Renal Dysfunction on Admission, Worsening Renal Function, and Severity of Acute Kidney Injury Predict 2-Year Mortality in Patients With Acute Myocardial Infarction

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Background: Recent studies have proven that initial renal dysfunction and worsening renal function during hospitalization can predict the clinical outcome of patients with acute myocardial infarction (AMI). There is limited study regarding acute kidney injury (AKI) by the RIFLE classification (Risk of renal failure, Injury to the kidney, Failure of kidney function, Loss of kidney function, and End-stage renal failure) to assess the outcome of AMI survivors.

Methods and Results: During a mean follow-up period of 635.3±204.9 days, the 2-year mortality rate was 10.6% in 613 AMI patients who survived to discharge. Adjusted Cox regression analysis revealed that left ventricular dysfunction (<40%) [hazard ratio (HR), 2.83; 95% confidence interval (CI), 1.11–7.20; P=0.029], estimated glomerular filtration rate <60 ml·min⁻¹·1.73 m⁻² on admission (HR, 4.01; 95% CI, 1.51–10.62; P=0.005), and AKI severity by RIFLE classification during first week after hospitalization (Injury: HR, 8.11; 95% CI, 2.53–26.05; P=0.001; Failure: HR, 19.28; 95% CI, 2.24–166.26; P=0.007) were independent predictors of 2-year mortality.

Conclusions: Independent of initial renal dysfunction on admission, the AKI severity by RIFLE classification may be useful in establishing the hospital discharge risk score for predicting long-term mortality in AMI patients who survive to discharge. (*Circ J* 2013; **77**: 217–223)

Key Words: Acute kidney injury; Acute myocardial infarction; Long-term outcome; RIFLE

Previous studies have reported that initial renal dysfunction on admission can predict the short- and long-term mortality of patients with acute myocardial infarction (AMI).^{1,2} On admission, AMI patients may present with impaired renal function, which may result from hemodynamic instability related to acute kidney injury (AKI) or an underlying chronic renal disease. Furthermore, during hospitalization periods ranging from several hours to days, these patients may also develop worsening renal function (WRF) because of various factors such as contrast-induced nephropathy or cardio-renal syndrome.^{3,4} The effect of WRF on mortality in AMI patients has been reported.⁵

Many different definitions of AKI are found in the literature.^{6–8} The Risk of renal failure, Injury to the kidney, Failure of kidney function, Loss of kidney function, and End-stage renal failure (RIFLE) classification was first proposed by the Acute Dialysis Quality Initiative (ADQI) group to standardize AKI study.⁶ The criteria resulting in the worst possible classifica-

tion were used. Patients were classified into 3 severity categories (risk, injury, and failure) and 2 clinical outcome categories (loss and end-stage renal disease). To date, the RIFLE classification system has been used to assess short- and long-term mortality in different critical populations.^{9–15} However, few studies have mentioned the role of AKI by RIFLE classification on AMI patients' outcomes.^{16,17} The purpose of this study was to evaluate the role of initial renal dysfunction on admission, WRF, and the RIFLE classification during hospitalization in predicting the long-term outcomes of AMI patients who survive to discharge.

Methods

Patients and Study Design

A total of 687 patients who were diagnosed with AMI and admitted to the cardiovascular department between January 2006 and December 2007 were evaluated. We excluded 74 patients,

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Table 1. Demographic and Clinical Characteristics of Patients

	Survivor (n=548)	Non-survivor (n=65)	P value
Age (years)	62.1±13.5	74.4±11.2	<0.001
Women [n (%)]	109 (19.9)	24 (37.5)	0.002
Hypertension [n (%)]	307 (56.0)	46 (70.8)	0.024
Diabetes [n (%)]	168 (30.8)	32 (49.2)	0.003
Smoking [n (%)]	291 (53.1)	27 (41.5)	0.088
Hypercholesterolemia [n (%)]	178 (32.8)	17 (26.6)	0.396
Previous history of MI [n (%)]	61 (11.1)	16 (24.6)	0.005
Peak cTn-I levels (μg/L)	15.7±39.1	11.5±19.5	0.411
LVEF (%)	55.1±15.7	48.9±17.9	0.003
eGFR on admission (ml·min ⁻¹ ·1.73m ⁻²)	70.8±26.1	39.9±26.4	<0.001
WRF during hospitalization [n (%)]	42 (7.7)	14 (21.5)	0.001
Killip class ≥3 [n (%)]	54 (9.8)	19 (29.7)	<0.001
CAD 2- or 3-vessel/total coronary angiography number (%)	228/442 (51.6)	13/22 (59.1)	0.521
STEMI [n (%)]	287 (52.3)	20 (31.3)	0.001
RIFLE classification [n (%)]			<0.001
Absence of AKI	494 (90.1)	40 (61.5)	
Risk	29 (5.3)	7 (10.8)	
Injury	17 (3.1)	6 (9.2)	
Failure	8 (1.5)	12 (18.5)	
Aspirin therapy [n (%)]	537 (98.2)	57 (87.7)	<0.001
β-blocker therapy [n (%)]	450 (82.1)	40 (61.5)	<0.001
ACEI/ARB therapy [n (%)]	378 (69.0)	39 (60.0)	0.16
PCI therapy [n (%)]	353 (64.4)	16 (24.6)	<0.001
CABG therapy [n (%)]	28 (5.1)	4 (6.2)	0.766

ACEI, angiotensin-converting enzyme inhibitors; AKI, acute kidney injury; ARB, angiotensin II receptor blockers; CABG, coronary artery bypass graft; CAD, coronary artery disease; cTn-I, cardiac troponin-I; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention; RIFLE, Risk, Injury, Failure, Loss, and End-stage kidney disease; STEMI, ST-elevation MI.

including 48 patients who died during hospitalization and 26 patients who underwent dialysis, or kidney transplantation therapy for chronic kidney disease before admission. Among the remaining 613 patients, patients who survived an AMI event were included in the study. The demographic and clinical characteristics of the patients were recorded on admission. Information regarding the laboratory and pharmacological data and interventional therapy was obtained from the hospital's computer database. This was an observational study, and the patients and their primary care physicians made all the clinical decisions. The primary endpoint of this study was all-cause mortality 2 years after an AMI. Assessment of survival status and validation of clinical outcomes were performed from the records of the outpatient clinic or by telephone interview with the patient, caregivers, or the patients' primary care physicians.

Definitions

On admission, the initial estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease formula.¹⁸ Initial renal dysfunction was defined as an eGFR <60 ml·min⁻¹·1.73m⁻². Using an established definition,^{19,20} WRF was defined on the basis of the maximal decrease in eGFR (≥25%) from the initial value at any time during the first 7 days after hospitalization. We also evaluated renal function during hospitalization by using the RIFLE classification system. The RIFLE classification was defined by comparing the change in the serum creatinine levels during the first 7 days after hospitalization with the baseline creatinine levels.²¹ The baseline creatinine levels (serum creatinine levels of the

patient up to 3 months before admission) of selected patients were retrieved from the hospital's computer database. For patients with no previous serum creatinine record, baseline creatinine levels were calculated by using the MDRD equation.²² The maximum RIFLE classification was determined by comparing the highest serum creatinine during hospitalization with the baseline creatinine level. Patients who met the RIFLE criteria were classified as the AKI group, and those who did not were classified as the non-AKI group. Patients in the AKI group were further classified into RIFLE-R (Risk, 150% increase in creatinine), RIFLE-I (Injury, 200% increase in creatinine), and RIFLE-F (Failure, 300% increase in creatinine) groups according to the maximal serum creatinine value obtained during the first 7 days after hospitalization. This study aimed to evaluate the effect of AKI severity by the RIFLE classification on long-term outcomes of AMI patients who survived to discharge. The patients who finally reached the level of either loss or end-stage renal disease were initially included in the the RIFLE-R, I or F groups.

AMI was defined as the occurrence of 2 or more of the following: chest pain, ischemic ECG change, and elevated cardiac marker. It was further classified as ST-elevation MI (STEMI) or non-STEMI (NSTEMI) according to the ECG findings. STEMI was defined as ST-segment elevation >1 mm in 2 contiguous leads. Elevated cardiac marker was defined as peak cardiac troponin I level >3-fold the upper limit of normal within 72 h after admission.

Table 2. Comparison of Baseline Clinical and Biochemical Characteristics on Admission Based on RIFLE Classification

	Non-AKI (n=534)	RIFLE-R (n=36)	RIFLE-I (n=23)	RIFLE-F (n=20)	P value
Age (years)	62.1±13.7	72.6±10.8	72.7±9.9	71.4±13.2	<0.001
Women [n (%)]	109 (20.4)	8 (22.2)	9 (39.1)	7 (35.0)	0.056
Hypertension [n (%)]	294 (55.0)	28 (77.8)	18 (78.3)	13 (65.0)	0.001
Diabetes [n (%)]	171 (32.0)	13 (36.1)	10 (43.4)	6 (30.0)	0.442
Smoking [n (%)]	287 (53.7)	18 (50.0)	5 (21.7)	8 (40.0)	0.021
Previous history of MI [n (%)]	68 (12.7)	3 (8.3)	3 (13.0)	3 (15.0)	0.87
Killip class >1 [n (%)]	116 (21.7)	16 (44.4)	10 (43.5)	15 (75.0)	<0.001
LVEF (%)	55.3±15.9	52.6±16.1	45.5±14.4	45.5±17.0	0.001
Peak cTn-I (ratio of the upper limit of normal)	34.3±81.6	54.5±158.8	94.6±186.4	41.2±72.6	0.017
Cholesterol (mg/dl)	186.6±44.4	164.2±45.1	163.3±26.9	178.4±74.1	0.003
HDL (mg/dl)	40.1±10.0	37.1±11.1	37.3±7.7	37.1±12.4	0.132
Triglyceride (mg/dl)	161.3±113.6	133.9±64.6	120.4±44.4	167.0±134.5	0.173
Mean blood pressure (mmHg)	100.4±21.6	92.2±29.0	98.2±21.1	92.1±20.0	0.07
WBC (1,000/ μ l)	10.6±4.9	10.6±3.8	11.4±4.8	12.9±6.5	0.18
hs-CRP (mg/L)	34.8±43.9	39.5±45.7	114.8±117.2	94.2±83.5	<0.001
Uric acid (mg/dl)	6.3±1.9	8.8±2.9	8.1±2.7	7.0±1.9	<0.001
eGFR on admission ($\text{ml} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2}$)	72.6±25.3	37.9±15.8	39.1±17.9	18.3±16.2	<0.001
PCI therapy [n (%)]	323 (60.5)	21 (58.3)	15 (65.2)	10 (50.0)	0.758
CABG therapy [n (%)]	26 (4.9)	3 (8.3)	3 (13.0)	0 (0.0)	0.553

HDL, high-density lipoprotein; hs-CRP, high-sensitivity C-reactive protein; WBC, white blood cells. Other abbreviations as in Table 1.

Statistical Analysis

Continuous variables are represented as mean±standard deviation, while categorical variables are expressed as frequencies and group percentages. The statistical normality was assessed using the Kolmogorov-Smirnov test. The χ^2 test was used to compare categorical variables. The ANOVA and the t-tests were used for variables with normal distributions, and the Mann-Whitney U test was used for other data.

The 2-year event-free survival curves were estimated by Kaplan-Meier method and compared by log-rank test. Incidence of death (deaths per 10,000 person-days) was calculated for each category. Univariate- and multivariate-adjusted hazard ratios (HR) were obtained by using the Cox regression analysis, with adjustment for multiple covariates that were potentially associated with patient survival. Multivariate binary logistic regression analysis was used to find out which patient group's renal function would worsen during the first 7 days after hospitalization. A P value less than 0.05 was considered statistically significant, and analysis was performed with the statistical software package (SPSS 17.0; Chicago, IL, USA) for Windows.

Results

Comparison of Demographic and Clinical Characteristics of 2-Year Survivors and Non-Survivors (Table 1)

There were no significant differences between the patients in the survivor and non-survivor groups for smoking, hypercholesterolemia, 2- or 3-vessel coronary artery disease, peak cardiac troponin I levels, angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker therapy, and coronary artery bypass graft therapy. More patients in the non-survivor group were elderly women with diabetes mellitus, hypertension, previous MI history, impaired left ventricular ejection fraction (LVEF <40%), NSTEMI and Killip class ≥ 3 , as well as patients who had not received aspirin therapy, β -blocker therapy, or interventional therapy, and patients with eGFR $<60 \text{ ml} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2}$ on admission, WRF, and severe RIFLE classification

during hospitalization. The reasons for patients not undergoing percutaneous coronary intervention (PCI) in the non-survivor group were NSTEMI with low TIMI risk score (28 patients), multiple comorbidities (12 patients) and disinclination to undergo coronary angiography (9 patients).

Comparison of Baseline Clinical and Biochemical Characteristics on Admission Based on the RIFLE Classification (Table 2)

We further analyzed the baseline clinical and biochemical characteristics based on the RIFLE classification. There were no significant differences between the 4 groups of patients for sex, diabetes mellitus, previous history of MI, mean blood pressure on admission, white blood count, high-density lipoprotein, triglyceride, PCI and coronary bypass therapy. AMI patients with severe AKI were older with hypertension, advanced Killip class, higher cardiac troponin I level, and impaired heart and kidney function on admission. In addition, the levels of inflammatory markers on admission, including high-sensitivity C-reactive protein (hs-CRP) and uric acid, were significantly higher in patients with AKI by the RIFLE classification.

Independent Predictors of 2-Year All-Cause Mortality

In the univariate analysis for survival, age >65 years, female sex, diabetes mellitus, hypertension, previous history of MI, cardiac troponin I >3-fold the upper limit of normal, Killip class ≥ 3 , LVEF <40%, NSTEMI, hyperuricemia, eGFR $<60 \text{ ml} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2}$ on admission, WRF during the first week after hospitalization, RIFLE classification, and no administration of aspirin or β -blocker, or PCI were major predictors of survival (Table 3). Risk factors other than the RIFLE classification were adjusted in Cox regression model 1, and eGFR $<60 \text{ ml} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2}$ on admission [HR, 5.58; 95% confidence interval (CI), 2.25–13.85; $P<0.001$] and WRF during hospitalization (HR, 3.99; 95% CI, 1.46–10.92; $P=0.007$) were found to be independent predictors of 2-year mortality. All the risk factors were adjusted in Cox regression model 2, and LVEF

Table 3. Univariate Cox Regression Analysis for 2-Year Mortality Following Discharge From Hospital

	HR	95% CI	P value
Age ≥ 65 years	4.89	2.65–8.95	<0.001
Female sex	2.4	1.46–3.95	0.001
Diabetes mellitus	2.04	1.25–3.32	0.004
Hypertension	1.90	1.11–3.24	0.019
Smoking	0.64	0.39–1.05	0.077
Hypercholesterolemia	0.78	0.45–1.36	0.385
Previous history of MI	2.45	1.39–4.31	0.002
cTn-I >3 -fold the upper limit of normal	2.89	1.77–4.71	<0.001
Hyperuricemia	2.35	1.32–4.19	0.004
hs-CRP >3 mg/L	0.72	0.43–1.19	0.203
eGFR <60 ml $\cdot$ min $^{-1}$ $\cdot$ 1.73 m $^{-2}$ on admission	9.24	4.83–17.67	<0.001
WRF during hospitalization	2.87	2.59–5.19	<0.001
CAD 2- or 3-vessel disease	1.34	0.57–3.13	0.503
Killip class ≥ 3	3.69	2.16–6.30	<0.001
LVEF $<40\%$	2.77	1.68–4.57	<0.001
NSTEMI (compare with STEMI)	2.36	1.39–4.00	0.001
RIFLE during hospitalization			<0.001
No AKI	1.00	–	–
Risk	2.66	1.19–5.93	0.017
Injury	3.72	1.58–8.77	0.003
Failure	11.39	5.96–21.78	<0.001
No aspirin therapy	4.10	2.42–6.94	<0.001
No β -blocker therapy	2.72	1.65–4.48	<0.001
No ACEI/ARB therapy	1.42	0.86–2.32	0.171
No PCI therapy	5.27	2.99–9.26	<0.001
No CABG therapy	0.8	0.29–2.21	0.672

CI, confidence interval; HR, hazard ratio; NSTEMI, non-STEMI; WRF, worsening renal function. Other abbreviations as in Table 1.

Table 4. Multivariate Cox Regression Analysis Models for 2-Year Mortality Following Discharge From Hospital

	HR	95% CI	P value
Model 1			
eGFR <60 ml $\cdot$ min $^{-1}$ $\cdot$ 1.73 m $^{-2}$	5.58	2.25–13.85	<0.001
WRF	3.99	1.46–10.92	0.007
Model 2			
eGFR <60 ml $\cdot$ min $^{-1}$ $\cdot$ 1.73 m $^{-2}$	4.01	1.51–10.62	0.005
LVEF $<40\%$	2.83	1.11–7.20	0.029
RIFLE classification			0.001
No AKI	1.00	–	–
Risk	1.48	0.32–6.90	0.620
Injury	8.11	2.53–26.05	0.001
Failure	19.28	2.24–166.26	0.007

Model 1: adjusted covariates were age ≥ 65 years, sex, diabetes mellitus, hypertension, smoking, hypercholesterolemia, previous MI history, cardiac troponin I >3 -fold of the upper limit of normal, STEMI or NSTEMI, hs-CRP >3 mg/L, hyperuricemia, eGFR <60 ml $\cdot$ min $^{-1}$ $\cdot$ 1.73 m $^{-2}$ on admission, WRF during hospitalization, multiple CAD, Killip grade, LVEF $<40\%$, aspirin, β -blocker, ACEI/ARB, PCI, and CABG therapy in the multivariate Cox regression model.

Model 2: adjusted for all the covariates in model 1 + RIFLE classification during hospitalization in the Cox regression analysis.

Abbreviations as in Tables 1,3.

$<40\%$ (HR, 2.83; 95% CI, 1.11–7.20; $P=0.029$), eGFR <60 ml $\cdot$ min $^{-1}$ $\cdot$ 1.73 m $^{-2}$ on admission (HR, 4.01; 95% CI, 1.51–10.62; $P=0.005$), and RIFLE classification during hospitalization (Injury: HR, 8.11; 95% CI, 2.53–26.05; $P=0.001$; Failure: HR, 19.28; 95% CI, 2.24–166.26; $P=0.007$) were found to be inde-

pendent predictors of 2-year mortality (Table 4).

2-Year Follow-up Prognosis

During 389,457 person-days of follow-up (mean, 635.3 $\pm$ 204.9 days), 65 patients (10.6%) died. The 2-year incidence of death

Table 5. Incidence of Death by RIFLE Classification

Groups	Patients (n)	Deaths [n (%)]	Person-days of follow-up	Incidence of deaths/10,000 person-days
Non-AKI	534	40 (7.5)	345,167	1.16
RIFLE-R	36	7 (19.4)	22,380	3.13
RIFLE-I	23	6 (26.1)	13,599	4.41
RIFLE-F	20	12 (60)	8,311	14.44

Abbreviations as in Table 1.

per 10,000 person-days of follow-up was 1.16 in non-AKI patients, 3.13 in RIFLE-R patients, 4.41 in RIFLE-I patients, and 14.44 in RIFLE-F patients (Table 5). The Kaplan-Meier survival plot and log-rank test showed that all-cause mortality during the 2-year follow-up period was proportional to the severity of AKI that was determined using the RIFLE classification during hospitalization (Figure).

Multivariate Analysis of Predictors of AKI During First 7 Days After Hospitalization

The baseline clinical and biochemical characteristics including age, sex, hypertension, diabetes mellitus, smoking, impaired LVEF, Killip class, cardiac troponin I >3-fold of the upper limit of normal, mean blood pressure, serum lipid profile levels, white blood count, hs-CRP, uric acid level and receiving reperfusion therapies, which were possible predictors for developing AKI by RIFLE classification, were adjusted in the multivariate logistic regression analysis. The eGFR on admission per $1 \text{ ml} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2}$ decrease [odds ratio (OR), 1.07; 95% CI, 1.04–1.09; $P < 0.001$] and hs-CRP per 1 mg/L increase (OR, 1.01; 95% CI, 1.01–1.02; $P = 0.045$) were independent predictors of AKI by the RIFLE classification (Table 6).

Table 6. Multivariate Analysis for Predictors of AKI During the First 7 Days After Hospitalization

	OR	95% CI	P value
eGFR per $(\text{ml} \cdot \text{min}^{-1} \cdot 1.73 \text{ m}^{-2})$ decrease	1.07	1.04–1.09	<0.001
hs-CRP (per 1 mg/L)	1.01	1.01–1.02	0.025

Adjusted covariates were age ≥ 65 years, sex, diabetes mellitus, hypertension, smoking, cardiac troponin I >3-fold of the upper limit of normal, Killip grade, LVEF <40%, cholesterol, HDL, triglyceride, eGFR on admission, WBC count, hs-CRP, serum uric acid level, and reperfusion therapy in the multivariate logistic regression analysis.

OR, odds ratio. Other abbreviations as in Tables 1,2.

Discussion

The main finding of this study is that AKI by the RIFLE classification, as used in other critical diseases,^{9–15} is an independent predictor of 2-year mortality in AMI patients. The overall 2-year all-cause mortality rate in this investigation was 10.6%, which is in agreement with the findings of previous reports.^{23,24} Many clinical risk factors and treatments, which showed significant differences in the survivor and non-survivor groups in this study, have been reported to affect the outcomes of AMI

patients.^{25–31} In addition, these clinical variables also have been proved to be AKI predictors in cardiovascular patients.^{32–36} In the present study, by adjusting the baseline risk factors, inflammatory markers, severity of AMI and treatment in the multivariate analysis, initial eGFR and hs-CRP level on admission were independent predictors for AKI occurrence during the first week of an AMI patient's hospitalization. This finding agrees with previous studies and prompts clinicians to pay more attention to those patients with initial renal dysfunction and greater inflammatory status in order to avoid further kidney injury.^{20,34,37,38} Adjusting these risk factors, treatment and the 3 types of assessment of renal function (ie, initial renal dysfunction on admission, WRF and RIFLE classification) in the multivariate Cox regression model, only initial renal dysfunction, RIFLE classification, and impaired LVEF were found to be independent predictors for 2-year mortality.

AKI is defined as a sudden deterioration of kidney function, which presents as an increase in serum creatinine levels or a decrease in urine output. In high-risk patients, such as those hospitalized for AMI, congestive heart failure, sepsis, and those who have undergone cardiac surgery, the incidence of AKI is high, ranging from 10% to 25%.³⁹ Many clinical trials have shown that decreased renal function is an independent predictor of adverse outcomes in AMI patients.^{40,41} However, the limitation of those clinical trials is that only the initial renal function levels were used. AMI and its complications, such as hypotension or shock-related kidney hypoperfusion, may be partly responsible for impaired initial renal function on admission. However, patients with chronic renal disease before admission can also present with decreased initial renal function. Therefore, unless the baseline renal function data are considered, initial renal function levels cannot represent the effect of AKI.

AKI can occur not only during the initial period but also during the course of hospitalization (ie, WRF). WRF during hospitalization can predict clinical outcomes in acute heart failure patients.^{42,43} However, few studies have evaluated the effect of WRF in the AMI population.⁵ Our study results agree that WRF is an independent predictor for 2-year mortality after adjusting for multiple risk factors except the RIFLE classification (model 1). However, when the RIFLE classification was included in the multivariate Cox analysis (model 2), initial renal dysfunction, RIFLE classification, and impaired LVEF were found to be independent predictors.

WRF was determined by comparing the worst renal function outcomes during hospitalization with the initial renal function levels. However, the RIFLE classification compares the worst outcomes of renal function during hospitalization with the baseline renal function levels. As we have previously mentioned, the initial renal function on admission may be affected by the disease course of AMI, thereby leading to underestimation of the severity of WRF. This may explain why RIFLE during hospitalization was more significant than WRF in predicting the 2-year mortality in AMI patients after adjusting all the risk factors. Moreover, with an increase in the RIFLE severity grade, the HR and incidence of deaths per 10,000 person-days also increased.

The mechanism of AKI by RIFLE classification resulting in worsening prognosis is very complex. It has been shown that a large percentage of AKI patients will not have an improvement in their renal function and, moreover, they will suffer from chronic kidney disease or require permanent renal replacement therapy.^{44,45} The incidence of progressive to chronic kidney disease was proportional to AKI severity by RIFLE classification.^{46,47} In addition, AKI will directly contribute to dis-

tant organ dysfunction through the mechanism of oxidative stress, immunity and inflammation.^{15,48–50} This means AKI severity by RIFLE classification represents not only kidney damage but also imbalance between the kidney and other organ systems. Therefore, AKI by RIFLE classification, rather than initial renal function and impaired LVEF, has a greater effect on mortality.

The hospital discharge risk score is a useful tool for assessing the long-term outcomes of AMI patients at the time of discharge.^{51,52} The initial renal dysfunction is used in this risk score system. Our findings indicated that AKI by RIFLE classification, in addition to initial renal dysfunction, may be considered as involved in the establishment of a new hospital discharge risk score to predict long-term outcomes in AMI patients who survive to discharge.

Study Limitations

The observational study has some limitations. First, using recorded serum creatinine levels rather than other methods as the baseline reference in the RIFLE classification can improve the accuracy of AKI.⁵³ However, previous records of the serum creatinine levels of only 310 patients were available in our database. Second, the volume of contrast used in diagnostic angiography and PCI therapy was not available in all cases, so the effect of contrast volume on renal impairment was not included in the analysis.

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Disclosures

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