〈Original Article〉

Clinical state of acute myocardial infarction with increased phosphoglucomutase activity: The relationship between phosphoglucomutase activity and acute thrombosis

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Summary Phosphoglucomutase (PGM) is a key enzyme in cellular glucose utilization and energy homeostasis. We previously found that patients with acute myocardial infarction (AMI) showed high PGM activity, especially those with ST-elevation myocardial infarction (STEMI). For clarifying whether PGM activity was related to acute thrombosis in patients with STEMI, the PGM activity in both a rat acute thrombosis model and in patients with AMI was evaluated. In the rat thrombosis model, PGM activity increased irrespective of the development of thrombosis, whereas PGM activity increased in AMI patients with coronary thrombosis, especially those in a shock state. Our findings suggest that PGM activity can increase with myocardial damage, the formation of a thrombus, and the presence of inflammation associated with coronary plaque rupture. PGM activity might be useful for the diagnosis of acute coronary thrombosis in AMI patients and a useful predictive marker in the prognosis of AMI patients.

Key words: Acute coronary thrombosis, Phosphoglucomutase (PGM) activity, ST-elevation myocardial infarction (STEMI)

1. Introduction

Acute myocardial infarction (AMI) is a major cause of death and hospitalization. Recently, several new cardiac biomarkers have emerged as strong predictors of risk among AMI patients, including markers of coronary risk factors (low-density lipoprotein cholesterol[1)] and hemoglobin A1c [HbA1c]), myocardial biomarkers (cardiac troponin T [TnT][2)]), thrombosis biomarkers (D-dimer[3)] and total plasminogen-activator inhibitor type 1 [PAI-1][4)]), and inflammatory biomarkers (C-reactive protein [CRP][5)]

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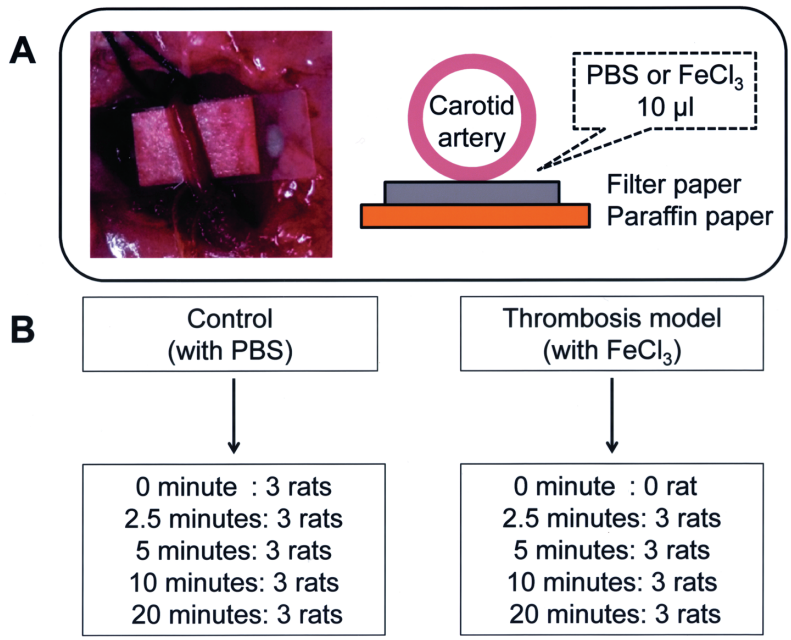

Fig. 1 Study design and methods in the acute thrombosis rat model.
A: An operative photograph of common carotid artery (CCA) in the rat acute thrombosis model.
B: A study chart of the acute thrombosis rat model. PBS, phosphate-buffered saline.

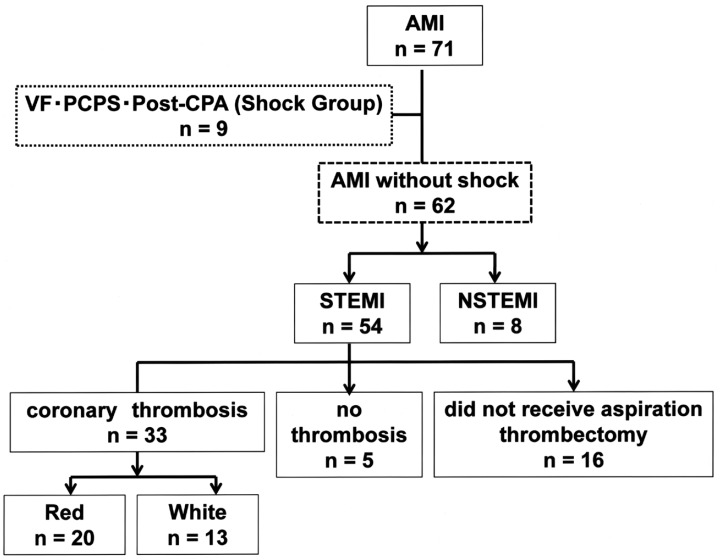

Fig. 2 Flow chart of patient enrollment.
A total of 235 EAP and 71 AMI patients were admitted to our hospital from March 2010 to January 2011. Nine patients who had developed shock were enrolled in the AMI with shock group. The remaining 62 patients (54 patients with STEMI, 8 with NSTEMI) were enrolled in the AMI without shock group. Patients with STEMI were divided into 3 groups in order to clarify the relationship between PGM activity and coronary thrombosis.
EAP, stable effort angina pectoris; AMI, acute myocardial infarction; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; VF, ventricular fibrillation; Post-CPA, post-cardiopulmonary arrest; PCPS, percutaneous cardiopulmonary system.

and plasma pentraxin 3 [PTX3][6]. Phosphoglucomutase (PGM) is a key enzyme in the metabolic pathway that catalyzes the interconversion of glucose-6-phosphate (G6P) and glucose-1-phosphate (G1P), with the latter then converted into uridine diphosphate (UDP)-glucose[7, 8, 9]. Several PGM isoforms (genes: PGM1 to PGM5) have been reported[10, 11, 12, 13], with PGM1 predominating in total PGM activity (approximately 90%)[11]. PGM1 is expressed ubiquitously, but high levels of expression are observed particularly in the heart, skeletal muscles, kidney, liver, and lungs[13]. In our previous study, serum PGM activities were measured with several myocardial, thrombosis, and inflammatory biomarkers in patients with stable effort angina pectoris (EAP) and AMI. PGM activity in the AMI group was significantly higher than that in the EAP group[14]. In addition, PGM activity may potentially be increased in parallel with myocardial, thrombosis, and inflammation biomarkers in patients with AMI. In particular, the peak PGM activity was higher in patients with ST-elevation myocardial infarction (STEMI) than in patients with non-STEMI (NSTEMI)[14]. STEMI is associated with acute coronary thrombosis[15]. However, the mechanism for the increase in PGM activity remains unclear. Moreover, the clinical importance of PGM activity has not been investigated in patients with STEMI.

A rat model has been generated for the study of arterial thrombosis[16, 17]. Although the mechanism of vascular injury remains unclear, arterial thrombosis by perivascular stimulation from $FeCl_3$ exposure was found to lead to the topical induction of luminal thrombus formation[16, 17]. The operation for inducing the injury is easy to perform, enables the evaluation of the rapid onset of endothelial injury, and has been valuable in thrombosis research[18, 19].

The aim of this study was to clarify whether PGM activity is associated with acute coronary thrombosis in patients with AMI. Further, the relationship between PGM activity and acute arterial thrombosis has been discussed on the basis of the results of a validation experimental study conducted using a thrombosis rat model.

2. Methods

1. Study design for the rat acute thrombosis model

All studies were performed in accordance with the Guide for the Care and Use of Laboratory Animals published by the US National Institutes of Health (NIH Publication No. 85-23, revised 1996) and Ethics Committee of Kitasato University School of Medicine.

The study design and methodology for the experiments involving the rat acute thrombosis model are shown in Figure 1. For the induction of arterial thrombosis, 9-week-old Sprague-Dawley male rats (body weight, 297.5 ± 19.7 g; CLEA Japan, Inc., Tokyo, Japan) were anesthetized with diethyl ether and placed in a supine position. After a midline incision extending from the mandible to the suprasternal notch and retraction of the overlying skin, the exposed anterior cervical triangle was blunt dissected to free the common carotid artery (CCA) from the surrounding tissue. Filter paper (10 × 5 mm) placed on a layer of paraffin paper (20 × 6 mm) was saturated with 40% aqueous solution of $FeCl_3$ (10 μl, thrombosis model) or phosphate-buffered saline (10 μl, control) and placed over the right CCA[20]. The exposure times to $FeCl_3$ were 2.5, 5, 10, and 20 minutes in both the thrombosis model and control groups (n = 3 per group). After each exposure time, the CCA was ligated at the proximal and distal sites, carefully dissected, and fixed overnight in a 4% paraformaldehyde phosphate-buffer solution at 4℃. The fixed samples were embedded in paraffin and cut into 4-μm thick serial sections for staining with hematoxylin-eosin (HE) for histological evaluation.

2. Blood collection and measurement of thrombosis markers in rats

Blood (5 ml) was sampled from the inferior vena cava of each anesthetized rat after each exposure of the carotid artery to $FeCl_3$ by withdrawing the blood through 21G needles into plastic syringes. PGM activity was examined with serum D-dimer, which was measured using an enzyme-linked immunosorbent assay (ELISA) kit for D-dimer (D2D; Uscn Life Science, Inc., Wuhan, China) in rats. The analytical

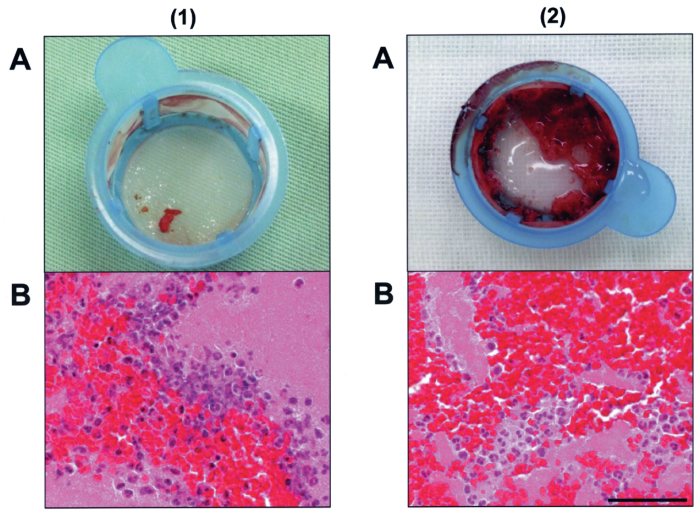

Fig. 3 Histology of acute coronary thrombosis in patients with STEMI.
A: Photographs of acute coronary thrombosis by aspiration thrombectomy.
(1) White thrombosis. (2) Red thrombosis.
B: Hematoxylin-eosin (HE) staining (×400: scale bar, 50 μ m). Both histological images show mixed thrombosis with red blood cells, neutrophilic leukocytes, and platelet cells.

range of this kit was 0.78-50.0 ng/ml (78.0-500.0 mg/dl) for the D-dimer assay.

3. Study design and patient population

PGM activity was examined in patients with EAP and AMI at Kitasato University Hospital between March 2010 and January 2011. The following patients were enrolled in the study: 1) 235 consecutive patients with EAP who were scheduled to receive a primary percutaneous coronary intervention (PCI) for the management of de novo lesions of a >75% diameter stenosis in a native coronary artery with the presence of typical stable effort angina or who showed positive results for the myocardial stress test, and 2) 71 consecutive AMI patients who received PCI. Nine AMI patients who had developed shock, such as ventricular fibrillation (VF), or post-cardiopulmonary arrest (CPA) and had used the percutaneous cardiopulmonary system (PCPS) were enrolled in the AMI with shock group (Figure 2). The remaining 62 patients (54 patients with STEMI, 8 with NSTEMI) were enrolled in the AMI without shock group. STEMI was defined as at least 1 mm of ST-segment elevation in more than 2 contiguous leads in V1-V6 or more than 2 leads in standard limb leads by electrocardiogram. The NSTEMI did not show a ST-segment elevation.

The baseline characteristics of the study subjects were age; gender; the diagnoses of hypertension, dyslipidemia, and diabetes mellitus (these were obtained from medical records or the patient's history of currently or previously received medical therapy); symptom onset-to-balloon time; and death within 3 months. The lesion characteristics were coronary thrombosis by aspiration thrombectomy, the locations (RCA, right coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery), thrombolysis in myocardial infarction trial (TIMI) grade (grade 0 to 3)[21], and collateral arteries (Rentrop grade, grade 0 to 3) to the culprit lesion.

The patients with AMI received dual antiplatelet therapy (DAPT, aspirin [100 mg] and clopidogrel [300 mg]) just before PCI. After a coronary stent was implanted, the patient received DAPT at least 6 months after the PCI. All local ethics committees

approved the protocol; all patients gave informed consent before enrollment. The study was performed in accordance with the Declaration of Helsinki.

4. Histology of acute coronary thrombosis by aspiration thrombectomy

Thrombosis samples were obtained from intracoronary arteries in patients with STEMI using a 6 or 7 Fr export aspiration catheter (ZEEK; Zeon Medical Inc. Tokyo, Japan or Thrombuster II; Kaneka Medical Products, Osaka, Japan) and were fixed overnight in a 4% paraformaldehyde phosphate buffer solution at 4℃, embedded in paraffin, and cut into 4-μm thick serial sections for staining with HE. Thrombosis composed primarily of red blood cells and fibrin was regarded as red thrombosis; thrombosis consisting predominantly of platelets and fibrin was regarded as white thrombosis (Figure 3). In 54 patients with STEMI, 33 patients had coronary thrombosis by aspiration thrombectomy (n = 33; 20 patients had red thrombosis; 13, white thrombosis), 5 had no thrombosis by aspiration thrombectomy, and 16 did not receive aspiration thrombectomy.

5. Blood sampling procedures and laboratory measurements in patients with AMI

Blood samples from patients were collected on admission. In patients with AMI, both PGM activity and creatinine phosphokinase (CPK) were measured until the level of CPK peaked out in order to clarify the relationship between PGM activity and CPK. PGM activity and well-established myocardial biomarkers (aspartate aminotransferase [AST], cardiac [TnT], and thrombosis biomarkers [plasma D-dimer[3)] and PAI-1[4)]]), and inflammatory biomarkers (CRP[5)] and PTX3[6)]) were also examined on admission. Plasma PTX3 levels were measured using the Human Pentraxin3 ELISA kit (Perseus Proteomics, Inc., Tokyo, Japan); the analytical range of this kit was 0.1-20.0 ng/ml for the PTX3 assay[6)].

6. Measurement of PGM activity

Blood samples were immediately centrifuged at 3000 × g for 10 minutes, and the clarified serum samples were frozen, stored at -70℃, and thawed just before the assay. All measurements of serum PGM activity were performed in duplicate; the researcher who performed the measurements was blinded to the other sample data. The activity of homogeneous cow cardiac muscle was measured in each assay as a control.

The levels of serum PGM activity were analyzed using an automated analyzer (AU 640; Beckman Coulter, USA). The reagent (100 mmol/l Tris-HCl buffer (pH 8.0), 0.11 mmol/l glucose-1,6-diphosphate [G1,6DP], 2.7 U/ml glucose-6-phosphate dehydrogenase [G6PD], 0.55 mmol/l NADP, 17 mmol/l G1P, 6.8 mmol/l EDTA-Mg (Ⅱ), and 0.27 mmol/l N-acetylcysteine [NAC]) for the measurement of PGM activity prepared made according to a previous study[22)], but with high-quality chemicals and enzymes. After pre-incubation at 37℃, 180 μl of reagent was added to 20 μl of serum. The PGM activity was calculated on the basis of the change in absorbance (340 nm) for 1 minute due to the formation of NADPH.

7. Statistical analysis

Continuous data are presented as the mean ± standard deviation (SD), and categorical data are presented using absolute frequencies. Statistical analyses between continuous variables were performed with one-way ANOVA. Chi-square analysis or Fisher's exact probability test was used to evaluate differences in categorical variables between the groups. A probability value (P value) of less than 0.05 was considered significant. Statistical analyses were performed with JMP 6 software for Windows (SAS Institute Inc., Cary, NC, USA) and Stata 11 software (StataCorp., TX, USA).

2. Results

1. Histology and PGM activity in the rat acute thrombosis model

The histology of the CCA in the acute thrombosis model is shown in Figure 4. In the control, the vessel wall showed a normal structure, and no luminal thrombosis was observed. After exposure of the CCA to $FeCl_3$ for 2.5 minutes, acute thrombosis and vessel wall degeneration were found. The thrombosis and

vessel wall-degeneration worsened following 5 minutes of exposure but did not increase in severity following 10 and 20 minutes of exposure.

The changes in PGM activity and D-dimer levels according to the exposure time are shown in Figure 5. The levels of PGM activity were high in both the

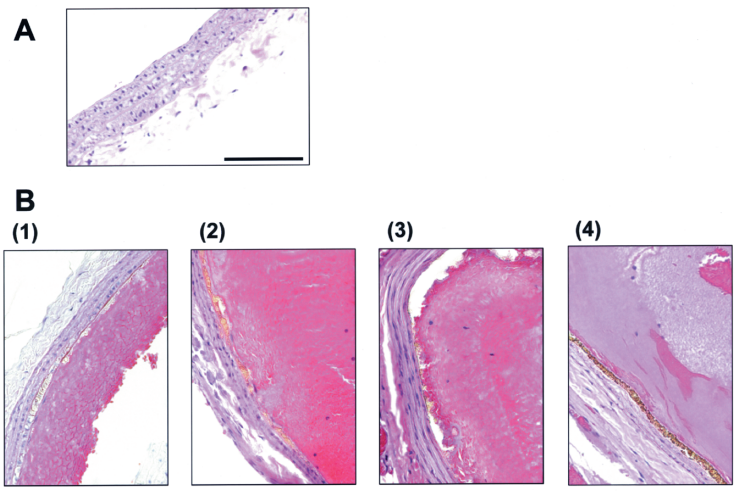

Fig. 4 Histology of the CCA in the rat acute thrombosis model.
Control CCA (PBS treatment) and CCAs exposed to FeCl3 for 2.5, 5, 10, and 20 minutes were fixed, processed, and stained with hematoxylin-eosin (HE) (×200: scale bar, 100 μm).
A: Control with PBS. B: Thrombosis model with $FeCl_3$.
(1) 2.5 minutes, (2) 5 minutes, (3) 10 minutes, (4) 20 minutes.
Acute thrombosis and vessel wall degeneration were observed in samples exposed to $FeCl_3$ for 2.5 minutes, increased in severity in those exposed for 5 minutes after the exposure, but did not worsen upon exposure.

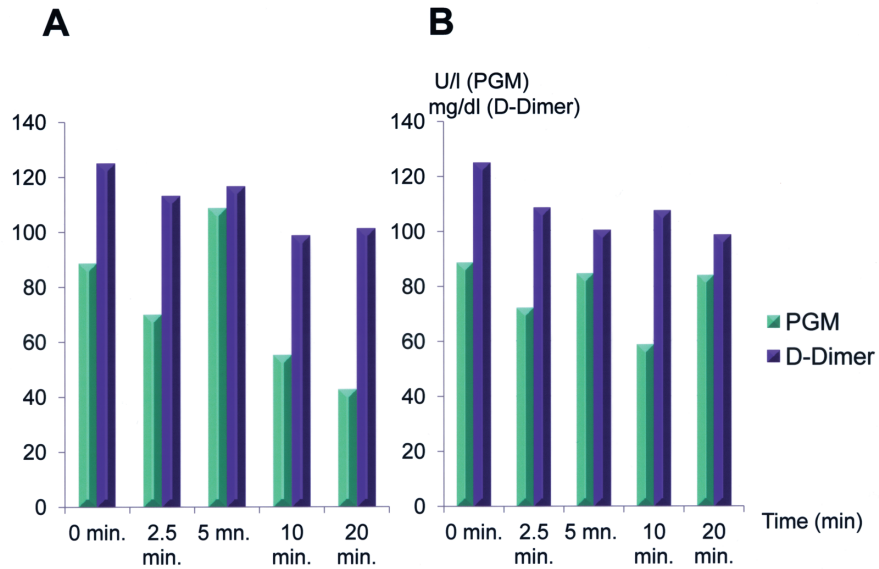

Fig. 5 PGM activity and D-dimer in rat acute thrombosis model. A: Control with PBS, B: Thrombosis model with $FeCl_3$. PGM, phosphoglucomutase.

thrombosis model and control groups, with no significant differences between the 2 groups relative to each exposure time. The changes in the D-dimer levels were within the normal range.

2. Characterization of STEMI with coronary artery thrombosis

The basic characteristics of the 235 patients with EAP and the 33 AMI patients with thrombosis are shown in Table 1. The levels of PGM activity on admission were increased in addition to CPK, AST, TnT, PAI-1, and PTX3 in AMI patients with thrombosis. The comparison of PGM activity in patients with red or white thrombosis is shown in Table 2. The onset-to-balloon-time was shorter in the red thrombosis group than that in the white thrombosis group, while the PAI-1 levels were higher in the former group than those in the latter group. However, the PGM activity and the other biomarkers (CPK, AST, TnT, CRP, and PTX3) were not significantly different between the red and white thrombosis groups.

3. Comparison of PGM activity between AMI patients with shock and without shock

The baseline and lesion characteristics and biomarkers of AMI patients with shock and without shock are summarized in Table 3. The baseline and lesion characteristics were not significantly different between the 2 groups, with the exception of death. The PGM activity, the thrombosis markers (D-dimer and PAI-1), the peak PGM activity, and the peak CPK in AMI patients with shock were significantly higher than those in AMI patients without shock on admission. However, the level of CPK on admission and CRP were not significantly different between the 2 groups. PTX3 was not measured on admission because the remaining plasma samples were insufficient.

3. Discussion

1. PGM activity in the rat acute thrombosis model

In the present study, we applied an established model of arterial injury to induce platelet-rich thrombosis in the CCA of rats. The histological study revealed that the thrombosis formation after 2.5 minutes of exposure to $FeCl_3$ was less severe than those after longer exposure times, but PGM and D-dimer levels were not correlated with the formation of thrombosis in the histological examination. Hence, although the PGM activity was high, it showed no relation to the development of thrombosis.

2. PGM activity with coronary thrombosis in STEMI patients

Most arterial thrombosis develops during platelet accumulation and the activation of coagulation factors[23]. Moreover, accumulated platelets and coagulated red blood cells were histologically evident in white and red thromboses[24]. The levels of D-dimer correlate well with subsequent coronary artery events[25]. In addition, increased PAI-1 expression has been reported to potentially constitute a considerable portion of thrombus enlargement[4].

The PGM activity in AMI patients with thrombosis was higher than that in patients with EAP. As well, the PGM activity increased with the myocardial biomarkers, PAI-1 and PTX3. Arimura has reported that rat cardiomyocytes are possibly associated with the abnormal recruitment of PGM at the Z-disc under stress[26]. Hence, it was assumed that the high PGM activity in patients with AMI originated from myocardial damage. As well, the levels of PAI-1 were significantly higher in patients with STEMI (AMI patients with thrombosis). Folsom et al. reported that elevated PAI-1 is a primary risk factor for AMI[27], and Collet et al. reported that the amount of the acute release of PAI-1 is significantly higher in AMI patients in Killip class > 3 on admission than in those with a less severe presentation[28]. Additionally, higher levels of PAI-1 on admission were also independent predictors of in-hospital and 1-year mortality in cardiac shock patients[29]. PAI-1 might be associated with not only thrombus formation but also with patient prognosis. Hence, the high PGM activity in patients with AMI might have originated from thrombus. However, the PGM activity was not influenced by the kind of thrombus, in particular red and white thrombosis. As well, CRP (especially highly sensitive CRP) has become a valuable inflammatory marker and provides

Table 1 Comparison of PGM activity and established biomarkers between EAP patients and AMI patients without shock

	EAP (n = 235)	**AMI with thrombosis** (n = 33)	**P-value**
Baseline Characteristics			
Age (years)	68.2 (±10.0)	61.9 (±10.7)	**0.0011**
Gender: (M:F)	172:63	26:7	NS
Hypertension, n (%)	126 (54)	17 (52)	NS
Dyslipidemia, n (%)	214 (91)	19 (58)	**0.0071**
Diabetes mellitus, n (%)	102 (43)	17 (52)	NS
PGM Activity			
PGM on admission (U/l)	11.9 (±8.6)	46.3 (±52.8)	**＜0.0001**
Myocardial Biomarkers			
CPK on admission (U/l)	67.0 (±43.8)	360.2 (±449.6)	**＜0.0001**
AST (U/l)	18.6 (±9.8)	60.2 (±67.8)	**＜0.0001**
TnT (ng/ml)	0.04 (±0.14)	1.99 (±3.68)	**＜0.0001**
Thrombosis Biomarkers			
D-Dimer (mg/dl)	1.5 (±2.4)	0.8 (±0.6)	NS
PAI-1 (ng/ml)	16.1 (±9.1)	36.7 (±40.9)	**＜0.0001**
Inflammatory Biomarkers			
CRP (mg/dl)	0.3 (±0.8)	0.3 (±0.5)	NS
PTX3 (ng/ml)	3.6 (±1.6)	7.5 (±8.3)	**0.0134**

M, male; F, female; PGM, phosphoglucomutase, CPK, creatinine phosphokinase; AST, aspartate aminotransferase; TnT, cardiac troponin T; PAI-1, total plasminogen-activator inhibitor type 1; CRP, C-reactive protein; PTX3, pentraxin 3.
Values are expressed as a number or as the mean ± standard deviation (SD).
A P value of less than 0.05 was considered significant (NS, not significantly different).

Table 2 Comparison of PGM activity and variation in thrombosis among STEMI patients

	Red Thrombosis (n = 20)	**White Thrombosis** (n = 13)	**P-value**
Baseline Characteristics			
Age (years)	61.2 (±10.9)	63.2 (±10.6)	NS
Hypertension, n (%)	11 (55)	6 (46)	NS
Dyslipidemia, n (%)	13 (65)	6 (46)	NS
Diabetes mellitus, n (%)	12 (60)	5 (38)	NS
Onset-to-balloon time (min)	21.9 (±13.1)	52.2 (±35.9)	**0.0042**
Lesion Characteristics			
Location (RCA:LAD:LCx) (n)	8:10:2	6:2:7	NS
TIMI grade (0:1:2:3) (n)	16:2:2:0	13:0:0:0	NS
Rentrop grade (0:1:2:3) (n)	17:3:0:0	9:4:0:0	NS
Thrombosis Biomarkers			
D-Dimer (mg/dl)	0.9 (±0.7)	0.8 (±0.6)	NS
PAI-1 (ng/ml)	34.2 (±51.1)	40.4 (±23.2)	**0.0469**
PGM Activity			
PGM on admission (U/l)	49.2 (±53.8)	42.2 (±53.3)	NS
Peak PGM (U/l)	187.9 (±110.9)	128.6 (±90.8)	NS
Myocardial Biomarkers			
CPK on admission (U/l)	242.4 (±348.6)	541.5 (±536.6)	NS
Peak CPK (U/l)	3898.2 (±2325.5)	3976.2 (±4170.0)	NS
AST (U/l)	47.7 (±53.4)	79.3 (±84.2)	NS
TnT (ng/ml)	2.52 (±4.44)	0.92 (±0.92)	NS
Inflammatory Biomarkers			
CRP (mg/dl)	0.2 (±0.2)	0.4 (±0.7)	NS
PTX3 (ng/ml)	5.3 (±5.3)	11.5 (±11.1)	NS

For the abbreviations, see Table 1.

Table 3 Comparison of PGM activity and established biomarkers in AMI patients with or without shock

	AMI with shock (n = 9)	AMI without shock (n = 62)	P-value
Baseline Characteristics			
Age (years)	65.6 (±11.6)	63.5 (±11.6)	NS
Hypertension, n (%)	4 (44)	34 (55)	NS
Dyslipidemia, n (%)	1 (11)	26 (42)	NS
Diabetes mellitus, n (%)	3 (33)	26 (42)	NS
Onset-to-balloon time (min)	49.4 (±36.2)	44.4 (±45.3)	NS
Death, n (%)	6 (67)	3 (5)	**＜0.0001**
Lesion Characteristics			NS
Thrombosis n (%)	4 (44)	33 (53)	NS
Location (RCA:LAD:LCx) (n)	4:5:0	25:27:10	NS
TIMI grade (0:1:2:3) (n)	8:1:0:0	40:6:8:7	NS
Rentrop grade (0:1:2:3) (n)	7:2:0:0	52:10:0:0	NS
Thrombosis Biomarkers			
D-Dimer (mg/dl)	9.7 (±11.9)	1.0 (±1.7)	**＜0.0001**
PAI-1 (ng/ml)	97.5 (±48.8)	45.4 (±41.2)	**0.0032**
PGM Activity			
PGM on admission (U/l)	148.1 (±133.3)	45.6 (±56.6)	**0.0074**
Peak PGM (U/l)	368.1 (±182.3)	143.8 (±103.6)	**0.0023**
Myocardial Biomarkers			
CPK on admission (U/l)	588.3 (±988.5)	460.8 (±585.1)	NS
Peak CPK (U/l)	7453.6 (±3654.0)	3627.1 (±3175.1)	**0.0036**
AST (U/l)	131.9 (±157.1)	64.3 (±157.1)	**0.0321**
TnT (ng/ml)	4.49 (－)	1.48 (±2.91)	NS
Inflammatory Biomarkers			
CRP (mg/dl)	1.2 (±2.6)	0.3 (±0.5)	NS
PTX3 (ng/ml)	None date	7.5 (±8.3)	－

STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; RCA, right coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery, TIMI, thrombolysis in myocardial infarction trial. For the other abbreviations, see Table 1.

important prognostic information about arteriosclerosis[30]. PTX3 is also a new biomarker for the acute-phase inflammatory response[31]. Our results showed that the levels of PTX3 were significantly higher, whereas CRP was not significantly different; thus, the arteriosclerosis of the AMI patients with thrombosis appeared to be related, at least to a limited extent, to coronary inflammation.

Thus, PGM activity might increase with myocardial damage, the formation of thrombus, and the presence of inflammation associated with coronary plaque rupture.

3. The relationship between the PGM activity and shock

In Table 3, the myocardial and thrombosis markers were higher in AMI patients with shock, with the exception of CPK and TnT on admission. The CPK and TnT levels on admission were not significantly different between AMI patients with or without shock, whereas the PGM activity in AMI patients with shock was 3 times that of AMI patients without shock on admission. The patients with shock had multiple organ failure. The isoform PGM1 has been reported to be found in multiple organs[13] while PGM 2 and 3 have been found to be expressed only in some organs. The PGM activity in AMI patients with shock may have resulted from the expression of a number of PGM isoforms increasing during multiple organ failure. The high PGM activity in AMI patients with shock indicated that PGM might be a strong predictor at the early phase of the shock state in AMI patients

and might be associated with the prognosis in AMI patients. In terms of inflammatory biomarkers, CRP is associated with the occurrence of cardiac shock and has prognostic value in cardiac shock complicating AMI[29]. In our study, the CRP levels in patients with shock tended to be higher than those in patients without shock; thus, CRP also had prognostic value in cardiac shock complicating AMI.

4. Study limitation and further study

In present study, we could not measure the PGM activity of various isoforms because the measuring methods have not yet been established. Further study is needed to show that the PGM isoform will be useful in the differentiation of myocardial damage, thrombus formation, and patients with shock.

Conclusion

PGM activity might be useful in diagnostic application in acute coronary thrombosis in AMI and a useful predictive marker in the prognosis of AMI patients.

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Disclosures

Conflict of interest and financial disclosure: none.

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