27). They concluded that the delay between PCI and CMRI did not cause systematic differences in the measured T2 W AAR or myocardial salvage. In addition, myocardial edema has been found to be a tissue footprint of the AAR, lasting for at least 7 days after AMI, enabling an extension of the window for assessing myocardial salvage. Botker et al recommended that the optimal time to evaluate myocardial salvage within 1 cardiac examination seems to be at 1–2 weeks after infarction, because the extent of the final infarct size on LGE CMRI remains almost constant after 1 week and AAR remains stable for at least 7 days. But we cannot exclude the possibility that earlier data acquisition would have influenced the extent of myocardial salvage. In terms of the optimal timing of measurement of T2 W AAR or myocardial salvage, further serial studies of individual patients are needed.

Conclusions

Stress hyperglycemia in patients without diabetes following AMI may be an important marker for risk stratification while potentially influencing therapeutic strategies.

Acknowledgment

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Disclosure

Conflict of interest and financial disclosure: None.

References

2. Norhammar AM, Ryden L, Malmberg K. Admission plasma glucose concentration of acute myocardial infarction even in nondiabetic patients.


