The GFR was calculated according to the abbreviated Modification of Diet in Renal Disease (MDRD) study formula [12,13] based on the serum creatinine level measured on admittance during the index hospitalization. We calibrated the baseline measurement of serum creatinine using the enzymatic method against that of the MDRD core laboratory. Moreover, we used an original race coefficient as one component of the MDRD equation that improves its accuracy of GFR estimation in the Japanese population [14].

The distribution of the estimated GFR was divided into four categories (<45.0, 45.0 to 59.9, 60.0 to 74.9, and ≥75.0 mL/min per 1.73 m^2), incorporating the guidelines of the National Kidney Foundation [2]. The primary study outcome was death from any cause (acute myocardial infarction, heart failure, sudden death, cerebrovascular disease, other cardiovascular cause, or non-cardiovascular causes). Secondary outcome was a composite of cardiovascular events (death from cardiovascular causes, nonfatal AMI, hospitalization for heart failure and sudden death). Each end point was specified in the original cohort study protocol. If a patient had ≥2 events, the results of the first were used in the combined end-point analysis. The incidence of end-point events was determined annually based on hospital records, contact with patients, or certificates issued by administrative authorities. These records were provided to the end-point classification committee (composed of 2 cardiologists), which then determined and categorized each event for use in analysis.

Analyses were performed using the SAS system ver. 9.1 software (SAS Institute Inc., Cary, NC, USA). Data are presented as means ± SD, medians with interquartile ranges or frequencies. Groups were compared with respect to normally distributed continuous variables using the one-way analysis of variance, and the Kruskal–Wallis test was applied for other variables. The χ^2 test was used to compare nominally scaled variables. The cumulative probabilities of event curves were estimated using the Kaplan–Meier method. We determined the influence of estimated GFR with respect to outcomes using univariate and multivariate Cox proportional hazards models. To evaluate the linearity of influence, we also analyzed trends. The proportional hazards assumption was confirmed by the log (− log survival function). The influence of profile, interaction and co-linearity in the multivariate model were examined using regression diagnostic analysis. Two-tailed P-values of <0.05 were considered to indicate a statistically significant difference. All analyses were performed at an independent biostatistics and data center (STATZ Institute, Inc., Tokyo, Japan).

3. Results

We identified a total of 4550 patients with a mean (±SD) age of 66.9 ± 12.1 years (26.7% women), who had known serum creatinine values, had not previously received dialysis, and had been discharged alive. The estimated baseline GFR for the patients was widely and normally distributed (Fig. 1). The mean (±SD) estimated GFR was 63.6 ± 19.0 mL/min per 1.73 m^2 (range, 3.9 to 120.4) (Table 1). The estimated GFR values were ≥75.0, 60.0 to 74.9, 45.0 to 59.9 and <45.0 mL/min per 1.73 m^2 in 1226 (26.9%), 1383 (30.4%), 1274 (28.0%) and 667 (14.7%) patients, respectively. A total of 1941 (42.7%) patients met the estimated GFR criteria for chronic kidney disease. The absolute difference in the median serum creatinine level between the contiguous groups was 0.2 to 0.4 mg/dL.

Percutaneous coronary intervention was performed in 75% of the patients and the mean left ventricular ejection fraction was 53%. Coexisting illness, patient status and treatment strategies during hospitalization varied with estimated baseline GFR (Table 1). Patients with a lower estimated GFR were older and more likely to be women. Patients in the lowest category of estimated GFR had the highest rates of hypertension, diabetes, prior myocardial infarction and coronary-artery bypass grafting, as well as atrial fibrillation. Serum lipid levels (total cholesterol, low-density lipoprotein cholesterol, and triglyceride) were lower in the categories of estimated GFR suggestive of chronic kidney disease. Serum C-reactive protein and uric acid levels increased with a decreased estimated GFR. A lower estimated GFR was associated with lower left ventricular ejection fraction. The proportions of patients who had undergone percutaneous coronary intervention and coronary-artery bypass grafting were lower and higher, respectively, in the categories of chronic kidney disease. Among cardiovascular medications received at baseline, angiotensin-converting-enzyme inhibitors, statins and aspirin were administered least frequently, whereas calcium-channel blockers and nitrates were administered most frequently to patients in the lowest category of estimated GFR.

The median follow-up among the 4550 patients was 4.1 years (interquartile range, 3.3 to 4.8) and the follow-up rate was 95.2%. A

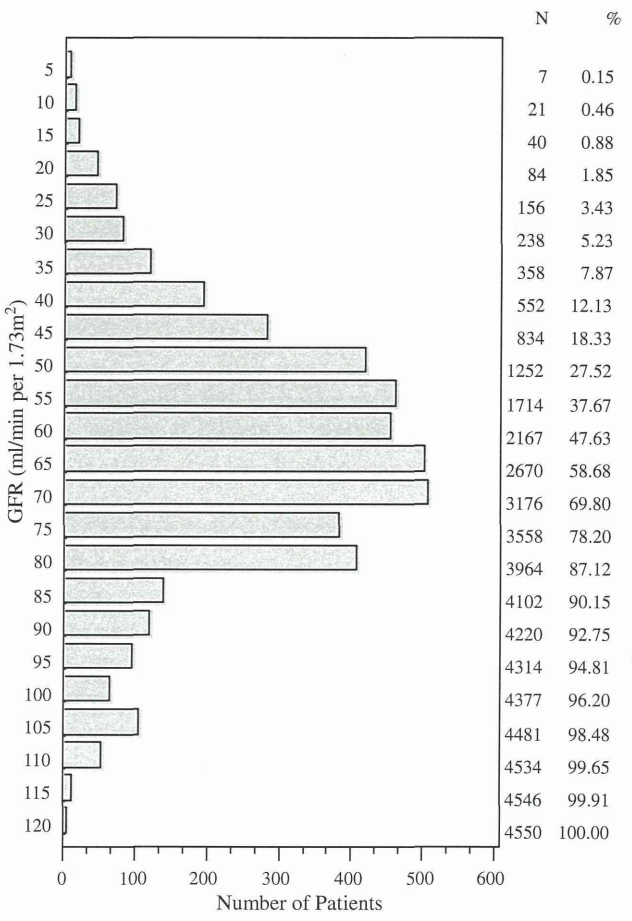

Fig. 1. Distribution of estimated GFR at baseline among the 4550 patients. GFR, glomerular filtration rate.

decreased estimated GFR<60.0 mL/min per 1.73 m^2 was associated with increasing mortality (Fig. 2). Unadjusted Kaplan–Meier estimates of total and cardiovascular death rates during the entire follow-up were 8.7 and 3.4%, 8.9 and 3.9%, 13.8 and 7.0% and 29.5 and 17.4% in group with an estimated GFR of ≥75.0, 60.0 to 74.9, 45.0 to 59.9 and <45.0 mL/min per 1.73 m^2 (Fig. 3). Cardiovascular death accounted for more than half of the total deaths in the groups with an estimated GFR of 45.0 to 59.9 and of <45.0 mL/min per 1.73 m^2. The other cardiovascular end points were also more frequent among patients with, than without chronic kidney disease at baseline. Consequently, the risk of death from any cause and cardiovascular events increased with increasing severity of kidney disease. Unadjusted hazard ratios for total and cardiovascular death in the group with an estimated GFR of 45.0 to 59.9 mL/min per 1.73 m^2 using the group with an estimated GFR of ≥75.0 mL/min per 1.73 m^2 as the reference were 1.63 (95% CI, 1.28 to 2.07) and 2.09 (95% CI, 1.45 to 3.01), respectively, and those in the group with an estimated GFR of <45.0 mL/min per 1.73 m^2 were 3.95 (95% CI, 3.12 to 5.00) and 5.87 (95% CI, 4.13 to 8.36), respectively. Age and gender-adjusted analysis showed that the risk of cardiovascular death based on the estimated GFR followed a similar pattern, and that of non-cardiovascular death was not associated with estimated GFR (Fig. 4). The results of the trend test for risk supported the difference between the risks of cardiovascular and non-cardiovascular death with declining estimated GFR (p<0.001 and 0.230, respectively).

4. Discussion

Our findings suggested that about half of the patients who survived from AMI had an estimated GFR suggestive of CKD, and that

Table 1
Baseline patient characteristics according to the estimated GFR.

Variables	Total (N = 4550)	GFR, <45.0 (mL/min per 1.73 m^2) (N = 667)	GFR, 45.0–59.9 (N = 1274)	GFR, 60.0–74.9 (N = 1383)	GFR, ≥75.0 (N = 1226)	P-value
Women	1215 (26.7)	286 (42.9)	391 (30.7)	268 (19.4)	270 (22.0)	<0.001
Age	66.9 ± 12.1	74.6 ± 10.3	69.3 ± 10.8	64.9 ± 11.6	62.7 ± 12.3	<0.001
Body mass index, kg/m^2	23.5 ± 3.4	22.9 ± 3.4	23.4 ± 3.4	23.8 ± 3.3	23.6 ± 3.5	<0.001
Risk factors						
Hypertension	2596 (57.1)	448 (67.2)	763 (59.9)	733 (53.0)	652 (53.2)	<0.001
Hypercholesterolemia[a]	1890 (41.5)	246 (36.9)	517 (40.6)	594 (43.0)	533 (43.5)	0.024
Diabetes mellitus	1578 (34.7)	264 (39.6)	425 (33.4)	418 (30.2)	471 (38.4)	<0.001
Smoking	2533 (55.7)	294 (44.1)	624 (49.0)	839 (60.7)	776 (63.3)	<0.001
Prior MI	635 (14.0)	137 (20.5)	198 (15.5)	168 (12.1)	132 (10.8)	<0.001
Atrial fibrillation	42 (0.9)	10 (1.5)	19 (1.5)	10 (0.7)	3 (0.2)	0.003
Prior PCI	388 (8.5)	62 (9.3)	122 (9.6)	114 (8.2)	90 (7.3)	0.199
Prior CABG	91 (2.0)	26 (3.9)	24 (1.9)	24 (1.7)	17 (1.4)	0.002
In-hospital revascularization						
PCI	3423 (75.2)	420 (63.0)	991 (77.8)	1113 (80.5)	899 (73.3)	<0.001
CABG	160 (3.5)	36 (5.4)	49 (3.8)	43 (3.1)	32 (2.6)	0.012
Ejection fraction,%	52.6 ± 12.7	49.7 ± 14.0	51.8 ± 13.0	53.0 ± 12.2	54.6 ± 11.8	<0.001
Examinations						
HbA1c, %	6.1 ± 1.5	6.2 ± 1.4	6.0 ± 1.5	5.9 ± 1.4	6.3 ± 1.7	<0.001
Total-cholesterol, mg/dL	195.7 ± 41.3	189.0 ± 42.3	194.5 ± 40.7	197.8 ± 40.6	198.3 ± 41.8	<0.001
Triglyceride, mg/dL	105.0 [71.0–152.0]	101.0 [66.0–141.0]	104.0 [71.0–150.0]	107.0 [74.0–155.0]	105.0 [68.0–156.0]	0.02
HDL-cholesterol, mg/dL	46.6 ± 13.5	45.6 ± 13.5	46.5 ± 13.1	46.5 ± 13.5	47.5 ± 14.0	0.075
LDL-cholesterol, mg/dL	125.1 ± 36.2	120.3 ± 36.1	123.5 ± 36.0	127.5 ± 35.8	126.8 ± 36.6	<0.001
C-reactive protein, mg/dL	0.30 [0.10–0.90]	0.60 [0.20–2.82]	0.30 [0.10–0.90]	0.30 [0.10–0.70]	0.26 [0.10–0.70]	<0.001
Serum creatinine, mg/dL	0.80 [0.70–1.00]	1.40 [1.20–1.70]	1.00 [0.81–1.10]	0.80 [0.80–0.90]	0.60 [0.50–0.70]	<0.001
GFR, mL/min per 1.73 m^2	63.6 ± 19.0	34.1 ± 9.2	53.1 ± 4.2	67.1 ± 4.1	86.7 ± 10.8	<0.001
Uric acid, mg/dL	5.8 ± 5.3	7.1 ± 9.2	6.0 ± 6.8	5.6 ± 2.4	5.1 ± 1.9	<0.001
Medications at discharge						
ACEIs	2265 (49.8)	256 (38.4)	665 (52.2)	730 (52.8)	614 (50.1)	<0.001
ARBs	711 (15.6)	104 (15.6)	191 (15.0)	220 (15.9)	196 (16.0)	0.328
CCBs	1188 (26.1)	252 (37.8)	378 (29.7)	300 (21.7)	258 (21.0)	<0.001
Beta-blockers	1618 (35.6)	260 (39.0)	500 (39.2)	495 (35.8)	363 (29.6)	0.006
Nitrates	2354 (51.7)	405 (60.7)	720 (56.5)	706 (51.0)	523 (42.7)	<0.001
Statins	1154 (25.4)	137 (20.5)	327 (25.7)	367 (26.5)	323 (26.3)	<0.001
Warfarin	368 (8.1)	62 (9.3)	118 (9.3)	119 (8.6)	69 (5.6)	0.017
Aspirin	4021 (88.4)	560 (84.0)	1151 (90.3)	1276 (92.3)	1034 (84.3)	<0.001

Values are shown as means ± SD, numbers of patients (percentages), or medians (interquartile ranges).
ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin II-receptor blocker; CABG = coronary-artery bypass grafting; CCB = calcium-channel blocker; GFR = glomerular filtration rate; Hb = hemoglobin; HDL = high-density lipoprotein; LDL = low-density lipoprotein; MI = myocardial infarction; PCI = percutaneous coronary intervention.
[a] Total cholesterol level ≥ 220 mg/dL.

kidney disease characterized by several coexisting cumulative cardiovascular risks and co-morbid conditions continued to be closely associated with cardiovascular death even in the early stage over the long term.

The risk stratification according to four categories of estimated GFR determined in the present study does not necessarily correspond to the standard classification proposed by the National Kidney Foundation [2]. This results in emphasizing that many strategies for cardiovascular risks are required at an early stage of CKD to improve global outcomes of myocardial infarction and kidney disease.

The present study demonstrated that cardiovascular death accounted for more than half of all deaths in patients with any graded severity of CKD. Furthermore, the frequency of any type of non-fatal or fatal cardiovascular events increased in the patients with CKD. These results are consistent with those of a larger cohort study [3] including participants recruited in the Valsartan in Acute Myocardial Infarction Trial (VALIANT) [15]. However, the absolute incidence of adverse events in patients with myocardial infarction appeared lower in the present study than in VALIANT, and the prevalence of CKD in the present study was higher. These differences might be explained by the fact that the inclusion and exclusion criteria of participants in VALIANT were very strict, since it was a randomized, controlled trial to evaluate the efficacy and safety of blockades of the renin–angiotensin system. VALIANT included patients with AMI complicated by heart failure, left ventricular systolic dysfunction, or both, and excluded those with a baseline serum creatinine level of ≥2.5 mg/dL. In contrast, our cohort had a wider spectrum of kidney function. Ventricular function was relatively preserved among the enrolled patients who mostly underwent percutaneous coronary intervention during the index hospitalization. Our findings, in routine clinical practice, would compensate for the inability to generalize results from participants in clinical trials, following the internal consistency of the relationship between CKD and outcomes of cardiovascular disease including myocardial infarction. Furthermore, the long follow-up in the present study would enhance the certainty of the predominant effects of CKD on cardiovascular complications subsequent to AMI.

The benefits of aspirin, beta-blockers, statins, and blockades of the renin–angiotensin system in patients with myocardial infarction have been established [16,17]. Angiotensin-converting-enzyme inhibitors and angiotensin II-receptor blockers also reduce the progression of kidney disease [18,19]. Consequently, recent guidelines have concluded that pharmacological intervention with these drugs must be applied to patients with CKD at high risk for subsequent cardiovascular events [20–22]. However, the present study revealed a therapeutic gap between the guidelines and actual practice. Furthermore, caution regarding the administration of such drugs might be clinically advisable and appropriate to some extent for patients with advanced kidney dysfunction, due to vulnerability to some medications.

Percutaneous coronary intervention is an effective strategy for early coronary revascularization in acute coronary syndrome [16,17]. Although

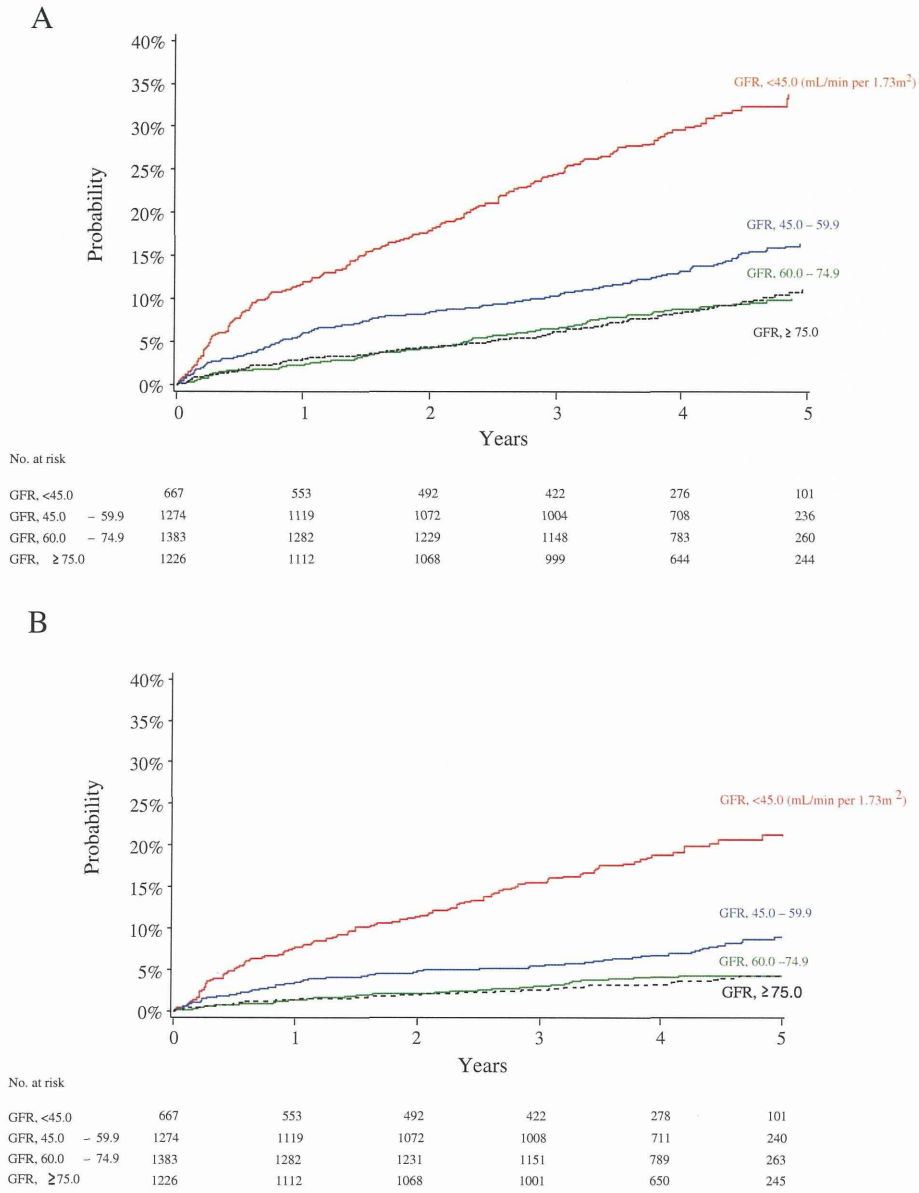

Fig. 2. Kaplan–Meier estimates of time to death from any cause (A) and to cardiovascular death (B) according to the estimated GFR at baseline. GFR, glomerular filtration rate.

we also found that the incidence of percutaneous coronary intervention for coronary revascularization was lowest in the group with the lowest estimated GFR, the patients in the present study apparently underwent this procedure more frequently than those in previous studies [3,4]. Even with these possible multiple interactions of kidney dysfunction with various confounders, reduced estimated GFR as a marker of kidney dysfunction has been considered a statistically independent predictor of long-term adverse outcomes [3,23].

5. Study limitations

The present study has several limitations. First, the MDRD equation estimating the GFR itself has limitations. We adopted the abbreviated MDRD equation from among several reliable choices to estimate GFR in the present study. Ethnicity might influence GFR estimation by serum creatinine-based equations, and the GFR could be underestimated in the population with GFR > 60.0 mL/min per 1.73 m^2 using calculations based on the abbreviated MDRD equation [24,25]. Although we used the equation modified by the Japanese coefficient, which is more predictive for the Japanese population, the equation was still not optimal for estimating GFR levels of >60.0 mL/min per 1.73 m^2 among Japanese [14]. Second, we cannot comment on the effects of the duration of kidney dysfunction and temporal changes in kidney function on the risk of adverse outcomes. Third, we did not evaluate the influence of nephropathy induced by contrast medium before and after acute myocardial infarction on risk. Fourth, we did not have information about urinary albumin or protein excretion, anemia, and other unmeasured factors that might drive the documented effect of the estimated baseline GFR on adverse outcomes. Fifth, we did not use a requirement for dialysis as a study endpoint, which possibly influenced morbidity and mortality. The final limitation is that the present study had no manifestations of an independent effect of kidney dysfunction on adverse outcomes subsequent to acute myocardial infarction. We described herein, the comprehensive characteristics of chronic kidney disease in the setting of acute myocardial infarction. The independent effects of chronic kidney disease on clinical outcomes will be addressed

	GFR	No. of Patient	No. of Event (%)	Crude Hazard Ratio (95%CI)			P-value for trend test
Primary Endpoint							
Total Death	<45.0	667	197 (29.5)	3.95	(3.12 – 5.00)	P<.001	<.001
	45.0 – 59.9	1274	176 (13.8)	1.63	(1.28 – 2.07)	P<.001	
	60.0 – 74.9	1383	123 (8.9)	1.01	(0.78 – 1.30)	P=.968	
	≥75.0	1226	107 (8.7)	1.00			
Secondary Endpoint							
Cardiovascular Death	<45.0	667	116 (17.4)	5.87	(4.13 – 8.36)	P<.001	<.001
	45.0 – 59.9	1274	89 (7.0)	2.09	(1.45 – 3.01)	P<.001	
	60.0 – 74.9	1383	54 (3.9)	1.12	(0.75 – 1.68)	P=.568	
	≥75.0	1226	42 (3.4)	1.00			
Non-cardiovascular Death	<45.0	667	81 (12.1)	2.70	(1.94 – 3.74)	P<.001	<.001
	45.0 – 59.9	1274	87 (6.8)	1.33	(0.96 – 1.83)	P=.085	
	60.0 – 74.9	1383	69 (5.0)	0.93	(0.66 – 1.30)	P=.669	
	≥75.0	1226	65 (5.3)	1.00			
Death from CHD or Non-fatal MI	<45.0	667	126 (18.9)	3.42	(2.58 – 4.53)	P<.001	<.001
	45.0 – 59.9	1274	112 (8.8)	1.41	(1.05 – 1.88)	P=.021	
	60.0 – 74.9	1383	98 (7.1)	1.10	(0.82 – 1.48)	P=.531	
	≥75.0	1226	78 (6.4)	1.00			
Fatal MI or Non-fatal MI	<45.0	667	51 (7.6)	1.92	(1.32 – 2.81)	P<.001	<.001
	45.0 – 59.9	1274	62 (4.9)	1.08	(0.75 – 1.55)	P=.667	
	60.0 – 74.9	1383	73 (5.3)	1.14	(0.81 – 1.62)	P=.455	
	≥75.0	1226	56 (4.6)	1.00			
Hospitalization for heart failure	<45.0	667	145 (21.7)	4.30	(3.25 – 5.69)	P<.001	<.001
	45.0 – 59.9	1274	109 (8.6)	1.46	(1.09 – 1.97)	P=.012	
	60.0 – 74.9	1383	84 (6.1)	1.00	(0.73 – 1.36)	P=.987	
	≥75.0	1226	74 (6.0)	1.00			
Sudden death	<45.0	667	17 (2.5)	4.45	(1.92 – 10.31)	P<.001	<.001
	45.0 – 59.9	1274	17 (1.3)	2.08	(0.90 – 4.83)	P=.087	
	60.0 – 74.9	1383	10 (0.7)	1.09	(0.43 – 2.76)	P=.859	
	≥75.0	1226	8 (0.7)	1.00			

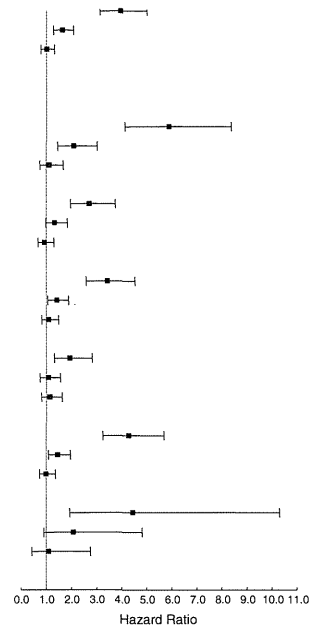

Fig. 3. Unadjusted hazard ratios for the primary and secondary endpoints. Unadjusted hazard ratios and P-values for trend test were obtained by Cox model. CHD = coronary heart disease; CI = confidence interval; GFR = glomerular filtration rate; MI = myocardial infarction.

in a subsequent report that focuses on particular subgroups of myocardial infarction. Despite these limitations, the current study provides important implications for the management of AMI concomitant with chronic kidney disease. In this context, aggressive and integral strategies for the prevention of kidney dysfunction are obviously essential to further improve the outcomes of AMI.

	GFR	Adjusted Hazard Ratio (95%CI)			P-value for trend test
Primary Endpoint					
Total Death	<45.0	1.92	(1.50 – 2.45)	P<.001	<.001
	45.0 – 59.9	1.10	(0.87 – 1.41)	P=.428	
	60.0 – 74.9	0.88	(0.68 – 1.14)	P=.347	
	≥75.0	1.00			
Secondary Endpoint					
Cardiovascular Death	<45.0	3.10	(2.15 – 4.48)	P<.001	<.001
	45.0 – 59.9	1.48	(1.02 – 2.14)	P=.039	
	60.0 – 74.9	1.00	(0.67 – 1.50)	P=.983	
	≥75.0	1.00			
Non-cardiovascular Death	<45.0	1.21	(0.86 – 1.71)	P=.265	.230
	45.0 – 59.9	0.87	(0.63 – 1.20)	P=.386	
	60.0 – 74.9	0.81	(0.57 – 1.13)	P=.211	
	≥75.0	1.00			
Death from CHD or Non-fatal MI	<45.0	2.44	(1.82 – 3.29)	P<.001	<.001
	45.0 – 59.9	1.17	(0.87 – 1.56)	P=.302	
	60.0 – 74.9	1.03	(0.77 – 1.39)	P=.835	
	≥75.0	1.00			
Fatal MI or Non-fatal MI	<45.0	1.71	(1.15 – 2.55)	P=.008	.042
	45.0 – 59.9	1.01	(0.70 – 1.46)	P=.940	
	60.0 – 74.9	1.12	(0.79 – 1.59)	P=.526	
	≥75.0	1.00			
Hospitalization for heart failure	<45.0	2.34	(1.74 – 3.14)	P<.001	<.001
	45.0 – 59.9	1.05	(0.78 – 1.41)	P=.757	
	60.0 – 74.9	0.91	(0.66 – 1.24)	P=.550	
	≥75.0	1.00			
Sudden death	<45.0	3.17	(1.32 – 7.62)	P=.010	.003
	45.0 – 59.9	1.72	(0.73 – 4.02)	P=.212	
	60.0 – 74.9	1.01	(0.40 – 2.56)	P=.987	
	≥75.0	1.00			

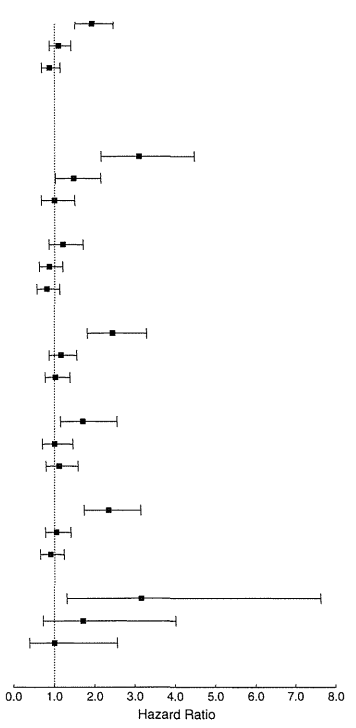

Fig. 4. Age and gender-adjusted hazard ratios for the primary and secondary endpoints. Adjusted hazard ratios and P-values for trend test were obtained by Cox model with adjustment for age and gender. CHD = coronary heart disease; CI = confidence interval; GFR = glomerular filtration rate; MI = myocardial infarction.

6. Conclusions

Even early-stage chronic kidney disease should be considered a powerful risk factor for long-term cardiovascular death after AMI with preserved left ventricular function in the acute revascularization era.

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References

[1] Eknoyan G, Lameire N, Barsoum R, Eckardt KU, Levin A, Levin N, et al. The burden of kidney disease: improving global outcomes. Kidney Int 2004;66:1310–4.
[2] Levey AS, Eckardt KU, Tsukamoto Y, Levin A, Coresh J, Rossert J, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int 2005;67:2089–100.
[3] Anavekar NS, McMurray JJ, Velazquez EJ, Solomon SD, Kober L, Rouleau JL, et al. Relation between renal dysfunction and cardiovascular outcomes after myocardial infarction. N Engl J Med 2004;351:1285–95.
[4] Schiele F, Legalery P, Didier K, Meneveau N, Seronde MF, Caulfield F, et al. Impact of renal dysfunction on 1-year mortality after acute myocardial infarction. Am Heart J 2006;151:661–7.
[5] Glynn LG, Reddan D, Newell J, Hinde J, Buckley B, Murphy AW. Chronic kidney disease and mortality and morbidity among patients with established cardiovascular disease: a West of Ireland community-based cohort study. Nephrol Dial Transplant 2007;22:2586–94.
[6] Okura N, Ogawa H, Katoh J, Yamauchi T, Hagiwara N. Long-term prognosis of patients with acute myocardial infarction in the era of acute revascularization (from the Heart Institute of Japan Acute Myocardial Infarction [HIJAMI] registry). Int J Cardiol 2012;159:205–10.
[7] Herzog CA, Ma JZ, Collins AJ. Poor long-term survival after acute myocardial infarction among patients on long-term dialysis. N Engl J Med 1998;339:799–805.
[8] Kasanuki H, Honda T, Haze K, Sumiyoshi T, Horie T, Yagi M, et al. A large-scale prospective cohort study on the current status of therapeutic modalities for acute myocardial infarction in Japan: rationale and initial results of the HIJAMI Registry. Am Heart J 2005;150:411–8.
[9] Yamaguchi J, Kasanuki H, Ishii Y, Yagi M, Ogawa H, Fujii SY, et al. Prognostic significance of serum creatinine concentration for in-hospital mortality in patients with acute myocardial infarction who underwent successful primary percutaneous coronary intervention (from the Heart Institute of Japan Acute Myocardial Infarction [HIJAMI] Registry). Am J Cardiol 2004;93:1526–8.
[10] Shiga T, Hagiwara N, Ogawa H, Takagi A, Nagashima M, Yamauchi T, et al. Sudden cardiac death and left ventricular ejection fraction during long-term follow-up after acute myocardial infarction in the primary percutaneous coronary intervention era: results from the HIJAMI-II registry. Heart 2009;95:216–20.
[11] Myocardial infarction redefined—a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. Eur Heart J 2000;21:1502–13.
[12] Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. Ann Intern Med 1999;130:461–70.
[13] Stevens LA, Coresh J, Greene T, Levey AS. Assessing kidney function-measured and estimated glomerular filtration rate. N Engl J Med 2006;354:2473–83.
[14] Imai E, Horio M, Nitta K, Yamagata K, Iseki K, Hara S, et al. Estimation of glomerular filtration rate by the MDRD study equation modified for Japanese patients with chronic kidney disease. Clin Exp Nephrol 2007;11:41–50.
[15] Pfeffer MA, McMurray JJ, Velazquez EJ, Rouleau JL, Kober L, Maggioni AP, et al. Valsartan, captopril, or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. N Engl J Med 2003;349:1893–906.
[16] Antman EM, Hand M, Armstrong PW, Bates ER, Green LA, Halasyamani LK, et al. 2007 focused update of the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the Canadian Cardiovascular Society endorsed by the American Academy of Family Physicians: 2007 Writing Group to review new evidence and update the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction, writing on behalf of the 2004 Writing Committee. Circulation 2008;117:296–329.
[17] Braunwald E, Antman EM, Beasley JW, Califf RM, Cheitlin MD, Hochman JS, et al. ACC/AHA guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction—2002: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). Circulation 2002;106:1893–900.
[18] Randomised placebo-controlled trial of effect of ramipril on decline in glomerular filtration rate and risk of terminal renal failure in proteinuric, non-diabetic nephropathy. The GISEN Group (Gruppo Italiano di Studi Epidemiologici in Nefrologia). Lancet 1997;349:1857–63.
[19] Brenner BM, Cooper ME, de Zeeuw D, Keane WF, Mitch WE, Parving HH, et al. Effects of losartan on renal and cardiovascular outcomes in patients with type 2 diabetes and nephropathy. N Engl J Med 2001;345:861–9.
[20] Sarnak MJ, Levey AS, Schoolwerth AC, Coresh J, Culleton B, Hamm LL, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Circulation 2003;108:2154–69.
[21] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003;289:2560–72.
[22] K/DOQI clinical practice guidelines on hypertension and antihypertensive agents in chronic kidney disease. Am J Kidney Dis 2004;43:S1–S290.
[23] Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N Engl J Med 2004;351:1296–305.
[24] Rule AD, Larson TS, Bergstralh EJ, Slezak JM, Jacobsen SJ, Cosio FG. Using serum creatinine to estimate glomerular filtration rate: accuracy in good health and in chronic kidney disease. Ann Intern Med 2004;141:929–37.
[25] Ma YC, Zuo L, Chen JH, Luo Q, Yu XQ, Li Y, et al. Modified glomerular filtration rate estimating equation for Chinese patients with chronic kidney disease. J Am Soc Nephrol 2006;17:2937–44.