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Original article

Association between hyperglycemia at admission and microvascular obstruction in patients with ST-segment elevation myocardial infarction

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ABSTRACT

Background: Blood glucose level at admission in ST-segment elevation myocardial infarction (STEMI) is a predictor of heart failure and mortality. A previous study showed the association between hyperglycemia and microvascular dysfunction using myocardial contrast echocardiography. Late gadolinium enhancement (LGE) cardiovascular magnetic resonance imaging (CMR) can demonstrate microvascular obstruction (MVO) as the area with hypointense core within LGE. This study was performed to investigate the association between hyperglycemia at admission and MVO using CMR in patients with STEMI.

Methods: Ninety-three patients with first STEMI who were treated by percutaneous coronary intervention (PCI) were included. CMR was performed within 7 days after PCI. Venous blood was collected routinely immediately after admission for plasma glucose determination before intravenous injection of some medications. Samples were analyzed in the hospital's central laboratory. We performed LGE-CMR to assess the presence of MVO.

Results: MVO was found in 34 (37%) of all 93 patients; their glucose level at admission was significantly higher than that of patients who did not exhibit MVO [204 (153–267) mg/dl vs. 157 (127–200) mg/dl, $p = 0.002$]. There were no differences in glycosylated hemoglobin and incidence of diabetes mellitus between the two groups. A multivariable logistic regression analysis showed that glucose level at admission was an independent predictor of MVO (odds ratio, 1.014; 95% confidence interval, 1.004 to 1.023; $p = 0.006$). The glucose level at admission 190 mg/dl was the best threshold value for identifying MVO. The occurrence of MVO was significantly higher in the patients with glucose level at admission $\geqq$190 mg/dl compared with the patients with glucose level $<$190 mg/dl [18 (53%) vs. 16 (27%), $p = 0.023$].

Conclusions: Hyperglycemia at admission in STEMI patients who were treated by PCI was associated with the presence of MVO assessed by LGE-CMR.

Introduction

Primary percutaneous coronary intervention (PCI) is an established reperfusion therapy for acute myocardial infarction (AMI) [1]. However, patency of the epicardial coronary artery does not always guarantee salvage of the myocardium at risk for ischemia. Patients with no-reflow phenomenon which is likely to be caused by microvascular obstruction (MVO) are associated with poor clinical outcomes when compared with patients with a good flow after reperfusion [2,3]. Late gadolinium enhancement (LGE) cardiovascular magnetic resonance imaging (CMR) was introduced as an effective method to assess infarct size and MVO [4,5]. However, the predictor of MVO after primary PCI has not been fully investigated.

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Hyperglycemia can be observed in patients with AMI, irrespective of a history of diabetes mellitus (DM) [6–8]. An association between plasma glucose level at admission and an increased risk of mortality and poor prognosis after AMI has been reported [8–12]. As the underlying mechanisms of these deleterious effects of hyperglycemia, a previous study showed the association between hyperglycemia at admission and microvascular dysfunction using myocardial contrast echocardiography [13]. The aim of the present study was to investigate the association between hyperglycemia at admission and CMR-derived MVO after primary PCI in patients with ST-segment elevation myocardial infarction (STEMI).

Materials and methods

Study population

Between September 2011 and April 2013, we enrolled a total of 93 patients with STEMI who were recanalized with PCI. We carefully excluded patients with cardiogenic shock, unstable hemodynamic status, previous myocardial infarction, previous coronary bypass surgery, significant renal dysfunction (glomerular filtration rate <30 ml/min/1.73 m^2), or contraindications for CMR such as metal implants. CMR was performed within the first week after PCI in all patients. A diagnosis of STEMI required the presence of chest symptoms continuing for >30 min, ST-segment elevation of >0.1 mV in 2 limb leads on electrocardiogram, and an increase in serum creatine kinase-myocardial band (CK-MB) levels to more than twice the upper limit of normal. Patients with a previous or current diagnosis of DM or an abnormal oral glucose tolerance test (75 g) 5 days after admission were defined as having DM. Venous blood was collected routinely and immediately after admission for plasma glucose determination, before intravenous injection of some medications. Samples were analyzed in the hospital's central laboratory. This study was approved by the Ethics Committee of Wakayama Medical University and all patients gave their written informed consent.

Primary PCI procedure

Oral aspirin (200 mg) and intravenous heparin (100 U/kg) were administered before coronary catheterization. Thrombolysis was not performed for any patients. Coronary angiograms were obtained from a standard series of 6 to 8 projections for the left coronary artery and 2 to 3 projections for the right coronary artery. The infarct-related lesion was identified on the basis of the findings by a coronary angiogram as well as an electrocardiogram and transthoracic echocardiogram. In patients with thrombolysis in myocardial infarction (TIMI) flow grade <2, aspiration thrombectomy was performed using a 5.1-French aspiration catheter (ExportCatheter, Medtronic Japan, Tokyo, Japan). PCI was performed using a 6-French guiding catheter, a 0.014-inch guidewire, a monorail balloon catheter, and a coronary stent with a conventional technique. Using the intracoronary imaging devices, decision-making related to the PCI strategy depended on the discretion of individual operators. All patients received dual antiplatelet therapy with aspirin (81 mg/day) and clopidogrel (75 mg/day) after primary PCI.

Angiographic analysis

Coronary angiograms were reviewed by independent observers blinded to MRI findings. The degree of perfusion was evaluated according to TIMI criteria [14]. Collaterals were graded according to Rentrop's classification, with good collateral flow defined as grade 2 or 3 [15]. No reflow phenomenon was defined as TIMI grade 0, 1, or 2 without mechanical obstruction on angiograms after PCI. Reperfusion injury was defined as ST-segment re-elevation or ventricular tachycardia/ventricular fibrillation just after recanalization in this study.

CMR protocol

All CMR examinations were performed using a 1.5-T clinical scanner (Intera Achieva; Philips Medical Systems, Best, The Netherlands) equipped with a 32-element cardiac phased-array coil for signal reception within the first week after PCI, as previously described [16]. During the examination, patients were continuously monitored on single-lead electrocardiogram, 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. LGE imaging covering the whole ventricle was required 10–15 min after intravenous injection of 0.1 mmol/kg gadolinium diethylenetriamine pentaacetic acid (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, 350 mm $\times$ 350 mm; partial echo; matrix, 224 $\times$ 256; and spatial resolution, 1.56 mm $\times$ 2.24 mm $\times$ 10 mm reconstructed to 0.68 mm $\times$ 20.68 mm $\times$ 5 mm. Inversion time was adjusted to null the signal from viable myocardium [17].

CMR analysis

All analyses were performed by consensus of independent blinded observers on an off-line workstation (View Forum, Philips Medical Systems).

On cine CMR, for quantification of LV volumes and LV ejection fraction (EF), endocardial borders were delineated on the end-diastolic and end-systolic short-axis slices.

On LGE-CMR, we assessed the presence of infarct area and MVO. We calculated infarct size by automatic summation of all slice volumes of infarct area and expressed as a percentage of LV volume [18]. According to a previous study [19], the infarct area was defined as the area with signal intensity 5 SDs above the mean signal obtained in the remote noninfarcted myocardium on LGE images. MVO was detected as the area with hypointense core within LGE, which was included in the area of infarction [20].

Statistical analysis

All statistical analyses were carried out using SPSS version 11.0 (SPSS, Chicago, IL, USA). Categorical variables were presented as frequency counts and percentages, with comparison with chi-square statistics. Continuous variables were presented as median and interquartile range and compared using the Mann–Whitney *U* test. A multivariable logistic regression analysis was used to determine predictors of MVO. The model included clinical and angiographic characteristics with $p < 0.05$ in the univariate analyses. Receiver operating characteristics (ROC) curve analysis was performed to establish glucose level at admission as a predictor of MVO. The resulting sensitivity, specificity and area under curve (AUC) were calculated. The best cut-off value was determined by the maximum sum of sensitivity and specificity. The relationship between glucose level at admission and infarct size was analyzed by Pearson correlation analysis. A p-value <0.05 was considered statistically significant.