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ORIGINAL ARTICLE

Prognostic impact of pulse pressure at admission on in-hospital outcome after primary percutaneous coronary intervention for acute myocardial infarction

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Abstract Data regarding relationship between pulse pressure (PP) at admission and in-hospital outcome in patients with acute myocardial infarction (AMI) undergoing primary percutaneous coronary intervention (PCI) are still lacking. A total of 1413 primary PCI-treated AMI patients were classified into quintiles based on admission PP (<40, $n = 280$; 40–48, $n = 276$; 49–57, $n = 288$; 58–70, $n = 288$; and ≥71 mmHg, $n = 281$). The patients with PP < 40 mmHg tended to have higher prevalence of male, smoking, and Killip class ≥3 at admission; right coronary artery, left main trunk (LMT), or multivessels as culprit lesions; larger number of diseased vessels; lower Thrombolysis in Myocardial Infarction (TIMI) grade in the infarct-related artery before/after primary PCI; and higher value of peak creatine phosphokinase concentration. Patients with PP < 40 mmHg had highest mortality, while patients with PP 49–57 mmHg had the lowest: 11.8 % (<40), 7.2 % (40–48), 2.8 % (49–57), 5.9 % (58–70), and 6.0 % (≥71 mmHg). On multivariate analysis, Killip class ≥3 at admission, LMT or multivessels as culprit lesions, chronic kidney disease, and age were the independent positive predictors of the in-hospital mortality, whereas admission PP 49–57 mmHg, hypercholesterolemia, and TIMI 3 flow before/after PCI were the negative ones, but admission PP < 40 mmHg was not. These results suggest that admission PP 49–57 mmHg might be correlated with better in-hospital prognosis in Japanese AMI patients undergoing primary PCI.

Keywords Acute myocardial infarction · Pulse pressure · Prognosis

On behalf of the AMI-Kyoto Multi-Center Risk Study Group. The institutions and principal investigators of AMI-Kyoto Multi-Center Risk Study Group are listed in the Appendix.

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Introduction

Accumulating evidence have indicated that high pulse pressure (PP), which depends on ventricular stroke volume, large arterial stiffness, and timing of wave reflections, is an independent predictor for mortality and cardiovascular events in a variety of populations, such as hypertensive patients [1–3], patients undergoing percutaneous/surgical revascularization [4], patients with left ventricular systolic dysfunction after myocardial infarction [5], and the general population [6, 7]. By contrast, several reports have shown that low PP is associated with increased mortality in patients with advanced or decompensated heart failure [8–10] and in patients with heart failure after myocardial

infarction [11]. In addition, a recent report has indicated a relationship between low PP at peak exercise and cardiac mortality in patients with systolic heart failure [12]. However, the relationship between admission PP, clinical manifestations, and in-hospital outcome in acute myocardial infarction (AMI) patients undergoing primary percutaneous coronary intervention (PCI) remains uncertain. The AMI-Kyoto Multi-Center Risk Study, a large multicenter observational study in which collaborating hospitals in Kyoto Prefecture have collected demographic, procedural, and outcome data on AMI patients, was established in 2000 in order to analyze these data and establish an emergency-hospital network for heart diseases in Kyoto [13]. The purpose of the present study was therefore to examine the prognostic impact of admission PP on in-hospital outcome in Japanese AMI patients undergoing primary PCI, using data from the AMI-Kyoto Multi-Center Risk Study.

Patients and methods

Patient population

From July 2006 to December 2010, 1636 consecutive patients with a diagnosis of AMI, who were admitted to AMI-Kyoto Multi-Center Risk Study Group Hospitals within 24 h after the onset of AMI, were enrolled in the present study. Of these, excluding cases with cardiopulmonary arrest on arrival, 1443 patients underwent primary PCI, of whom data on clinical background and PP at admission were available in 1413. The 1413 primary PCI-treated AMI patients were classified into quintiles based on PP at hospital admission (mmHg: <40, $n = 280$; 40–48, $n = 276$; 49–57, $n = 288$; 58–70, $n = 288$; and ≥ 71, $n = 281$). Pulse pressure at admission was calculated as the difference between admission systolic blood pressure (SBP) and admission diastolic blood pressure (DBP). Admission SBP and DBP were defined as SBP and DBP first recorded in supine position or sitting position just after presentation to the emergency room or the outpatient clinic. We retrospectively compared clinical background, coronary risk factors, angiographic findings, acute results of primary PCI, and in-hospital prognosis among the five groups. The diagnosis of AMI required the presence of two of the following three criteria: (1) characteristic clinical history, (2) serial changes on the electrocardiogram suggesting infarction (Q-waves) or injury (ST-segment elevations), and (3) transient increase in cardiac enzymes to more than twofold the normal laboratory value.

Data collection

The patients' demographic information, cardiovascular history, and risk factors (i.e., smoking, hypercholesterolemia, hypertension, and diabetes mellitus) were recorded. Hypercholesterolemia was defined as total cholesterol ≥ 220 mg/dl or the use of cholesterol-lowering agents; hypertension was defined as systemic blood pressure $\geq 140/90$ mmHg or the use of antihypertensive treatment; diabetes mellitus was defined as fasting blood sugar ≥ 126 mg/dl or the use of specific treatment. Chronic kidney disease (CKD) was defined as estimated glomerular filtration rate (eGFR) of smaller than 60 ml/min/1.73 m^2, and eGFR was calculated using the Japanese equation: eGFR (ml/min/1.73 m^2) = 194 $\times$ serum creatinine$^{(-1.094)}$ $\times$ age$^{(-0.287)}$ $\times$ 0.739 (if female) [14]. All in-hospital data were transmitted to the center located at the Department of Cardiovascular Medicine in Kyoto Prefectural University School of Medicine for analysis. The study protocol was approved by each hospital's ethics committee.

Emergency coronary angiography and reperfusion therapy

Emergency coronary angiography (CAG) was performed using the standard technique. The coronary flow in the infarct-related artery (IRA) was graded according to the classification used in the Thrombolysis in Myocardial Infarction (TIMI) trial. Significant coronary artery stenosis was defined as at least a 75 % reduction in the internal diameter of the right, left anterior descending, or left circumflex coronary arteries and their major branches, or a 50 % reduction in the internal diameter of the left main trunk (LMT). Nonsignificant stenosis was defined as coronary arterial narrowing less than a significant stenosis. Multivessels as culprit lesions was defined as simultaneous thromboses of multiple coronary arteries in the initial CAG. After the culprit lesions were ascertained by CAG, primary PCI was subsequently performed according to standard techniques, with the final decision about the appropriate strategy in each patient left to the judgment of the physician in charge. Primary PCI was defined as a coronary intervention as the initial mode of revascularization therapy, performed at the time of presentation for ST-elevation myocardial infarction (MI) or non-ST-elevation MI including posterior MI, multivessel MI, and LMT-culprit MI, without prethrombolytic therapy.

End-point measures

In-hospital death was defined as all-cause death during the admission period.

Statistics

Data are expressed as mean $\pm$ standard deviation (SD). Five groups were compared using the Chi-square test for

discrete variables and the Kruskal–Wallis test or one-way analysis of variance (ANOVA), followed by Scheffe's test as the post hoc test for continuous variables, as appropriate, according to standard statistical methods. The odds ratio and 95 % confidence intervals assessing the risk of in-hospital death were estimated by univariate and multivariate analyses using a logistic regression model. Potent variables with a P value of less than 0.05 on the univariate analyses were entered into a multivariate analysis. In the logistic regression analysis, TIMI flow grade in the IRA was categorized into two groups: grade 3 and grade ≤2 or unknown, and CKD was categorized into two groups: with CKD and without CKD or without eGFR value. In all analyses, statistically significance was accepted at $P < 0.05$.

Results

Patient characteristics and risk factors

The clinical characteristics and risk factors among the five groups are summarized in Table 1. Age and gender, and frequency of smoking, hypertension, diabetes mellitus, and Killip class ≥3 at admission were significantly different among the five groups. The patients with PP <40 mmHg tended to be male and have lower SBP, lower DBP, lower heart rate, and larger prevalence of smoking and Killip class ≥3 at admission, while the patients with PP ≥71 mmHg had higher age and higher frequency of female, hypertension, and diabetes mellitus.

Angiographic data

Table 2 shows the emergency CAG data for the five groups. The distribution of culprit coronary artery and the number of diseased vessels were significantly different among the five groups. The patients with PP <40 mmHg were more likely to have right coronary artery (RCA), LMT, or multivessels as culprit lesions as well as larger number of diseased vessels or diseased LMT on initial CAG.

Results of coronary intervention

Table 3 shows the results of primary PCI in the five groups. Data on TIMI grade in the IRA were available in 279 of the 280 patients with PP <40 mmHg, 271 of the 276 patients with PP 40–48 mmHg, 283 of the 288 patients with PP 49–57 mmHg, 285 of the 288 patients with PP 58–70 mmHg, and 279 of the 281 patients with PP ≥71 mmHg. The distributions of TIMI grade in the IRA before/after primary PCI were significantly different among the five groups. The patients with PP <40 mmHg tended to have lower TIMI grade in the IRA before/after primary PCI and

Table 1 Clinical characteristics by pulse pressure at admission

	Pulse pressure at admission quintile, mmHg					*P* value
	<40 (n = 280)	40–48 (n = 276)	49–57 (n = 288)	58–70 (n = 288)	≥71 (n = 281)	
Age (mean years ± SD)	67.4 ± 12.8	65.7 ± 12.5	67.5 ± 12.3	68.6 ± 12.2	71.0 ± 11.7	<0.001
Male (%)	233 (83.2)	219 (79.3)	220 (76.4)	201 (69.8)	179 (63.7)	<0.001
Previous MI (%)	33 (11.8)	34 (12.3)	38 (13.2)	30 (10.4)	24 (8.5)	0.441
Risk factors						
Smoking (%)	134 (47.9)	126 (45.7)	125 (43.4)	111 (38.5)	101 (35.9)	0.024
Hypercholesterolemia (%)	121 (43.2)	120 (43.5)	122 (42.4)	125 (43.4)	121 (43.1)	0.999
Hypertension (%)	136 (48.6)	145 (52.5)	167 (58.0)	188 (65.3)	198 (70.5)	<0.001
Diabetes mellitus (%)	66 (23.6)	70 (25.4)	75 (26.0)	100 (34.7)	111 (39.5)	<0.001
eGFR (ml/min/1.73 m^2)	66.6 ± 35.4	68.5 ± 24.4	70.3 ± 21.3	69.5 ± 24.1	69.9 ± 37.1	0.576
CKD (%)	115 (41.8)	91 (33.1)	89 (31.0)	94 (32.9)	96 (34.4)	0.071
Systolic blood pressure (mmHg)	102 ± 23	122 ± 20	132 ± 21	147 ± 20	164 ± 24	<0.001
Diastolic blood pressure (mmHg)	71 ± 22	78 ± 20	80 ± 21	83 ± 20	81 ± 20	<0.001
Heart rate (beats/min)	72.4 ± 23.1	73.5 ± 21.7	73.3 ± 20.7	78.3 ± 20.9	77.0 ± 19.2	0.002
Killip 3/4 (%)	60 (21.4)	34 (12.3)	24 (8.3)	30 (10.4)	26 (9.3)	<0.001

Data on eGFR at admission were available in 275 of the 280 patients with pulse pressure (PP) <40 mmHg, 275 of the 276 patients with PP 40–48 mmHg, 287 of the 288 patients with PP 49–57 mmHg, 286 of the 288 patients with PP 58–70 mmHg, and 279 of the 281 patients with PP ≥71 mmHg. Data on admission heart rate were available in 273 of the 280 patients with PP <40 mmHg, 275 of the 276 patients with PP 40–48 mmHg, 286 of the 288 patients with PP 49–57 mmHg, 280 of the 288 patients with PP 58–70 mmHg, and 274 of the 281 patients with PP ≥71 mmHg

MI myocardial infarction, *eGFR* estimated glomerular filtration rate, *CKD* chronic kidney disease, *SBP* systolic blood pressure, *DBP* diastolic blood pressure

Table 2 Angiographic findings by pulse pressure at admission

	Pulse pressure at admission quintile, mmHg					*P* value
	<40 (*n* = 280)	40–48 (*n* = 276)	49–57 (*n* = 288)	58–70 (*n* = 288)	≥71 (*n* = 281)	
Culprit lesions						
RCA (%)	127 (45.4)	107 (38.8)	112 (38.9)	84 (29.2)	73 (26.0)	<0.001
LAD (%)	96 (34.3)	121 (43.8)	132 (45.8)	148 (51.4)	140 (49.8)	
LCx (%)	25 (8.9)	35 (12.7)	32 (11.1)	44 (15.3)	58 (20.6)	
LMT (%)	16 (5.7)	7 (2.5)	2 (0.7)	8 (2.8)	2 (0.7)	
Multivessels (%)	16 (5.7)	6 (2.2)	8 (2.8)	4 (1.4)	8 (2.8)	
SVG	0 (0.0)	0 (0.0)	2 (0.7)	0 (0.0)	0 (0.0)	
No. of diseased vessels						
1 (%)	156 (55.7)	172 (62.3)	170 (59.0)	167 (58.0)	169 (60.1)	0.013
2 (%)	72 (25.7)	69 (25.0)	88 (30.6)	79 (27.4)	79 (28.1)	
3 (%)	38 (13.6)	30 (10.9)	29 (10.1)	34 (11.8)	32 (11.4)	
LMT (%)	14 (5.0)	5 (1.8)	1 (0.3)	8 (2.8)	1 (0.4)	

CAG coronary angiography, *RCA* right coronary artery, *LAD* left anterior descending coronary artery, *LCx* left circumflex coronary artery, *LMT* left main trunk, *SVG* saphenous vein graft

Table 3 Results of coronary intervention by pulse pressure at admission

	Pulse pressure at admission quintile, mmHg					*P* value
	<40 (*n* = 280)	40–48 (*n* = 276)	49–57 (*n* = 288)	58–70 (*n* = 288)	≥71 (*n* = 281)	
Pre-TIMI grade						
0	188 (67.4)	162 (59.8)	169 (59.7)	162 (56.8)	133 (47.7)	<0.001
1	32 (11.5)	32 (11.8)	25 (8.8)	32 (11.2)	36 (12.9)	
2	31 (11.1)	38 (14.0)	51 (18.0)	44 (15.4)	49 (17.6)	
3	28 (10.0)	34 (12.5)	38 (13.4)	47 (16.5)	61 (21.9)	
Post-TIMI grade						
0	4 (1.4)	2 (0.7)	5 (1.8)	2 (0.7)	4 (1.4)	0.011
1	7 (2.5)	1 (0.4)	4 (1.4)	1 (0.4)	0 (0.0)	
2	22 (7.9)	14 (5.2)	7 (2.5)	20 (7.0)	11 (3.9)	
3	246 (88.2)	254 (93.7)	267 (94.3)	262 (91.9)	264 (94.6)	
Stent (%)	245 (87.5)	250 (90.6)	251 (87.2)	248 (86.1)	238 (84.7)	0.316
IABP (%)	63 (22.5)	35 (12.7)	28 (9.7)	33 (11.5)	22 (7.8)	<0.001
PCPS (%)	12 (4.3)	2 (0.7)	2 (0.7)	3 (1.0)	1 (0.4)	<0.001
Pacing (%)	45 (16.1)	30 (10.9)	28 (9.7)	18 (6.3)	12 (4.3)	<0.001

Data on TIMI grade were available in 279 of the 280 patients with pulse pressure (PP) <40 mmHg, 271 of the 276 patients with PP 40–48 mmHg, 283 of the 288 patients with PP 49–57 mmHg, 285 of the 288 patients with PP 58–70 mmHg, and 279 of the 281 patients with PP ≥71 mmHg

PCI percutaneous coronary intervention, *TIMI* Thrombolysis in Myocardial Infarction, *IABP* intra-aortic balloon pumping, *PCPS* percutaneous cardiopulmonary support

higher frequency of intra-aortic balloon pumping, percutaneous cardiopulmonary support, and temporary pacing during procedures and/or the admission period.

In-hospital outcomes

Table 4 shows the in-hospital prognoses in the five groups. The patients with lower admission PP were likely to have a higher value of peak creatine phosphokinase (CK) concentration. Patients with PP <40 mmHg had the highest mortality, while patients with PP 49–57 mmHg had the lowest: 11.8 % (<40 mmHg), 7.2 % (40–48 mmHg), 2.8 % (49–57 mmHg), 5.9 % (58–70 mmHg), and 6.0 % (≥71 mmHg) ($P < 0.001$). The relationship between PP and cardiac-related death was similar to that between PP and all-cause death. Death ascribed to cardiogenic shock and congestive heart failure were common in the patients with PP <40 mmHg. To assess the contribution of clinical

Table 4 In-hospital outcomes by pulse pressure at admission

	Pulse pressure at admission quintile, mmHg					*P* value
	<40 (*n* = 280)	40–48 (*n* = 276)	49–57 (*n* = 288)	58–70 (*n* = 288)	≥71 (*n* = 281)	
Peak CK (IU/l)	4143.3 ± 6377.7	3287.4 ± 2927.0	2571.8 ± 2270.5	2520.5 ± 2619.8	2180.8 ± 2192.4	<0.001
Death (%)	33 (11.8)	20 (7.2)	8 (2.8)	17 (5.9)	17 (6.0)	<0.001
Cardiac-related (%)	20 (7.1)	13 (4.7)	6 (2.1)	10 (3.5)	9 (3.2)	0.030
Shock	11	5	5	7	5	
Heart failure	7	4	0	0	2	
Rupture	2	4	1	3	1	
Vf	0	0	0	0	1	
Noncardiac-related (%)	13 (4.6)	7 (2.5)	2 (0.7)	7 (2.4)	8 (2.8)	0.067

Data on CK value were available in 275 of the 280 patients with pulse pressure (PP) <40 mmHg, 273 of the 276 patients with PP 40–48 mmHg, 287 of the 288 patients with PP 49–57 mmHg, 284 of the 288 patients with PP 58–70 mmHg, and 273 of the 281 patients with PP ≥71 mmHg

CK creatine phosphokinase, *Vf* ventricular fibrillation

background, risk factors, angiographic findings, results of primary PCI, and PP at admission, univariate and multivariate logistic regression analyses were developed for overall death during hospitalization. Potent variables included age, gender, medical history (previous MI, smoking, hypercholesterolemia, hypertension, diabetes mellitus, and CKD), angiographic findings (multivessels or LMT as culprit lesions, and number of diseased vessels ≥2 or diseased LMT), PCI results (TIMI-3 flow before/immediately after primary PCI, and stent usage), Killip class ≥3 at admission, admission SBP, admission DBP, and PP <40 mmHg, PP 40–48 mmHg, PP 49–57 mmHg, PP 58–70 mmHg, or PP ≥71 mmHg at admission. On univariate testing, except for hypertension ($P = 0.6545$), PP 40–48 mmHg ($P = 0.6990$), PP 58–70 mmHg ($P = 0.5336$), and PP ≥71 mmHg (0.6148), all variables mentioned above were associated with in-hospital death. Excluding hypertension, all the available variables described above and admission PP <40 mmHg or admission PP 49–57 mmHg were therefore entered into multivariate models. On multivariate analysis, Killip class ≥3 at admission, LMT or multivessels as culprit lesions, CKD, and age were the independent positive predictors of the in-hospital mortality, whereas admission PP 49–57 mmHg, hypercholesterolemia, and TIMI-3 flow before/after PCI were the negative ones (Table 5A, B). By contrast, admission PP <40 mmHg was not tightly associated with in-hospital death (Table 5B).

Discussion

The major findings of the present multicenter study are as follows. Among AMI patients undergoing primary PCI, patients with PP <40 mmHg had the highest mortality, while patients with PP 49–57 mmHg had the lowest mortality: 11.8 % (<40 mmHg), 7.2 % (40–48 mmHg), 2.8 % (49–57 mmHg), 5.9 % (58–70 mmHg), and 6.0 % (≥71 mmHg) ($P < 0.001$). On multivariate analysis, Killip class ≥3 at admission, LMT or multivessels as culprit lesions, CKD, and age were the independent positive predictors of the in-hospital mortality, whereas admission PP 49–57 mmHg, hypercholesterolemia, and TIMI-3 flow before/after PCI were the negative ones, but admission PP <40 mmHg was not.

This study is the first to investigate the prognostic impact of PP at admission on in-hospital prognosis among AMI patients undergoing primary PCI. Pulse pressure reflects left ventricular stroke volume, compliance of the aorta and its major branches, and reflected pressure waves [15]. The wide PP with age in healthy population relates to falls in aortic compliance [16], while malignant cycle among high PP, enhanced aortic stiffness, and progression of atherosclerosis has been proposed [15]. High PP enhances vascular endothelial/structural damage, and results in atherosclerosis progression accompanied by increased aortic stiffness, leading to higher PP. In addition, high PP might worsen myocardial ischemia through increased afterload and reduced coronary perfusion [17]. Indeed, numerous studies from the United States and European investigators have demonstrated that wide PP is a significant risk factor for mortality and cardiovascular events in various populations including patients with coronary artery disease [1–7]. However, the relationship between admission PP and in-hospital prognosis in AMI patients undergoing primary PCI remains to be elucidated. A recent report has indicated that initial low PP was associated with in-hospital mortality in patients with acute coronary syndrome, in which CAG was performed only in approximately 20 % of the study population [18]. In the present report concerning the primary PCI-treated AMI patients, the patients with admission PP <40 mmHg had a

Table 5 Predictors of in-hospital mortality in the study patients (multivariate logistic regression analysis)

	OR	95 % CI	*P* value
A			
Killip 3/4	7.344	4.268–12.639	<0.0001
Multivessels or LMT as culprit	2.779	1.311–5.890	0.0076
Age	1.069	1.039–1.099	<0.0001
Pulse pressure 49–57 mmHg	0.373	0.164–0.848	0.0186
TIMI-3 before PCI	0.290	0.106–0.795	0.0162
TIMI-3 after PCI	0.247	0.129–0.473	<0.0001
Hypercholesterolemia	0.573	0.334–0.984	0.0434
CKD	1.657	0.973–2.819	0.0628
SBP	0.993	0.981–1.006	0.2946
DBP	1.000	0.981–1.020	0.9737
No. of diseased vessels ≥2 or LMT	1.086	0.624–1.892	0.7707
Male	0.717	0.412–1.249	0.2403
Previous MI	1.400	0.707–2.771	0.3339
B			
Killip 3/4	7.433	4.330–12.761	<0.0001
Multivessels or LMT as culprit	2.855	1.342–6.071	0.0064
CKD	1.706	1.006–2.892	0.0474
Age	1.067	1.037–1.097	<0.0001
Hypercholesterolemia	0.566	0.331–0.969	0.0381
TIMI-3 before PCI	0.278	0.101–0.766	0.0133
TIMI-3 after PCI	0.233	0.122–0.445	<0.0001
Pulse pressure <40 mmHg	1.145	0.539–2.432	0.7249
SBP	0.996	0.980–1.012	0.6154
DBP	0.998	0.977–1.019	0.8292
No. of diseased vessels ≥2 or LMT	1.033	0.596–1.793	0.9068
Male	0.733	0.422–1.274	0.2714
Previous MI	1.299	0.660–2.554	0.4488

(A) Admission pulse pressure (PP) 49–57 mmHg was included as a variable. (B) Admission PP <40 mmHg was included as a variable

OR odds ratio, *CI* confidence intervals, *SBP* systolic blood pressure, *CKD* chronic kidney disease, *PCI* percutaneous coronary intervention, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *LMT* left main trunk, *MI* myocardial infarction

significantly higher in-hospital mortality rate than other quintiles; however, admission PP <40 mmHg was not an independent predictor of in-hospital death. Interestingly, the patients with PP 49–57 mmHg had the lowest in-hospital mortality rate, and admission PP 49–57 mmHg was an independent predictor of in-hospital survival.

Lower PP on admission tended to be correlated with worse in-hospital prognosis in the present report. Contrary to the relatively stable/chronic conditions such as hypertension, diabetes, and postmyocardial infarction [1–3, 19–21], in which high PP is related to mortality and morbidity, some recent studies have shown that low PP is associated with increased mortality among patients with advanced or decompensated heart failure [8–10] and among patients with heart failure after AMI [11]. In the acute myocardial disorders such as acute heart failure and AMI, lower PP might reflect reduced stroke volume and circulatory impairment rather than decreased arterial stiffness, and lead to increased mortality. Even in the present report, although data regarding left ventricular function were missing, death due to cardiogenic shock or heart failure mainly contribute to higher in-hospital mortality in those patients with lower PP on admission.

On the other hand, the mechanism by which admission PP 49–57 mmHg was tightly associated with better in-hospital outcome remains unclear. Although based on the peak CK value, the extent of myocardial damage was inversely correlated with admission PP; the patients with admission PP 49–57 mmHg had lesser prevalence of deaths ascribed to heart failure and ventricular rupture, compared with the patients with lower PP or those with higher PP. In the present study population, admission PP was positively correlated with admission SBP. Thus, although data regarding SBP and PP during the admission period were missing, there is a possibility that heart failure due to afterload mismatch might be prone to occur in the patients with admission PP ≥71 mmHg. In addition, not only the patients with lower PP characterized by larger myocardial damage but also the patients with higher PP characterized by higher prevalence of hypertension and female gender might be at high risk of ventricular rupture [22, 23]. Another potential explanation is that relatively lesser frequency of admission CKD might account for lower in-hospital mortality in patients with admission PP 49–57 mmHg, because among AMI patients undergoing PCI admission CKD has been shown to be tightly associated with in-hospital mortality as well as 30-day and 1-year mortality [24, 25], while CKD at the first visit to the outpatient clinic after discharge has also been reported to be relevant to long-term prognosis [26].

Study limitations

First, this is a retrospective observational analysis of a relatively small number of patients. Second, data regarding clinical background and angiographic results of primary PCI were not available for the whole study population. Third, we did not have enough detailed data regarding left ventricular function, elapsed time (the time interval between symptom onset and hospital admission), the time interval between blood pressure measurement and subsequent primary PCI, ischemic time, and in-hospital medical treatment, which might be significant predictors of in-hospital death. In particular, antihypertensive treatments in the preadmission period might significantly affect

admission PP. Fourth, "ST-elevation MI" was not distinguished from "non-ST-elevation MI". Fifth, we did not have enough data regarding nonfatal major adverse cardiovascular events during the admission period. Sixth, data concerning an individual decisive factor of primary PCI for the patients with elapsed time >12 h were lacking. Seventh, we did not have enough data about 30-day mortality as end point in the present study population.

Conclusion

The present study suggests that admission PP 49–57 mmHg might be correlated with better in-hospital prognosis among Japanese AMI patients undergoing primary PCI.

Appendix

The following institutions and principal investigators participated in the present study as the AMI-Kyoto Multi-Center Risk Study Group. Kyoto City Hospital: Okada T, Shima M, Takamiya A, Nakajima N, Matsunaga S; Kyoto Kidugawa Hospital: Miyanaga H, Nakagawa T, Matsui H, Kunieda Y; Kyoto Second Red Cross Hospital: Fujita H, Inoue K, Matsuo A, Kimura S, Sakatani T, Isodono K, Tsubakimoto Y; Social Insurance Kyoto Hospital: Yamada C, Tanabe S, Yagi T; Tanabe Central Hospital: Kusuoka S, Takechi N, Nishizawa S; Nantan General Hospital: Tatsumi T, Keira N, Nomura T; Ayabe Municipal Hospital: Shiga K, Kohno Y, Adachi Y; Maizuru Medical Center: Harada Y, Hikosaka T, Nakagami T; Kyoto Saiseikai Hospital: Yamahara Y, Ishibashi K, Takeda M; Gakkentoshi Hospital: Sakai R, Akashi K; Kouseikai Takeda Hospital: Matsumoto K, Kinoshita N, Sawanishi T, Nakamura R; Kameoka Municipal Hospital: Matsuo R; Aiseikai Yamashina Hospital: Yamamoto T; Fukuchiyama Municipal Hospital: Nishio M, Sakamoto T; Saiseikai Shiga Hspital: Nakamura T, Nakahara Y, Kurata H, Hadase M; Omihachiman Community Medical Center: Maki K, Tatsukawa H, Zen K, Kambayashi D; Kyoto Prefectural Yosanoumi Hospital: Honsho S; Kyoto Prefectural University School of Medicine: Shiraishi H, Yamano T.

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