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ORIGINAL ARTICLE

Decreased glomerular filtration rate is a significant and independent risk for in-hospital mortality in Japanese patients with acute myocardial infarction: report from the Hokkaido acute myocardial infarction registry

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Renal dysfunction is a significant risk factor in the prognosis of patients with cardiovascular diseases. We sought to determine the relationship between estimated glomerular filtration rate (eGFR) values and in-hospital mortality in Japanese acute myocardial infarction (AMI) patients. A total of 2266 consecutive AMI patients admitted to 22 hospitals in Hokkaido were registered. The eGFR values were determined using the following equation: eGFR=194×(serum creatinine)$^{-1.094}$×(age)$^{-0.287}$ (×0.739 if female). Patients were classified into four groups according to their eGFR values: ⩾60 (*n*=1304), 30–59 (*n*=810), 15–29 (*n*=87) and <15 ml min^{-1} per 1.73 m^2 (*n*=65). A total of 110 patients (4.9%) died during hospitalization. The in-hospital mortality rate was significantly higher in patients with reduced eGFR values at 2.3, 5.4, 24.1 and 23.1% for eGFR values of ⩾60, 30–59, 15–29, and <15 ml min^{-1} per 1.73 m^2, respectively. The odds ratios for in-hospital all cause death were 8.26 (95% confidence interval (CI): 2.22–30.77) for eGFR <15 ml min^{-1} per 1.73 m^2 and 3.42 (95% CI: 1.01–11.61) for eGFR 15–29 ml min^{-1} per 1.73 m^2 compared with eGFR ⩾60 ml min^{-1} per 1.73 m^2. Similarly, the odds ratios for in-hospital cardiac death were 8.43 (95% CI: 1.82–39.05) for eGFR <15 ml min^{-1} per 1.73 m^2 and 5.47 (95% CI: 1.51–19.80) for eGFR 15–29 ml min^{-1} per 1.73 m^2. In conclusion, the eGFR of <30 ml min^{-1} per 1.73 m^2 was a significant and independent risk for in-hospital mortality in abroad cohort of Japanese patients with AMI.
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Keywords: acute myocardial infarction; chronic kidney disease; estimated glomerular filtration rate; mortality

INTRODUCTION

Chronic kidney disease (CKD) is increasingly becoming recognized as a global public health problem.[1] The National Kidney Foundation has published clinical guidelines on the evaluation, classification and risk stratification in patients with CKD.[2] Despite the recognized association between a reduced estimated glomerular filtration rate (eGFR) and poor prognosis, screening for CKD is frequently limited to the measurement of serum creatinine,[3,4] which does not accurately reflect the GFR. As a result, the management of this risk is often not optimized. The risks of CKD included not only the progression to end-stage renal failure but also the occurrence of adverse cardiovascular outcomes.[5–9] Previous studies have demonstrated that CKD is an independent risk factor for morbidity and mortality in the general population,[10] as well as in patients with cardiovascular diseases such as post acute myocardial infarction (AMI).[11] Anavekar *et al.*[11] reported that CKD was a common and significant independent risk factor for cardiovascular events in AMI patients based on data from the Valsartan in Acute Myocardial Infarction Trial (VALIANT). The risk was progressive, and each 10 unit reduction in the eGFR was significantly associated with a 10% increase in the relative risk of death or nonfatal cardiovascular complications.[11] However, the patients in the VALIANT study had heart failure, left ventricular dysfunction, or both as a complication of AMI, and patients with a baseline serum creatinine level >2.5 mg dl^{-1} were excluded. Therefore, the patients enrolled in the study by Anavekar *et al.*[11] were not representative of the general AMI population routinely encountered in clinical practice. It is critically important to determine the prognostic impact of CKD in the registry data of Japanese patients with AMI.

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The aim of the present study was to examine the prognostic significance of eGFR values on the in-hospital mortality in Japanese AMI patients in routine clinical practice.

METHODS

Patients

The study patients consisted of 2266 consecutive patients hospitalized because of AMI in 22 hospitals in Hokkaido from 2005 to 2007.

AMI was defined by the presence of at least two of the following criteria:[12–14] (1) a clinical history of chest pain persisting for $\geqslant$30 min, (2) ischemic electrocardiographic changes and (3) a peak creatine kinase level equivalent to more than twice the upper limit of normal. All patients underwent coronary catheterization within 24 h after the onset of AMI. Body weight and height were measured in the morning after fasting. Body mass index was calculated as body weight (kg) divided by squared height (m). Smoking habits were determined using a self-reported questionnaire. Patients who had never smoked and ex-smokers were classified as 'nonsmokers'. Hypertension was defined as a history of systolic blood pressure $\geqslant$140 mm Hg and/or diastolic blood pressure $\geqslant$90 mm Hg or the use of oral antihypertensive drugs. Dyslipidemia was defined as a fasting total cholesterol $\geqslant$220 mg per 100 ml or the use of anti-hypercholesterol drugs. Diabetes mellitus was defined as fasting plasma glucose $\geqslant$126 mg per 100 ml or the use of oral hypoglycemic drugs or insulin. Patients who had suffered from myocardial infarction and stroke were defined as 'prior cardiovascular disease'. Blood samples were obtained after an overnight fast in the hospital. The creatine kinase values were measured every 4 h after admission to determine the peak value. The information regarding all cause death and cardiac death during hospitalization was obtained by physicians in the hospitals where the patients were admitted. Cardiac death was defined as a death due to heart failure, fatal arrhythmia, cardiac rupture or recurrent myocardial infarction. The patient data were registered in each hospital and reported to the data management office at Hokkaido University. Written informed consent was obtained from each patient or a family member. The study protocol was approved by the ethics committee at Hokkaido University School of Medicine.

Measurement of the eGFR

To calculate the eGFR, serum creatinine was measured using the compensated Jaffe creatinine method at the time of admission to the hospital. The eGFR was calculated using the equation for Japanese as follows:[15] eGFR=194$\times$(serum creatinine)$^{-1.094}\times$(age)$^{-0.287}$ ($\times$0.739 if female) ml min^{-1} per 1.73 m^2.

Statistical analysis

The characteristics of the study subjects were expressed as means $\pm$ s.d. for continuous variables, median (and interquartile range) for skewed distribution variables, and percentages for categorical variables according to the eGFR values. The differences in variables among groups were examined by analysis of variance, Kruskal–Wallis test or chi-square test. The association between the risk factors and in-hospital deaths of AMI patients was assessed using multiple logistic regression analysis. The principal model included the following candidate variables: demographics (age, sex, body mass index, smoking, prior cardiovascular disease), medical history (hypertension, diabetes mellitus, dyslipidemia, Q wave myocardial infarction and peak creatine kinase), angiographic data (number of diseased vessels, Killip classification, thrombolysis in myocardial infarction flow grade 0 at admission and thrombolysis in myocardial infarction flow grade 3 after treatment) and procedural information (mechanical support and treatment). Variables that were regarded as significant ($P<0.05$) were included in subgroup multivariate analyses. A P-value of <0.05 indicated statistical significance. All statistical analyses were performed using the SPSS statistical package for Windows version 11.0 (Chicago, IL, USA).

RESULTS

Patient characteristics

Figure 1 shows the distribution of the eGFR in the study patients. The mean eGFR value was 64.4 $\pm$ 23.7 ml min^{-1} per 1.73 m^2, ranging from 3.3 to 171.2 ml min^{-1} per 1.73 m^2 for 2254 patients. In all, 12 patients under hemodialysis were included in the group of eGFR values $<$15 ml min^{-1} per 1.73 m^2. In all, 962 (42.5%) patients had an eGFR $<$60 ml min^{-1} per 1.73 m^2 or dialysis treatment.

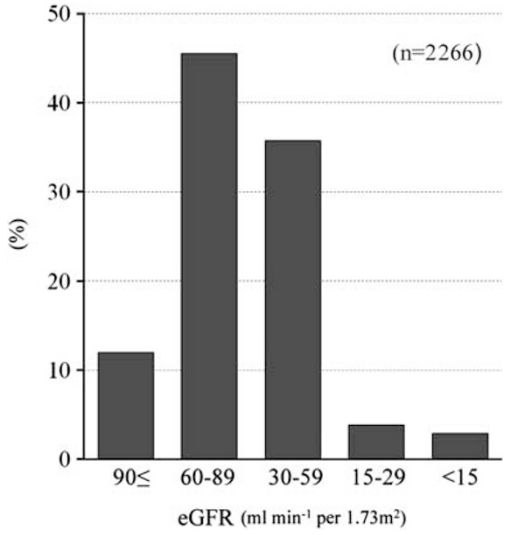

Figure 1 The distribution of the eGFR in the study patients.

Table 1 shows the baseline demographic and medical characteristics of the patients according to eGFR levels. The mean age of the patients was 66 $\pm$ 12 years and 72.0% were men. Patients with a reduced eGFR were older and more often women. They were more likely to have hypertension, diabetes mellitus, dyslipidemia and prior cardiovascular disease.

Table 2 shows the baseline angiographic and procedural characteristics of the patients according to eGFR levels. Patients with a reduced eGFR were more likely to have severe coronary artery stenosis, severe heart failure symptoms based on Killip classifications and higher use of mechanical supports such as intraaortic balloon pumping or percutaneous cardiopulmonary support. The prevalence of thrombolysis in myocardial infarction flow grade 0 at admission and thrombolysis in myocardial infarction flow grade 3 after treatment was lower with reduced eGFR levels. The performance of percutaneous coronary intervention by stent implantation was lower and that of thrombolysis, balloon angioplasty and coronary artery bypass grafting was higher with reduced eGFR levels.

Outcomes

A total of 110 (4.9%) patients died because of any causes and 84 (3.7%) patients died because of cardiac events during hospitalization during the follow-up period of 20 $\pm$ 17 (2–92) days.

Table 3 shows the odds ratios and 95% confidence interval (CI) for all cause and cardiac death according to eGFR levels. The rates of all cause death were 2.3, 5.4, 24.1 and 23.1% in patients with eGFR values $\geqslant$60, 30–59, 15–29 and $<$15 ml min^{-1} per 1.73 m^2, respectively. The rates of cardiac death were 1.8, 4.1, 19.5 and 15.4% in subjects with eGFR values $\geqslant$60, 30–59, 15–29 and $<$15 ml min^{-1} per 1.73 m^2, respectively. Decreases in the eGFR levels were associated with a significant progressive elevation of risk for all cause ($P<0.05$ for trend) and cardiac death ($P<0.01$ for trend). By multivariate analysis with an eGFR $\geqslant$60 ml min^{-1} per 1.73 m^2 as the reference, patients with eGFR of 15–29 and $<$15 ml min^{-1}per 1.73 m^2 had a significantly elevated risk for all cause death (OR 3.42, 95% CI 1.01–11.61 and OR 8.26, 95% CI 2.22–30.77, respectively) and cardiac death (OR 5.47, 95% CI 1.51–19.80 and OR 8.43, 95% CI 1.82–39.05, respectively). By multiple logistic regression analysis, age, Killip classification $\geqslant$II at admission, prior cardio-

Table 1 Demographic and medical characteristics of the patients according to eGFR levels

		eGFR				
	Total (n=*2266*)	⩾*60* (n=*1304*)	*30–59* (n=*810*)	*15–29* (n=*87*)	<*15* (n=*65*)	P-*value*
Age (years)	66±12	63±12	71±11	77±9	78±11	<0.001
Male (%)	72.0	76.5	67.9	55.2	56.4	<0.001
Body mass index (kg m^{-2})	24.4±3.7	24.5±3.6	24.3±3.5	23.5±4.4	24.2±6.5	0.15
Smoking (%)	50.5	57.3	41.8	30.8	48.3	<0.001
Hypertension (%)	44.6	36.1	53.0	69.4	76.2	<0.01
Diabetes mellitus (%)	22.7	19.5	23.8	38.8	50.0	<0.001
Dyslipidemia (%)	20.6	18.7	23.0	28.0	20.0	<0.05
Prior cardiovascular disease (%)	20.7	16.5	26.1	26.4	29.7	<0.01
Q wave myocardial infarction (%)	66.1	72.3	55.7	67.5	70.2	0.57
Peak creatine kinase (IU per 100 ml)	2559 (1441–4201)	2623 (1486–4275)	2492 (1440–4107)	2505 (1018–3692)	2604 (1112–4281)	0.26

Abbreviation: eGFR, estimated glomerular filtration rate.
Values are means±s.d., median (and interquartile range) and percentage.

Table 2 Angiographic and procedural characteristics of the patients according to eGFR levels

		eGFR				
	Total (n=*2266*)	⩾*60* (n=*1304*)	*30–59* (n=*810*)	*15–29* (n=*87*)	<*15* (n=*65*)	P-*value*
Number of diseased vessels (%)						
LMT	4.3	2.8	5.1	9.2	16.9	<0.001
One vessel	55.1	59.6	50.9	47.8	29.2	<0.001
Two vessel	27.3	26.1	29.1	22.1	35.4	<0.01
Three vessel	13.3	11.5	14.9	20.9	18.5	<0.001
Killip classification ⩾II						
At admission (%)	18.5	11.7	24.5	47.1	43.5	<0.01
Mechanical support (IABP/PCPS) (%)	13.7	10.8	15.6	26.4	30.8	<0.001
TIMI flow grade 0 at admission (%)	68.8	69.7	69.9	57.5	53.8	<0.01
TIMI flow grade 3 after treatment (%)	99.1	99.4	98.9	97.3	96.2	<0.05
Treatment (%)						
Thrombolysis	6.8	5.2	8.1	13.8	13.9	<0.001
Balloon angioplasty	6.6	6.6	6.3	5.8	12.3	<0.001
Coronay stent	85.9	87.5	85.1	79.3	72.3	<0.001
CABG	0.7	0.7	0.5	1.1	1.5	<0.001

Abbreviations: CABG, coronary artery bypass grafting; eGFR, estimated glomerular filtration rate; IABP, intraaortic balloon pumping; LMT, left main trunk; PCPS, percutaneous cardiopulmonary support; TIMI, thrombolysis in myocardial infarction.

vascular disease, peak creatine kinase and eGFR values were significant and independent predictors for all cause death. Age, the use of mechanical support, peak creatine kinase and eGFR values were significant and independent predictors for cardiac death (Table 4).

Table 5 shows the results of subgroup analysis for all cause death according to eGFR levels stratified by sex, age (⩾65 *vs.* <65 years) and comorbidities (hypertension *vs.* no hypertension and diabetes mellitus *vs.* no diabetes mellitus). The eGFR <30 ml min^{-1} per 1.73 m^2 was associated with poor outcomes in each subgroup, which is in agreement with the results of the primary analysis.

DISCUSSION

The present study demonstrated that the prevalence of AMI patients with an eGFR <60 ml min^{-1} per 1.73 m^2 was 42.5% based on a large-scale, multicenter trial. A reduced eGFR was a significant and independent risk for in-hospital all-cause and cardiac mortality. Moreover, AMI patients with eGFR values <30 ml min^{-1} per 1.73 m^2 had a significantly greater mortality risk than patients with values ⩾60 ml min^{-1} per 1.73 m^2.

Previous studies used serum creatinine levels rather than the eGFR to detect renal dysfunction.[3,4] However, the accuracy of serum creatinine levels is limited as a marker of renal function because significant kidney dysfunction can be present despite a normal serum creatinine concentration. Serum creatinine has a nonlinear association with eGFR according to age, sex and lean body mass.[16,17] The National Kidney Foundation[2] and Kidney Disease Improving Global Outcomes (KDIGO)[18] have recommended using an eGFR estimated by serum creatinine, and eGFR <60 ml min^{-1} per 1.73 m^2 is selected as the cutoff value for the diagnosis of CKD. The eGFR values were generally estimated by the modification of diet in renal disease or creatinine clearance. Imai *et al.*[19] reported that the modification of diet in renal disease equation might overestimate the GFR in Japanese populations compared with the GFR measured using insulin clearance. Matsuo *et al.*[15] demonstrated that the accuracy of the eGFR estimation was

Table 3 The odds ratios and 95% CI for all cause and cardiac death according to eGFR values

	eGFR				
	⩾60 (n=1304)	*30–59 (n=810)*	*15–29 (n=87)*	*<15 (n=65)*	*P for trend*
All cause death					
n (%)	30 (2.3)	44 (5.4)	21 (24.1)	15 (23.1)	
Model 1, odds ratio (95% CI)	1.00	1.66 (1.01–2.73)	7.58 (3.95–14.53)	10.64 (5.31–21.30)	<0.001
Model 2, odds ratio (95% CI)	1.00	1.58 (0.99–2.81)	6.24 (3.03–12.85)	9.55 (4.39–20.77)	<0.001
Model 3, odds ratio (95% CI)	1.00	1.05 (0.47–2.36)	3.42 (1.01–11.61)	8.26 (2.22–30.77)	<0.05
Cardiac death					
n (%)	24 (1.8)	33 (4.1)	17 (19.5)	10 (15.4)	
Model 1, odds ratio (95% CI)	1.00	1.52 (0.87–2.67)	7.49 (3.67–15.29)	8.90 (4.01–20.00)	<0.001
Model 2, odds ratio (95% CI)	1.00	1.46 (0.85–2.52)	7.10 (3.31–15.20)	8.88 (3.74–21.04)	<0.001
Model 3, odds ratio (95% CI)	1.00	1.02 (0.41–2.53)	5.47 (1.51–19.80)	8.43 (1.82–39.05)	<0.01

Abbreviations: CI, confidence interval; eGFR, estimated glomerular filtration rate.
Model 1, adjusted for demographic (age and sex) variables.
Model 2, adjusted for demographic (age and sex) and medical (hypertension, diabetes mellitus and dyslipidemia) variables.
Model 3, adjusted for demographic (age and sex), medical (body mass index, smoking, hypertension, diabetes mellitus, dyslipidemia, prior cardiovascular disease, Q wave myocardial infarction and peak creatine kinase), angiographic (number of diseased vessles and killip classification ⩾II) and procedural (mechanical support, TIMI (thrombolysis in myocardial infarction) flow grade 0 at admission and TIMI flow grade 3 after treatment) variables.

Table 4 Multivariate predictors of outcomes by multiple logistic regression analysis

	Odds ratio	*95% CI*	P-*value*
All cause death			
Age (per 1 year increase)	1.06	1.02–1.10	<0.01
Killip classification ⩾II at admission	2.60	1.19–5.69	<0.05
Prior cardiovascular disease	2.21	1.02–4.79	<0.05
Peak creatine kinase (per 100 IU per 100 ml increase)	1.02	1.01–1.03	<0.01
eGFR (per 10 ml min^{-1} per 1.73 m^2 decrease)	1.36	1.14–1.62	<0.01
Cardiac death			
Age (per 1 year increase)	1.06	1.02–1.10	<0.01
Mecanical support (IABP/PCPS)	3.51	1.39–8.84	<0.01
Peak creatine kinase (per 100 IU per 100 ml increase)	1.01	1.01–1.02	<0.05
eGFR (per 10 ml min^{-1} per 1.73 m^2 decrease)	1.37	1.13–1.68	<0.01

Abbreviations: CI, confidence interval; eGFR, estimated glomerular filtration rate; IABP, intraaortic balloon pumping; PCPS, percutaneous cardiopulmonary support.
Adjusted for demographic (age and sex), medical (body mass index, smoking, hypertension, diabetes mellitus, dyslipidemia, prior cardiovascular disease, Q wave myocardial infarction, peak creatine kinase and eGFR), angiographic (number of diseased vessles and killip classification ⩾II) and procedural (mechanical support, TIMI (thrombolysis in myocardial infarction) flow grade 0 at admission and TIMI flow grade 3 after treatment) variables.

more improved using the new Japanese equation rather than using the modification of diet in renal disease equation in Japanese populations. Therefore, the present study used the new Japanese equation to calculate the eGFR values.

Previous studies demonstrated that CKD is an independent risk factor for cardiovascular disease in the general population in Japan.[20,21] Anavekar *et al.*[11] showed that the prevalence of CKD patients suffering from AMI in Western countries was 33.5%, and Nakamura *et al.*[22] reported that it was 31.6% in Japanese patients with coronary artery disease. The prevalence of CKD in the present study was 42.4%, which is higher than the rates reported in previous studies.[11,22] These discrepancies may be partially explained by the differences in ethnicity and other risk factors such as age and obesity. More importantly, the registry used in this study enrolled all patients that were admitted to the hospital because of AMI and did not exclude those who had higher levels of serum creatinine or dialysis treatment. Thus, the patients in the present study had a high prevalence of renal dysfunction and were considered to be more reflective of current routine clinical practice.

The present study extended the previous studies and demonstrated the prognostic significance of reduced eGFR in patients with coronary artery disease.[3,10,20,21,23–28] The Atherosclerosis Risk in Communities (ARIC) study[23] and the Second National Health and Nutrition Examination Survey (NHANES II)[3] demonstrated that a mild reduction in eGFR was a significant risk factor for both coronary vascular disease and all-cause mortality. The present study confirmed the prior studies by Anavekar *et al.* in which reduced eGFR was independently associated with an increased risk of fatal and nonfatal adverse cardiovascular events after AMI.[11] However, in their study, the study patients were limited to have baseline serum creatinine levels $<2.5\ mg\ dl^{-1}$ and heart failure. In addition, only 20% of the patients were treated with percutaneous coronary intervention. Thus, the impact of CKD has not been elucidated in a representative cohort of patients receiving contemporary therapy for AMI. To examine the

Table 5 Subgroup analysis for all cause death according to eGFR levels

	Total patients, n	*All cause death,* n	*Odds ratio*	*95% CI*	P-*value*
Male	1632	39			
⩾60	997	6	1.00	Reference	
30–59	550	16	1.55	0.83–2.92	0.17
<30	85	17	4.60	1.81–11.70	<0.01
Female	634	71			
⩾60	307	24	1.00	Reference	
30–59	260	28	1.76	0.61–5.08	0.30
<30	67	19	10.23	3.43–34.52	<0.001
Age ⩾65 years	1327	91			
⩾60	597	23	1.00	Reference	
30–59	610	39	1.45	0.81–2.60	0.21
<30	120	29	4.13	1.68–10.14	<0.01
Age <65 years	939	19			
⩾60	707	7	1.00	Reference	
30–59	200	5	2.40	0.60–9.68	0.22
<30	32	7	27.12	6.70–78.28	<0.001
Hypertension	1010	70			
⩾60	471	14	1.00	Reference	
30–59	429	30	2.02	0.98–4.19	0.06
<30	110	26	7.33	2.56–21.05	<0.001
No hypertension	1256	40			
⩾60	833	16	1.00	Reference	
30–59	381	14	1.13	0.50–2.56	0.76
<30	42	10	2.69	1.12–6.58	<0.05
Diabetes mellitus	513	44			
⩾60	254	8	1.00	Reference	
30–59	193	18	1.25	0.67–2.35	0.49
<30	66	18	10.66	2.66–42.73	<0.01
No diabetes mellitus	1753	66			
⩾60	1050	22	1.00	Reference	
30–59	617	26	2.68	0.99–5.66	0.06
<30	86	18	3.38	1.17–9.83	<0.05

Abbreviations: CI, confidence interval; eGFR, estimated glomerular filtration rate.
Adjusted for demographic (age and sex), medical (body mass index, smoking, hypertension, diabetes mellitus, dyslipidemia, prior cardiovascular disease, Q wave myocardial infarction and peak creatine kinase), angiographic (number of diseased vessles and killip classification ⩾II) and procedural (mechanical support, TIMI (thrombolysis in myocardial infarction) flow grade 0 at admission and TIMI flow grade 3 after treatment) variables.

impact of eGFR in AMI and to determine whether it is independently associated with prognosis, we analyzed data from a prospective broad cohort of patients with AMI. The patients in the present study had baseline serum creatinine levels ranging from 0.3 to 12.6 mg dl^{-1}, and percutaneous coronary intervention was performed in 92.5% of the patients during the acute phase. We thus could extend the prognostic impact of CKD from selected patients in large-scale clinical trials to a diverse cohort of AMI patients in general. Kasai *et al.*[29] demonstrated that lower eGFR values were significant long-term predictors for all-cause and cardiac mortality in Japanese patients who underwent complete coronary revascularization using a 10-year cohort study. The present study also confirmed these findings. Although the present study included both patients that underwent complete and incomplete coronary revascularization, adjustments for this variable were included in the statistical analysis. As a result, the findings in this study might be more applicable to a general population.

The present study demonstrated that the significant risk factors differed depending on the outcomes such as all cause and cardiac death (Table 4). There are potential explanations for these findings. Cardiac death was defined as death due to heart failure, fatal arrhythmia, cardiac rupture and recurrent myocardial infarction. In contrast, all cause death included not only cardiac death but also other causes of death such as infection. Killip classification and prior cardiovascular disease were independent predictors for all cause death, suggesting that heart failure associated with AMI might induce systemic organ failure and other complications leading to death. Mechanical support was an independent predictor for cardiac death, indicating that severe circulatory shock associated with

AMI may result in cardiac death, including fatal arrhythmia and cardiac rupture.

There are several possible explanations by which CKD increases the in-hospital mortality in patients with AMI. CKD may be indicative of traditional risk factors such as older age, hypertension, dyslipidemia and diabetes mellitus, which have been established to be closely related to cardiovascular outcomes.[30] Therefore, CKD may reflect the presence of severe coronary artery disease. Even after adjustments for demographic, medical, angiographic and procedural variables, the eGFR remained a significant risk for in-hospital all-cause and cardiac mortality. The increase in mortality with a reduced eGFR can be partly explained by nontraditional factors associated with CKD, including increased inflammatory factor levels,[31] elevated homocysteine levels,[32] enhanced coagulability[31] and endothelial dysfunction,[33] which were not assessed in this study.

Study limitations

First, we only assessed the baseline eGFR and could not determine the effects of changes in eGFR on outcomes. Second, the present study did not collect data regarding the use of medication after hospitalization because it varies widely among patients according to their clinical status, especially during the acute phase of AMI. However, the use of medication may affect the outcomes of patients during the long-term follow-up. Third, information was not collected during the follow-up after discharge, and the impact of CKD on the long-term outcomes in AMI patients could not be assessed. Fourth, coronary catheterization was performed after the onset of AMI in all patients, but severe patients who could not undergo coronary catheterization were not included. Therefore, the present results cannot be applied to all patients with AMI in general.

CONCLUSION

The reduced eGFR was a significant and independent risk for in-hospital all-cause and cardiac mortality in a broad cohort of Japanese patients hospitalized with AMI. Evaluation of renal function and effective management of these high-risk patients with AMI is important.

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