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ORIGINAL RESEARCH

Renal Function and its Time-Sensitive Influence on Survival Rates of Acute Myocardial Infarction

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BACKGROUND: Primary percutaneous coronary intervention is the preferred treatment for acute myocardial infarction. However, in patients with chronic kidney disease (CKD), the use of contrast media can exacerbate renal dysfunction, often necessitating alternative strategies. The impact of CKD on acute myocardial infarction prognosis, particularly in the context of percutaneous coronary intervention, is not fully understood.

METHODS AND RESULTS: This study utilized real-world registry data from the OACIS (Osaka Acute Coronary Insufficiency Study) to evaluate prognosis across different CKD grades, including advanced

CKD and hemodialysis. From a database of 12 093 patients with acute myocardial infarction, we identified 8411 patients with renal function data at admission (a median follow-up period of 1765 days). These patients were classified into 8 CKD categories based on estimated glomerular filtration rate (eGFR); G1 (eGFR≥90 mL/min per 1.73 m^2): n=1122, G2 (90>eGFR ≥60 mL/min per 1.73 m^2): n=3588, G3a (60>eGFR≥45 mL/min per 1.73 m^2): n=1923, G3b (45>eGFR≥30 mL/min per 1.73 m^2): n=1030, G4: (30>eGFR≥15 mL/min per 1.73 m^2): n=473, G5a: (15>eGFR≥8 mL/min per 1.73m^2): n=80, G5b: (eGFR<8 mL/min per 1.73 m^2): n=53 and hemodialysis: n=142. Percutaneous coronary intervention rates declined with advancing CKD, reaching the lowest in G5a (80.3%) but increasing again in G5b and hemodialysis groups (≈90%). Thirty-day all-cause mortality rates increased with CKD severity, with a notable reduction in G5b (9.4%) before rising again in patients with hemodialysis (16.9%). Long-term data showed a progressive worsening of prognosis with advanced CKD, culminating in the poorest outcomes among patients with hemodialysis.

CONCLUSIONS: This study demonstrated differential impacts of CKD severity on short- and long-term clinical outcomes in the context of patients with acute myocardial infarction.

Key Words: acute myocardial infarction ■ chronic kidney disease ■ hemodialysis ■ primary percutaneous coronary intervention ■ real-world data

In the context of acute myocardial infarction (AMI), primary percutaneous coronary intervention (PCI) is currently the first-line treatment. However, in patients with chronic kidney disease (CKD), the use of contrast media can exacerbate renal dysfunction, often leading to alternative strategies being used. The

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CLINICAL PERSPECTIVE

What Is New?

- In general, patients with acute myocardial infarction with a higher stage of chronic kidney disease or on hemodialysis exhibit progressively worse short- and long-term outcomes.
- However, this real-world study indicated that patients with severe chronic kidney disease (estimated glomerular filtration rate <8 mL/min per 1.73 m^2) but not on hemodialysis nevertheless demonstrated unexpectedly favorable short-term survival rates, potentially due to the increased frequency of primary percutaneous coronary intervention.

What Are the Clinical Implications?

- In patients with acute myocardial infarction and advanced chronic kidney disease, aggressive revascularization strategies might be given precedence over the preservation of renal function. Future prospective randomized trials are warranted to investigate the potential advantages of the strategy.

Nonstandard Abbreviations and Acronyms

OACIS	Osaka Acute Coronary Insufficiency Study

real-world impact of this on overall prognosis, considering the comorbidity of CKD and AMI, is not fully understood. In Japan, the number of patients with CKD is estimated to be 13.3 million, and the prevalence of patients with hemodialysis has been gradually increasing.[1] CKD is recognized as an independent risk factor for cardiovascular events in patients with AMI, significantly influencing treatment strategies due to potential renal deterioration from contrast media during PCI.[2–4]

This study aims to comprehensively evaluate the differences in prognosis across various CKD grades, including advanced CKD and patients undergoing hemodialysis. Utilizing real-world registry data from a large-scale prospective cohort study of patients with AMI OACIS (Osaka Acute Coronary Insufficiency Study), we seek to elucidate how different CKD stages impact both short- and long-term outcomes. Understanding these differences is crucial for optimizing treatment strategies and improving prognostic outcomes for patients with AMI with varying degrees of renal impairment.

METHODS

Our study data will not be made available to other researchers for purposes of reproducing the results because of institutional review board restrictions.

Study Subjects

The OACIS is a prospective, multicenter, observational study in which 25 collaborating hospitals in the Osaka region of Japan recorded demographic, procedural, and outcome data from patients with AMI. The OACIS is registered in the University Hospital Medical Information Network Clinical Trials Registry (UMINCTR) in Japan (ID: UMIN000004575). The study was conducted in accordance with the Declaration of Helsinki Ethical Principles. Written informed consent was obtained at enrollment. The present study was approved by the institutional review board of Osaka University Hospital (ID: 14360-9). A detailed description of the OACIS has been published elsewhere.[5–9] A total of 12 093 patients with AMI were registered in the OACIS registry between 1998 and 2014. A diagnosis of AMI was made if the patient fulfilled at least 2 of the following 3 criteria: (1) clinical history of central chest pressure, pain, or tightness lasting ≥30 minutes, (2) ST-segment elevation >0.1 mV in at least 1 standard or 2 precordial leads, and (3) a rise in serum creatine phosphokinase concentration to more than twice the normal laboratory value. Collaborating hospitals were encouraged to enroll consecutive cases of AMI irrespective of their treatment strategy or outcome. Of these, 3824 patients with renal function data not available on hospital admission were excluded from the current analysis. Finally, we identified 8411 consecutive cases where the estimated glomerular filtration rate (eGFR: mL/min per 1.73 m^2) was available upon admission. We classified these patients into 8 categories according to their renal function on admission; G1 (eGFR ≥90 mL/min per 1.73 m^2), G2 (90>eGFR≥60 mL/min per 1.73 m^2), G3a (60>eGFR ≥45 mL/min per 1.73 m^2), G3b (45>eGFR ≥30 mL/min per 1.73 m^2), G4: (30>eGFR ≥15 mL/min per 1.73 m^2), G5a (15>eGFR≥8 mL/min per 1.73 m^2), G5b (eGFR<8 mL/min per 1.73 m^2) and hemodialysis based on the guideline.[10,11] The grade 5 was further divided into G5a group, which has not yet crossed the border of indication of hemodialysis and G5b group, which has already reached to the indication level of hemodialysis according to the guideline but not yet received hemodialysis.[12]

Data Collection and Patient Follow-Up

Investigative cardiologists and research coordinators collected patient demographic and clinical data by direct interview of patients or their family members. Patient follow-up was conducted 3 and 12 months

after discharge for AMI and annually thereafter for up to 5 years. Information on the clinical event was collected by local investigators when visiting outpatient clinics or through verbal or written contact with patients or family members. All data were transmitted to the data collection center (Osaka University Hospital) for processing and analysis.

Study End Point

The primary end point of this study was all-cause mortality. Short-term and long-term outcomes were evaluated at 30 days and 5 years after the onset of AMI, respectively. The definitions of the causes of death in our study are tabulated in Table S1. Of note, cardiovascular death within 30 days after AMI is generally defined as death due to AMI. However, in this study, we further identified detailed causes such as fatal arrhythmias and cardiogenic shock, even within 30 days of AMI study.[13,14] Three researchers reviewed the causes of death described in the data set. In cases of any doubt, they discussed until a consensus was reached as to the most likely cause of death. Two matching votes were needed to finalize the classification.

Statistical Analysis

Categorical variables were presented as number (frequency) and compared using the χ^2 test or Fisher exact test among 8 groups based on level of kidney function. Continuous variables were presented as medians (interquartile range) and compared using 1-way ANOVA for normal distribution or the Kruskal–Wallis test for skewed distribution between 8 groups. Missing data were imputed by random forest imputation using "missForest" package in R before the following logistic regression analysis and the Cox regression analysis. The "missForest" algorithm fits a random forest model on the observed data to predict missing values, under the assumption of missing at random.[15] The Cochran-Armitage trend test was used to examine the trends of 30-day mortality among the different CKD grades. Univariable and multivariable logistic regression models were utilized to assess whether CKD grade was associated with 30-day all-cause, cardiovascular, and noncardiovascular death. We defined G2 as a reference, since G2 had the largest number of patients. The multivariable logistic regression model was used to evaluate the impact of CKD grade with adjustment for the following clinically relevant covariates selected based on clinical consensus: age, sex, diabetes, hypertension, dyslipidemia, prior myocardial infarction, prior cerebrovascular disease, primary PCI, cardiac pulmonary arrest on admission, creatinine kinase >3000 IU/L, and medications such as catecholamine, β-blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor blocker, diuretics, and statins. As for all-cause death after hospital admission up to final follow-up, differences in survival curves among the 8 CKD grades were estimated by the Kaplan–Meier method and analyzed using the log-rank test. Also, we performed landmark analysis at 30 days from hospital admission in order to separately evaluate the impact of CKD grades on short- and long-term prognosis. The trend among different CKD grades was analyzed by Log-rank trend test. Considering the proportional hazards assumption, the follow-up period was segmented into 2 distinct phases: (1) the initial 6 months starting from the landmark point at 30 days, and (2) the subsequent phase from 6 months to 5 years. The impact of CKD on all-cause mortality was assessed using a univariable and multivariable Cox proportional hazard model, with G2 as reference, across the

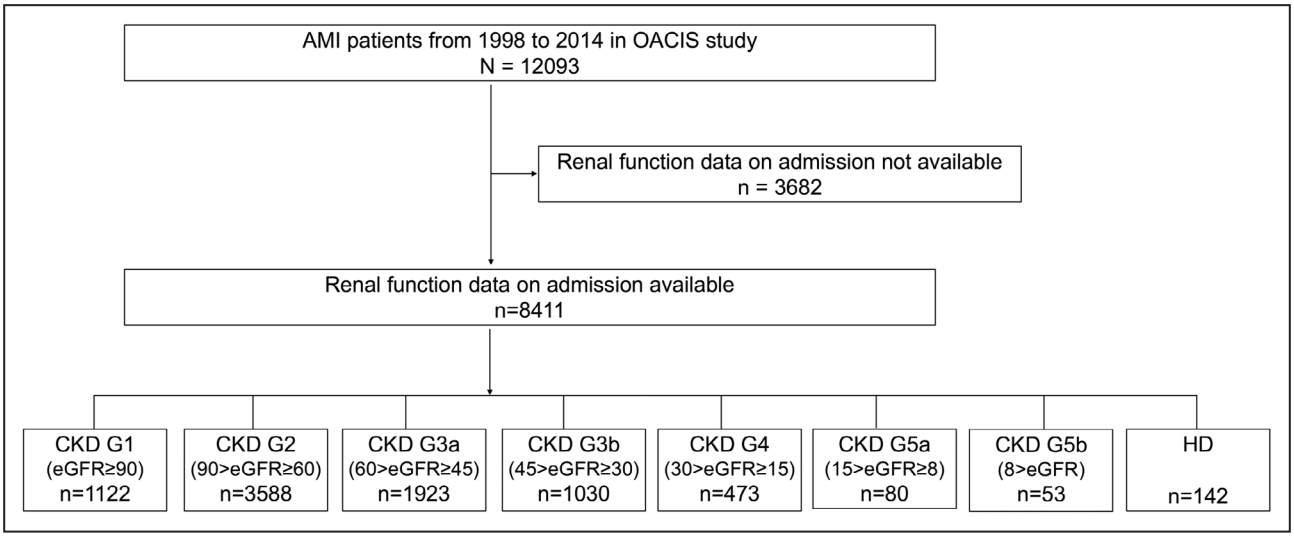

Figure 1. Study population.
AMI indicates acute myocardial infarction; CKD G, chronic kidney disease grade; eGFR, estimated glomerular filtration rate; HD, hemodialysis; and OACIS; Osaka Acute Coronary Insufficiency Study.

Table 1. Baseline Characteristics

	All patients	G1	G2	G3a	G3b	G4	G5a	G5b	HD	Data missing	
		(eGFR≥90)	(90>eGFR≥60)	(60>eGFR≥45)	(45>eGFR≥30)	(30>eGFR≥15)	(15>eGFR≥8)	(8>eGFR)			
Number	8411	1122	3588	1923	1030	473	80	53	142	(%)	*P* value
Patient data											
Age (y)	68 [59– 76]	59 [50– 66]	66 [58– 73]	70 [62–- 77]	74 [67– 82]	77 [70– 83]	76 [66– 83]	69 [63– 79]	67 [59– 76]	0	<0.001
Male	6327 (75.2)	863 (76.9)	2847 (79.3)	1463 (76.1)	695 (67.5)	274 (57.9)	47 (58.8)	27 (50.9)	111 (78.2)	0	<0.001
BMI (kg/m^2)	23.5 [21.4– 25.7]	23.8 [21.7– 26.3]	23.7 [21.7– 25.8]	23.5 [21.4– 25.6]	23.2 [20.9– 25.4]	22.8 [20.4– 25.4]	22.8 [20.8– 25.4]	22.7 [19.5– 24.5]	22.3 [20.1– 24.6]	5.1	<0.001
Hypertension	5179 (63.9)	559 (51.5)	2127 (61.1)	1233 (66.7)	727 (74.8)	368 (80.3)	58 (75.3)	40 (78.4)	126 (92.0)	3.6	<0.001
Diabetes	2765 (34.0)	383 (34.7)	1054 (30.2)	605 (32.7)	369 (38.0)	225 (49.6)	41 (53.9)	28 (54.9)	83 (58.9)	3.3	<0.001
Dyslipidemia	3619 (45.1)	502 (46.5)	1608 (46.6)	853 (46.5)	397 (41.6)	168 (37.3)	27 (37.0)	19 (38.0)	43 (31.9)	4.7	<0.001
Cerebrovascular disease	809 (10.0)	48 (4.4)	274 (8.0)	196 (10.7)	155 (15.8)	96 (21.1)	17 (22.1)	6 (12.0)	23 (16.2)	4.3	<0.001
Smoking	5039 (62.1)	832 (75.2)	2278 (65.0)	1070 (58.2)	508 (52.5)	198 (45.5)	34 (47.2)	19 (40.4)	64 (47.1)	3.6	<0.001
Prior myocardial infarction	921 (11.2)	57 (5.1)	336 (9.5)	244 (13.0)	158 (15.8)	76 (16.6)	14 (17.9)	7 (13.5)	21 (15.0)	1.8	<0.001
Prior PCI	761 (9.3)	60 (5.4)	316 (8.9)	177 (9.3)	125 (12.4)	63 (13.8)	10 (12.7)	8 (15.7)	30 (21.6)	3.1	<0.001
Status on admission											
STEMI	6930 (84.0)	952 (85.5)	2979 (84.0)	1580 (84.6)	825 (82.7)	379 (81.9)	59 (74.7)	43 (81.1)	113 (80.7)	1.8	0.115
CPA on admission	266 (3.3)	5 (0.5)	80 (2.3)	97 (5.2)	56 (5.7)	21 (4.7)	4 (5.1)	1 (2.0)	8 (5.7)	4.3	<0.001
Systolic blood pressure (mm Hg)	135 [115– 156]	140 [124– 160]	138 [120– 158]	131 [110– 153]	125 [101– 150]	124 [98– 146]	122 [100– 144]	140 [122– 160]	132 [114– 156]	6	<0.001
Diastolic blood pressure (mm Hg)	80 [68– 92]	84 [74– 96]	80 [70– 94]	78 [64– 90]	73 [60– 88]	70 [58– 85]	70 [58– 84]	80 [67– 86]	73 [60– 90]	8.3	<0.001
Heart rate (beats/min)	78 [65– 92]	78. [67– 90]	78 [66– 90]	78 [64– 92]	80 [60– 100]	82 [60– 100]	82 [60– 102]	85 [72– 100]	88 [7– 105]	6	0.041
Killip class ≥II	1718 (21.4)	79 (7.3)	431 (12.5)	481 (26.2)	394 (41.2)	234 (53.3)	44 (58.7)	22 (45.8)	49 (39.8)	4.6	<0.001
Creatinine (mg/dL)	0.90 [0.70– 1.10]	0.60 [0.50– 0.66]	0.80 [0.70– 0.90]	1.04 [0.96– 1.10]	1.31 [1.20– 1.50]	2.00 [1.70– 2.30]	3.63 [3.39– 4.51]	7.10 [6.20– 8.86]	8.21 [6.09– 10.43]	1.6	<0.001
Procedure											
Primary PCI	7189 (93.8)	987 (96.0)	3142 (95.1)	1646 (94.4)	858 (92.0)	354 (88.3)	57 (80.3)	48 (90.6)	116 (89.9)	8.9	<0.001
CABG	163 (2.3)	21 (2.1)	49 (1.6)	30 (1.8)	32 (3.6)	14 (3.9)	3 (4.8)	6 (12.5)	7 (5.6)	14.4	<0.001
Infarct-related artery											
Left main trunk	239 (3.1)	9 (0.9)	68 (2.1)	73 (4.2)	57 (6.2)	22 (5.6)	4 (6.6)	4 (8.3)	10 (7.5)	8.9	<0.001
Left anterior descending	3607 (47.1)	571 (54.2)	1665 (50.3)	754 (43.1)	362 (39.4)	138 (35.0)	31 (50.8)	28 (58.3)	60 (45.1)	8.9	<0.001
Left circumflex	1278 (16.7)	197 (18.7)	537 (16.2)	278 (15.9)	147 (16.0)	81 (20.6)	10 (16.4)	4 (8.3)	24 (18.0)	8.9	0.115
Right coronary	2757 (36.0)	294 (27.9)	1121 (33.8)	702 (40.1)	382 (41.6)	179 (45.4)	23 (37.7)	17 (35.4)	51 (38.3)	8.9	<0.001
Multivessel disease	3795 (47.8)	442 (40.6)	1536 (44.5)	925 (50.5)	548 (57.1)	247 (61.1)	43 (66.2)	33 (63.5)	87 (64.4)	5.7	<0.001

(*Continued*)

Table 1. Continued

	All patients	G1	G2	G3a	G3b	G4	G5a	G5b	HD	Data missing	
		(eGFR≥90)	(90>eGFR≥60)	(60>eGFR≥45)	(45>eGFR≥30)	(30>eGFR≥15)	(15>eGFR≥8)	(8>eGFR)			
Number	8411	1122	3588	1923	1030	473	80	53	142	(%)	*P* value
Peak-CK (IU/L)	1979 [913– 3876]	2113 [1029– 4029]	1903 [863– 3611]	2116 [966– 4035]	2020 [890– 4380]	1944 [876– 3995]	1630 [1031– 3063]	798 [393– 1931]	1395 [696– 2486]	5.2	<0.001
Peak-CK >3000 IU/L	2718 (34.1)	400 (37.2)	1086 (31.7)	664 (36.4)	354 (36.3)	150 (34.4)	18 (25.0)	10 (20.0)	28 (20.1)	5.2	<0.001
Peak-CK-MB (IU/L)	177 [81– 352]	210 [102– 372	174 [80– 337]	179 [81– 363]	177 [78– 399]	176 [73– 330]	156 [85– 409]	89 [48– 159]	165 [80– 294]	10.8	<0.001
TIMI grade 3 post PCI	6541 (89.7)	923 (91.7)	2963 (92.2)	1479 (87.3)	761 (87.3)	290 (82.2)	46 (79.3)	40 (85.1)	106 (90.6)	13.3	<0.001

Data are expressed as median [interquartile range] or number (percentage).
BMI indicates body mass index; CABG, coronary artery bypass grafting; CK, creatine kinase; CK-MB, creatine kinase myocardial band; CPA, cardiopulmonary arrest; eGFR, estimated glomerular filtration rate; HD, hemodialysis; PCI, percutaneous coronary intervention; STEMI, ST-segment–elevation myocardial infarction; and TIMI, thrombolysis in myocardial infarction.

2 consecutive time periods. The proportional hazards assumption for CKD grade concerning each end point was validated through the analysis of Schoenfeld residuals (P >0.05) (Table S2). The distribution of Martingale and Deviance residuals for each Cox proportional hazard model is shown in Figures S1, S2, S3, and S4. Martingale and Deviance residuals in each Cox proportional hazard model exhibited positive skewness. Impact of CKD grade was assessed with adjustment for the clinically relevant covariates selected based on clinical consensus: age, sex, diabetes, hypertension, dyslipidemia, peak creatinine kinase >3000 (IU/L), β-blockers, angiotensin-converting enzyme or angiotensin II receptor blocker, diuretics, and statins on discharge were analyzed. As a sensitivity analysis, we assessed the short-term and long-term impact of CKD by developing 2 alternative models each incorporating additional covariates. These models are summarized in Tables S3 and S4. Moreover, we conducted complete case analyses using univariable and multivariable logistic regression and Cox proportional hazard models as a sensitivity analysis. All analyses were performed using R software (version 4.0.0; R Foundation for Statistical Computing, Vienna, Austria) with R Studio (version 3,6,1; Boston, MA).

RESULTS

Baseline Characteristics

From a database of the OACIS registry (N=12 093), we identified 8411 patients with renal function data available on admission (Figure 1). These patients were classified into 8 CKD categories based on eGFR: G1 (eGFR ≥90 mL/min per 1.73 m^2): n=1122, G2 (90>eGFR ≥60 mL/min per 1.73 m^2): n=3588, G3a (60>eGFR ≥45 mL/min per 1.73 m^2): n=1923, G3b (45>eGFR ≥30 mL/min per 1.73 m^2): n=1030, G4: (30>eGFR ≥15 mL/min per 1.73 m^2): n=473, G5a: (15>eGFR≥8 mL/min per 1.73 m^2): n=80, G5b: (eGFR<8 mL/min per 1.73 m^2): n=53 and hemodialysis: n=142. Baseline clinical characteristics by CKD grade and hemodialysis are shown in Table 1. Patients with advanced CKD (G4 and G5) and hemodialysis had more comorbidities such as diabetes and hypertension, more multivessel disease, and more frequently presented Killip class ≥II than G1-G3 groups. Performance rate of primary PCI exhibited a gradual decrement as the CKD grade advanced (Figure 2) with the lowest rate of 80% in G5a group (80.3%). However, there was a slight increase again in patients with G5b and hemodialysis to ≈90%.

Treatments and Complications Within 1 Week From Admission

Medical treatments during 1 week from hospital admission stratified by CKD grades are shown in Table 2. β-blockers, angiotensin-converting enzyme inhibitors,

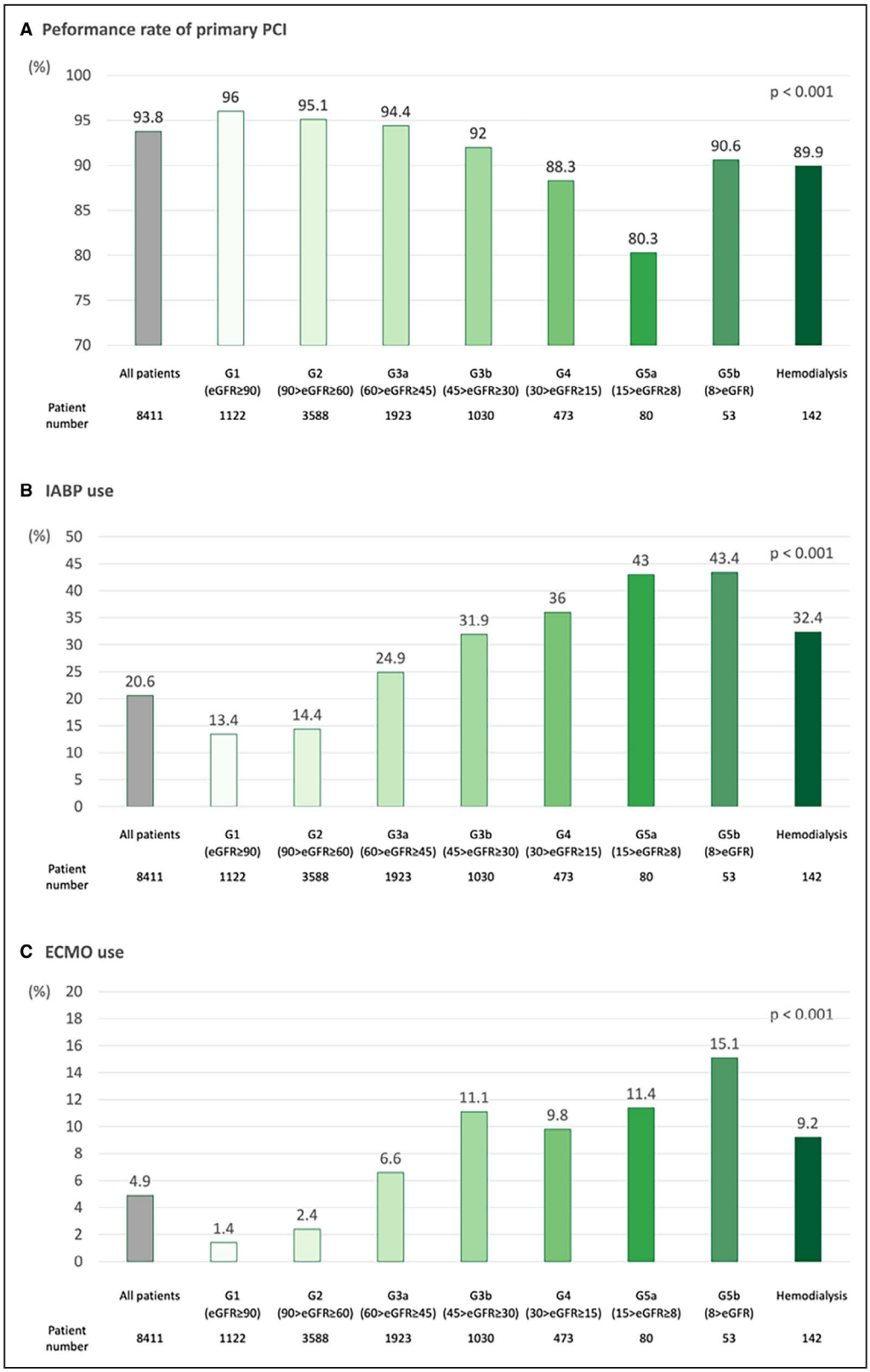

Figure 2. Performance rates of primary PCI, IABP, and ECMO.
Bar charts indicate the performance rates of primary percutaneous coronary intervention (PCI) (**A**), intra-aortic balloon pumping (IABP) (**B**), and extracorporeal membrane oxygenation (ECMO) (**C**). CKD indicates chronic kidney disease; and eGFR, estimated glomerular filtration rate.

Table 2. Treatment in the First Week

	All patients	G1	G2	G3a	G3b	G4	G5a	G5b	HD	Data missing	
		(eGFR≥90)	(90>eGFR≥60)	(60>eGFR≥45)	(45>eGFR≥30)	(30>eGFR≥15)	(15>eGFR≥8)	(8>eGFR)			
Number	8411	1122	3588	1923	1030	473	80	53	142	(%)	*P* value
Catecholamine use	2351 (28.2)	160 (14.4)	653 (18.4)	622 (32.7)	517 (50.7)	283 (60.5)	55 (69.6)	23 (45.1)	59 (41.5)	1	<0.001
ACEI use	3350 (40.2)	482 (43.3)	1554 (43.7)	780 (41.0)	358 (35.1)	99 (21.2)	14 (17.7)	8 (15.7)	38 (26.8)	1	<0.001
ARB use	2298 (27.6)	361 (32.4)	1031 (29.0)	515 (27.0)	249 (24.4)	115 (24.6)	16 (20.3)	10 (19.6)	38 (26.8)	1	<0.001
ACEI or ARB use	5441 (65.3)	813 (73.0)	2505 (70.5)	1243 (65.3)	577 (56.6)	202 (43.2)	28 (35.4)	18 (35.3)	71 (50.0)	1	<0.001
B-blocker use	3544 (42.5)	580 (52.1)	1655 (46.6)	765 (40.2)	362 (35.5)	144 (30.8)	19 (24.1)	16 (31.4)	66 (46.5)	1	<0.001
Statin use	3037 (36.5)	464 (41.7)	1459 (41.1)	678 (35.6)	285 (28.0)	119 (25.4)	17 (21.5)	12 (23.5)	39 (27.5)	1	<0.001
Diuretics use	3444 (41.3)	318 (28.6)	1222 (34.4)	877 (46.1)	583 (57.2)	328 (70.1)	50 (63.3)	23 (45.1)	32 (22.5)	1	<0.001
DAPT	6321 (78.2)	880 (79.1)	2799 (79.8)	1455 (79.1)	717 (75.4)	292 (69.0)	41 (59.4)	37 (77.1)	100 (76.3)	3.9	<0.001
ASA	7811 (99.7)	1086 (99.8)	3383 (99.8)	1775 (99.7)	922 (99.7)	405 (99.0)	68 (98.6)	45 (97.8)	127 (100.0)	6.8	0.034
P2Y12i	6571 (99.8)	904 (100.0)	2919 (99.8)	1513 (99.7)	743 (99.7)	306 (99.7)	42 (100.0)	40 (100.0)	104 (100.0)	21.7	0.864
Anticoagulation*	8204 (97.9)	1098 (98.0)	3522 (98.4)	1870 (97.7)	995 (97.0)	450 (95.7)	79 (100.0)	52 (98.1)	138 (97.2)	0.3	0.004
ECMO use	409 (4.9)	16 (1.4)	87 (2.4)	126 (6.6)	113 (11.1)	46 (9.8)	9 (11.4)	8 (15.1)	13 (9.2)	0.9	<0.001
IABP use	1718 (20.6)	149 (13.4)	513 (14.4)	474 (24.9)	326 (31.9)	169 (36.0)	34 (43.0)	23 (43.4)	46 (32.4)	0.9	<0.001

Data are expressed as number (percentage) with listwise deletion. ACEI indicates angiotensin converting enzyme inhibitor; ARB, angiotensin II receptor blocker; ASA, acetylsalicylic acid; DAPT, dual antiplatelet therapy; ECMO, extracorporeal membrane oxygenation; eGFR, estimated glomerular filtration rate; HD, hemodialysis; IABP, intra-aortic balloon pumping; and P2Y12i, P2Y12 inhibitor.

*Anticoagulation indicates intravenous heparin use and/or oral anticoagulants.

Table 3. Complications in the First Week

	All patients	G1	G2	G3a	G3b	G4	G5a	G5b	HD	Data missing	
		(eGFR≥90)	(90>eGFR≥60)	(60>eGFR≥45)	(45>eGFR≥30)	(30>eGFR≥15)	(15>eGFR≥8)	(8>eGFR)			
Number	8411	1122	3588	1923	1030	473	80	53	142	(%)	*P* value
Recurrent AMI	214 (2.6)	32 (2.9)	92 (2.6)	52 (2.7)	20 (1.9)	11 (2.4)	3 (3.8)	1 (1.9)	1 (0.7)	0.4	0.687
Cardiogenic shock	791 (9.4)	28 (2.5)	159 (4.4)	242 (12.6)	196 (19.0)	122 (26.0)	21 (26.6)	10 (19.2)	19 (13.4)	0.3	<0.001
VT/VF	1429 (17.1)	147 (13.1)	525 (14.7)	393 (20.5)	232 (22.6)	93 (19.8)	14 (17.7)	11 (21.2)	38 (26.8)	0.4	<0.001
Major bleeding	367 (4.4)	21 (1.9)	90 (2.5)	98 (5.1)	88 (8.6)	49 (10.4)	11 (13.9)	10 (19.2)	15 (10.7)	0.5	<0.001
Stroke	121 (1.4)	6 (0.5)	38 (1.1)	29 (1.5)	27 (2.6)	13 (2.8)	1 (1.3)	3 (5.8)	3 (2.1)	0.5	<0.001
Mechanical complication	198 (2.4)	12 (1.1)	53 (1.5)	47 (2.5)	43 (4.2)	35 (7.4)	3 (3.8)	1 (1.9)	4 (2.8)	0.3	<0.001
Cardiac rupture	122 (1.5)	12 (1.1)	33 (0.9)	31 (1.6)	30 (2.9)	15 (3.2)	0 (0.0)	1 (1.9)	1 (0.7)	0.3	<0.001
Ventricular septal perforation	43 (0.5)	0 (0.0)	12 (0.3)	9 (0.5)	8 (0.8)	11 (2.3)	2 (2.5)	1 (1.9)	0 (0.0)	0.3	<0.001
Ruptured mitral chordae tendineae	39 (0.5)	0 (0.0)	9 (0.3)	9 (0.5)	7 (0.7)	9 (1.9)	1 (1.3)	0 (0.0)	3 (2.1)	0.3	<0.001

Data are expressed as number (percentage) with listwise deletion. AMI indicates acute myocardial infarction; eGFR, estimated glomerular filtration rate; HD, hemodialysis; and VT/VF, ventricular tachycardia/ventricular fibrillation.

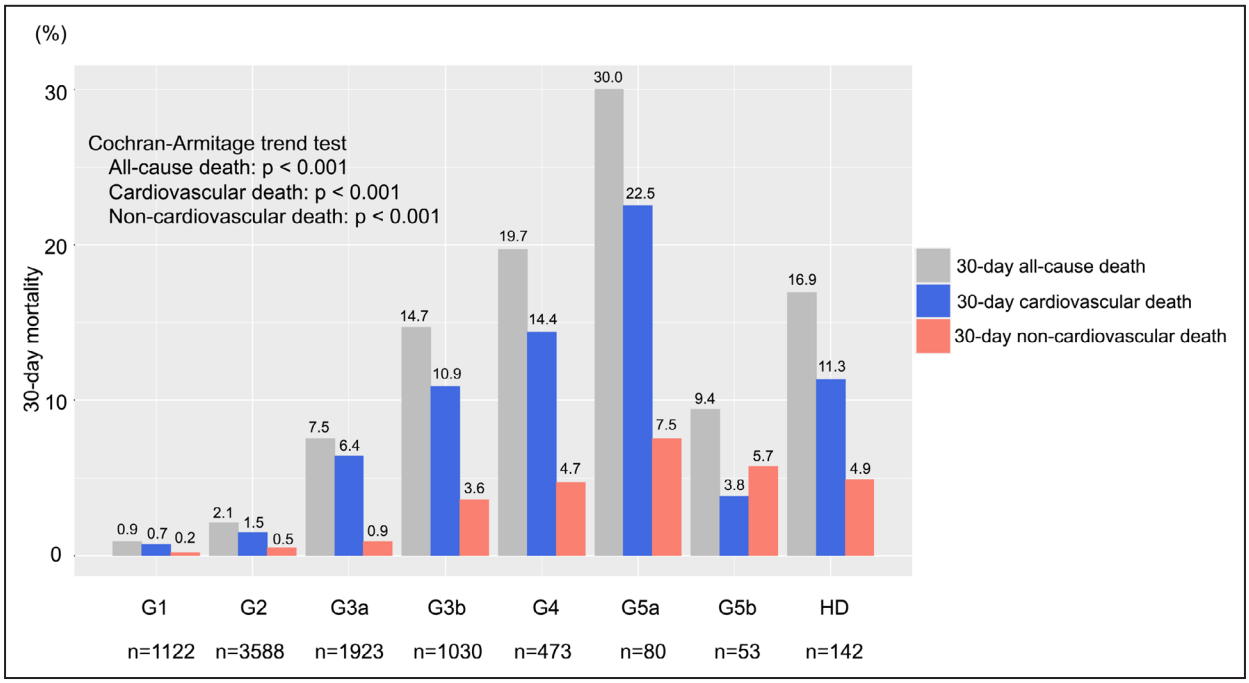

Figure 3. Thirty-day mortality stratified by CKD grades.
Bar chart indicates the incidence of short-term death stratified by CKD grade. CKD indicates chronic kidney disease.

and statins were less frequently used as the CKD grade progressed. Extracorporeal membrane oxygenation and intra-aortic balloon pumping were more frequently used as the CKD grade advanced, with the highest rate in the G5b group (15.1% and 43.4%, respectively) (Figure 2). Cardiovascular complications during 1 week from hospital admission stratified by CKD grades are summarized in Table 3. Patients with a higher CKD grade more frequently experienced the cardiovascular complications.

Clinical End Point

30 Days Short-Term Follow-up Data

Among total 8411 patients, 526 patients (6.3%) died within 30 days after admission. All-cause and cardiovascular mortality rates increased with advancing CKD grade (Figure 3). Notably, there was, however, a marked decrease to all-cause mortality rate of 9.4% and cardiovascular mortality rate of 3.8% for G5b patients, before rising again to 16.9% and 11.3% among patients with hemodialysis, respectively. Logistic regression models for 30-day mortality are shown in Figure 4. These results showed a distinct pattern at G5b group. Specifically, a significant reduction in mortality was observed for both all-cause death and cardiovascular death at this group, whereas no significant reduction was noted in noncardiovascular death. Cardiogenic shock was the most common cause of death in almost all CKD groups (Figure 5). However, in G5b groups, cardiovascular cause of mortality markedly decreased. The 2 alternative models with additional covariates and the complete case analyses consistently showed similar results (Figures S5 and S6). Kaplan–Meier curves up to 30 days are shown in Figure 6.

Five-Year Long-Term Follow-Up Data

During a median follow-up period of 1765 [492, 1825] days, 1209 patients (14.4%) died. Landmark analysis at 30 days showed that there was a significant difference in the survival rate across different CKD grades (Kaplan–Meier estimated 5-year survival rate: G1 95.9%, G2 93.0%, G3a 87.8%, G3b 80.6%, G4 66%, G5a 71.0%, G5b 63.4%, hemodialysis 63.3%, Log-rank test P <0.001, Log-rank trend test, P <0.001) (Figure 6). Cox proportional hazard model showed incrementally worse prognosis as the CKD grade advanced, with the worst clinical outcome in G5b patients during 6 months (Figure 7) and in patients with hemodialysis during the period from 6 months to 5 years (Figure 8). The marked drop observed in the G5b group in short-term data was not found in the long-term follow-up data. Cardiovascular cause of death increased as the CKD grade got advanced (Figure 9). The 2 alternative models with additional covariates consistently showed similar results (Figures S7 and S8). The complete case analysis of Cox proportional hazard model during 6 months from landmark at 30 days indicated that certain models failed to converge due to the reducing number of observed events (Figure S9); conversely, during the

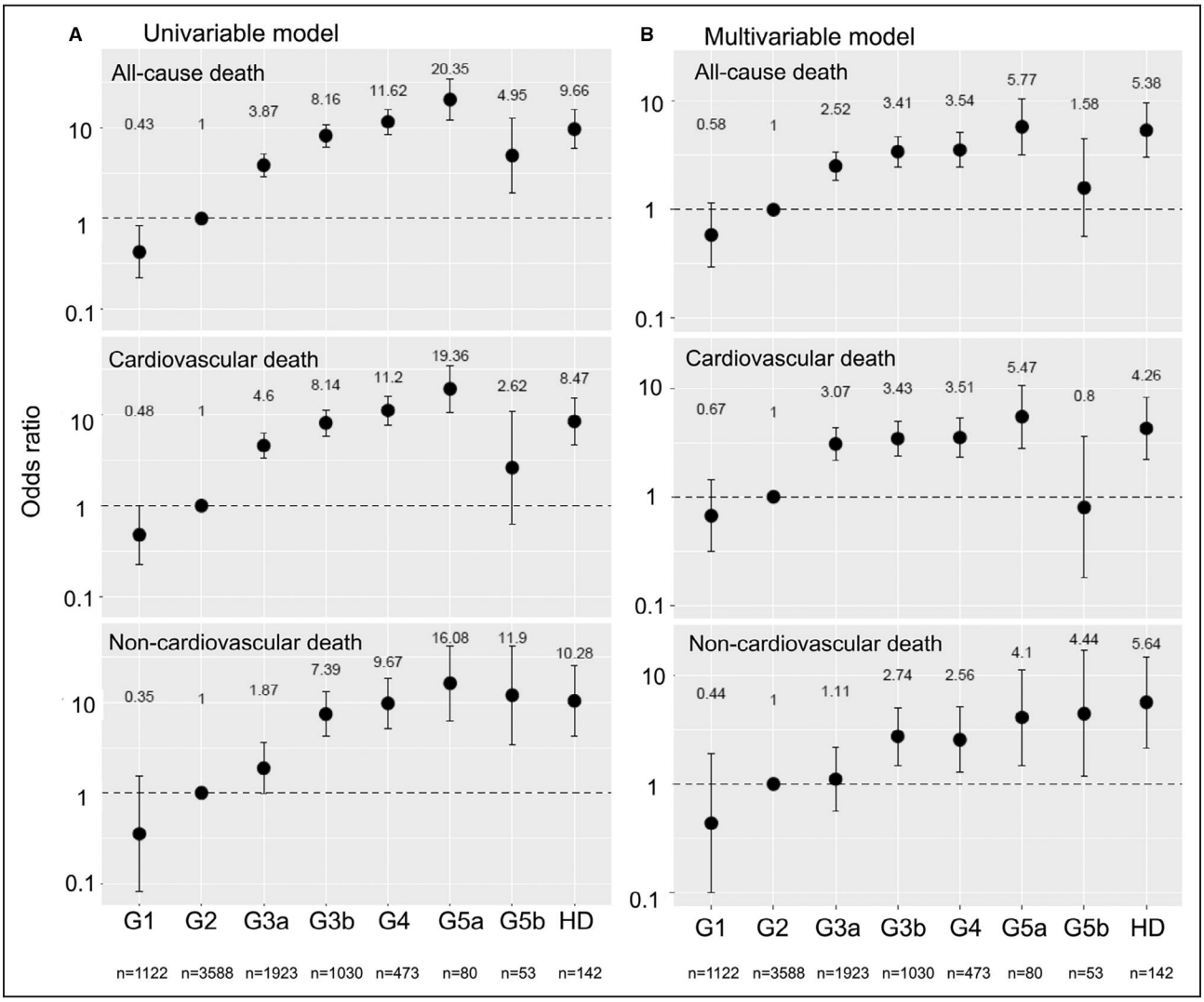

Figure 4. Logistic regression analysis for 30-day mortality.
We used the logistic regression model to evaluate the impact of CKD grade on short-term mortality (30 days). Panel (**A**) indicates the univariable model. Panel (**B**) indicates the multivariable model. In each panel, the top panel shows odds ratios for all-cause death, the middle panel for cardiovascular death, and the bottom panel for noncardiovascular death. In the multivariable model (**B**), the impact of CKD was adjusted for age, sex, diabetes, hypertension, dyslipidemia, prior myocardial infarction, primary percutaneous coronary intervention, prior cerebrovascular disease, cardiopulmonary arrest on admission, peak creatine kinase, and medication: catecholamine, β-blockers, angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, diuretics, and statins. CKD indicates chronic kidney disease; eGFR, estimated glomerular filtration rate; and HD, hemodialysis.

subsequent period from 6 months to 5 years consistently showed similar results (Figure S10).

DISCUSSION

The present study from the real-world registry of patients with AMI demonstrated the following findings: (1) patients with AMI with more advanced CKD grade or hemodialysis had worse short- and long-term outcomes; (2) CKD G5a group (15>eGFR ≥8 mL/min per 1.73 m^2) showed the highest 30-day mortality; (3) CKD G5b group (8>eGFR mL/min per 1.73 m^2) showed a huge drop of mortality rate in short-term data but not in long-term data; and (4) patients with hemodialysis showed comparable short- and long-term data in comparison with patients with advanced CKD (G4 and G5).

In our study, patients with AMI with more advanced CKD grade or hemodialysis had worse short- and long-term outcomes. Several studies have already reported the significant association between renal dysfunction and clinical outcomes in patients with AMI.[16–19] However, there is no report comparing the long-term prognosis between advanced CKD and patients with hemodialysis after MI, nor on the long-term 5-year outcome of patients with hemodialysis after

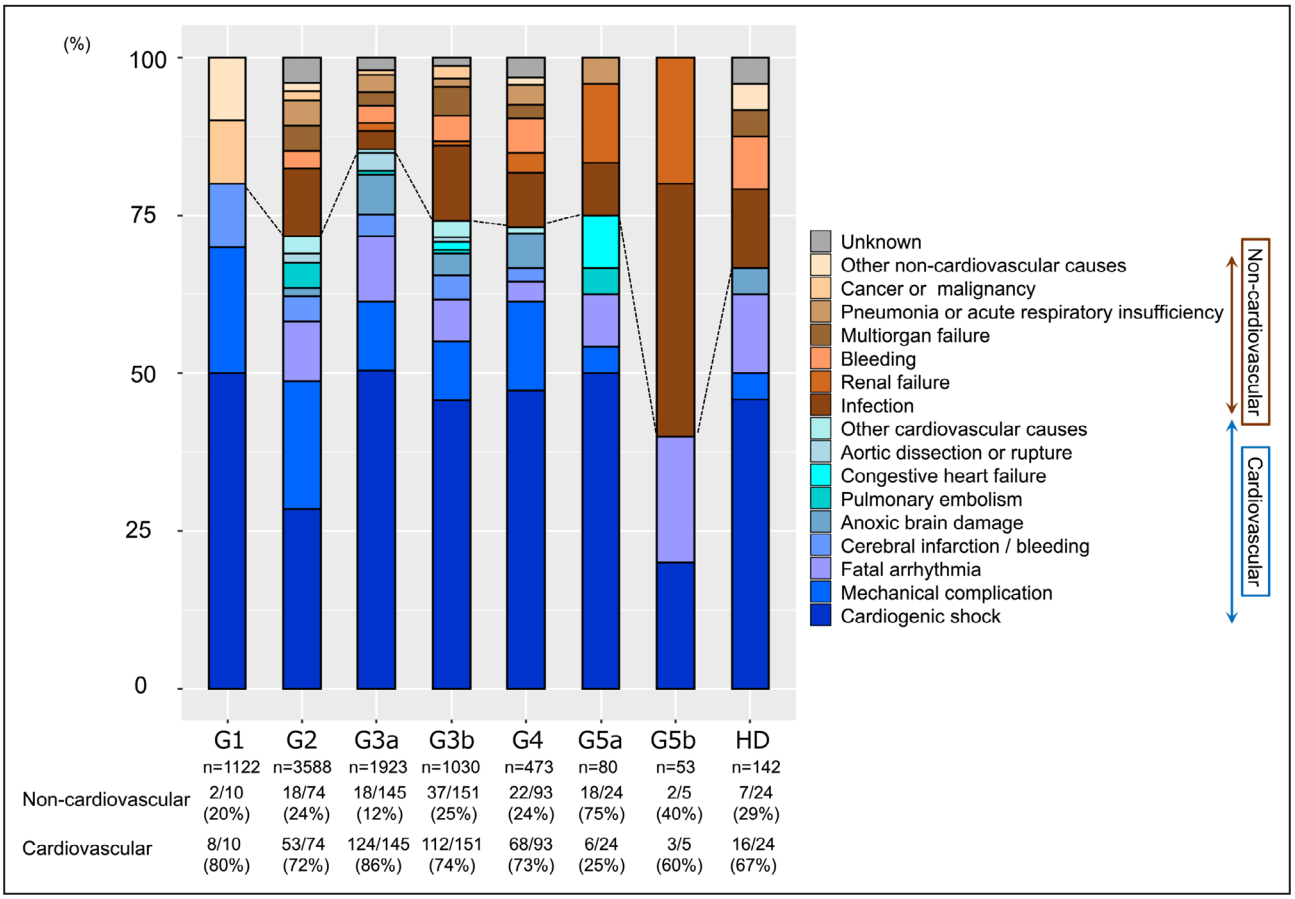

Figure 5. Cause of short-term mortality (30 days).
Bar chart indicates the distribution of cause of short-term death stratified by CKD grade. CKD indicates chronic kidney disease.

MI. Our study is the first to demonstrate that patients with hemodialysis had comparable 30-day and 5-year outcomes to patients with advanced CKD in the PCI era. The previous study from the Korea AMI Registry database (n=12 636) showed that patients with more advanced CKD grades had higher rates of in-hospital and 1-year death.[16] However, the impact of hemodialysis was not evaluated in this study. The previous study from the Acute Coronary Treatment and Intervention Outcomes Network registry, a nationwide sample of ST-segment–elevation myocardial infarction (n=19 029) and non–ST-segment–elevation myocardial infarction (n=30 462) patients also showed that patients with more advanced CKD had higher rates of in-hospital death.[17] However, this study is limited by the short-term follow-up (only in-hospital outcomes). A study from the J-MINUTE study (N=3281 patients with AMI) extended the follow-up duration and showed that CKD remains a useful predictor of 3-year mortality after AMI in the modern PCI and optimal medical therapy era.[18] Nevertheless, this study did not separate the patients undergoing hemodialysis and advanced patients with CKD not receiving hemodialysis. The latest study from the MIE ACS registry (N=4509 patients with AMI: 89 AMI/hemodialysis versus 4420 AMI/nonhemodialysis) presented that hemodialysis was independently associated with worse 2-year mortality (hazard ratio, 3.43 [95% CI, 1.88–6.26]; P <0.001).[19] However, this study simply compared patients with versus without hemodialysis, and therefore, did not provide insights into the difference between advanced CKD (G4 and G5) and patients with hemodialysis.

The strength of our study lies in its relatively large sample size, which may enable a comprehensive evaluation of the disparities between patients with advanced CKD not yet on hemodialysis and those already receiving hemodialysis. Contrary to expectations, the results indicated that the G5b group exhibited a more favorable prognosis than the G5a and hemodialysis groups during a short-term follow-up of 30 days. This may be attributable to the relatively high rates of primary PCI and the use of mechanical circulatory supports such as extracorporeal membrane oxygenation and intra-aortic balloon pumping, which likely led to increased rates of coronary revascularization in the acute phase, regardless of hemodialysis status. Notably, 90.6% of G5b patients received primary PCI, compared with 80.3% of G5a patients, a factor that likely contributed to the significantly lower

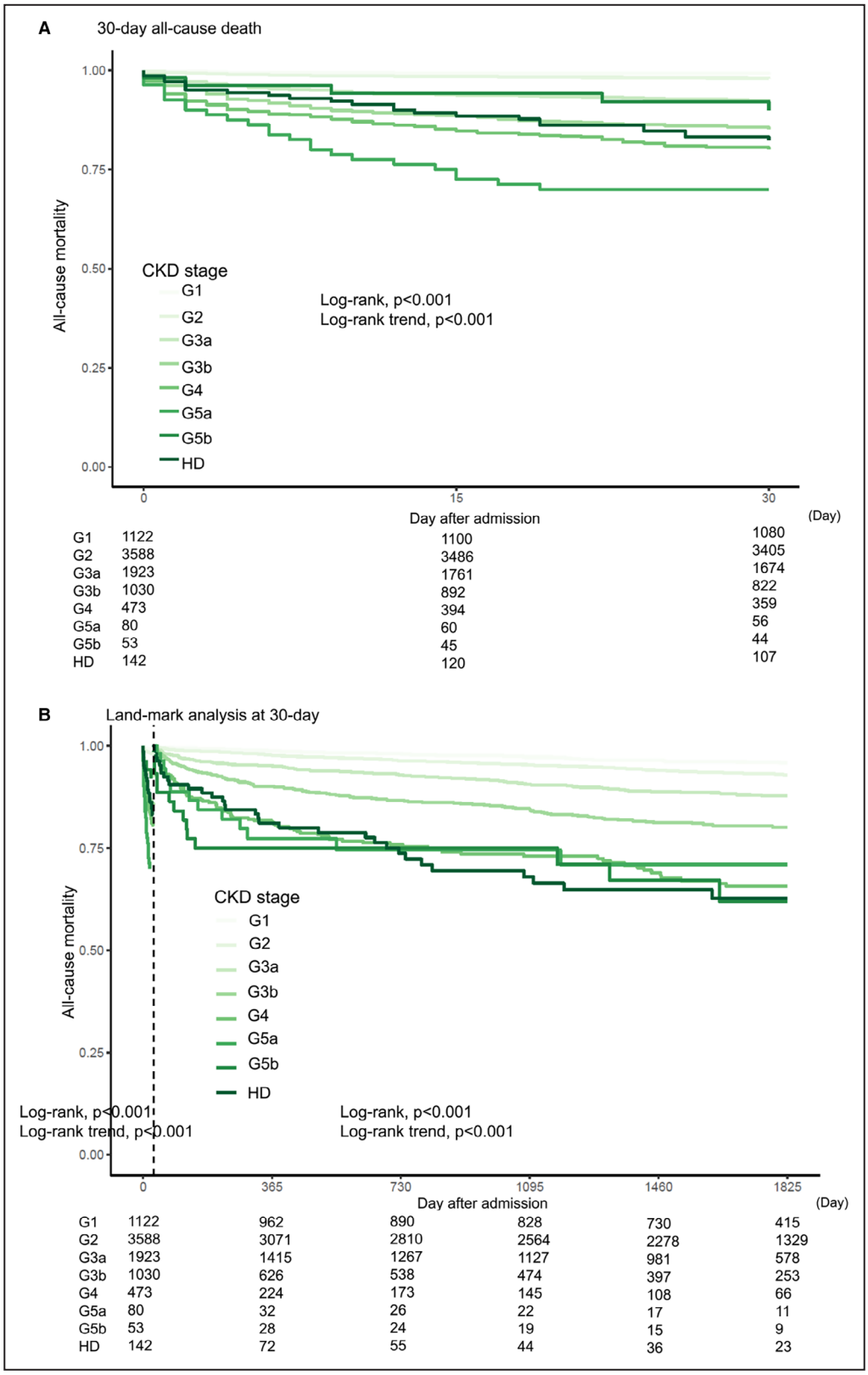

Figure 6. Kaplan–Meier curves stratified by CKD grade.
Panel (**A**) indicates Kaplan–Meier curves for a short-term follow-up period (30 days). Panel (**B**) indicates Kaplan–Meier curves for the overall follow-up period with a landmark analysis at 30 days (5 years). CKD indicates chronic kidney disease.

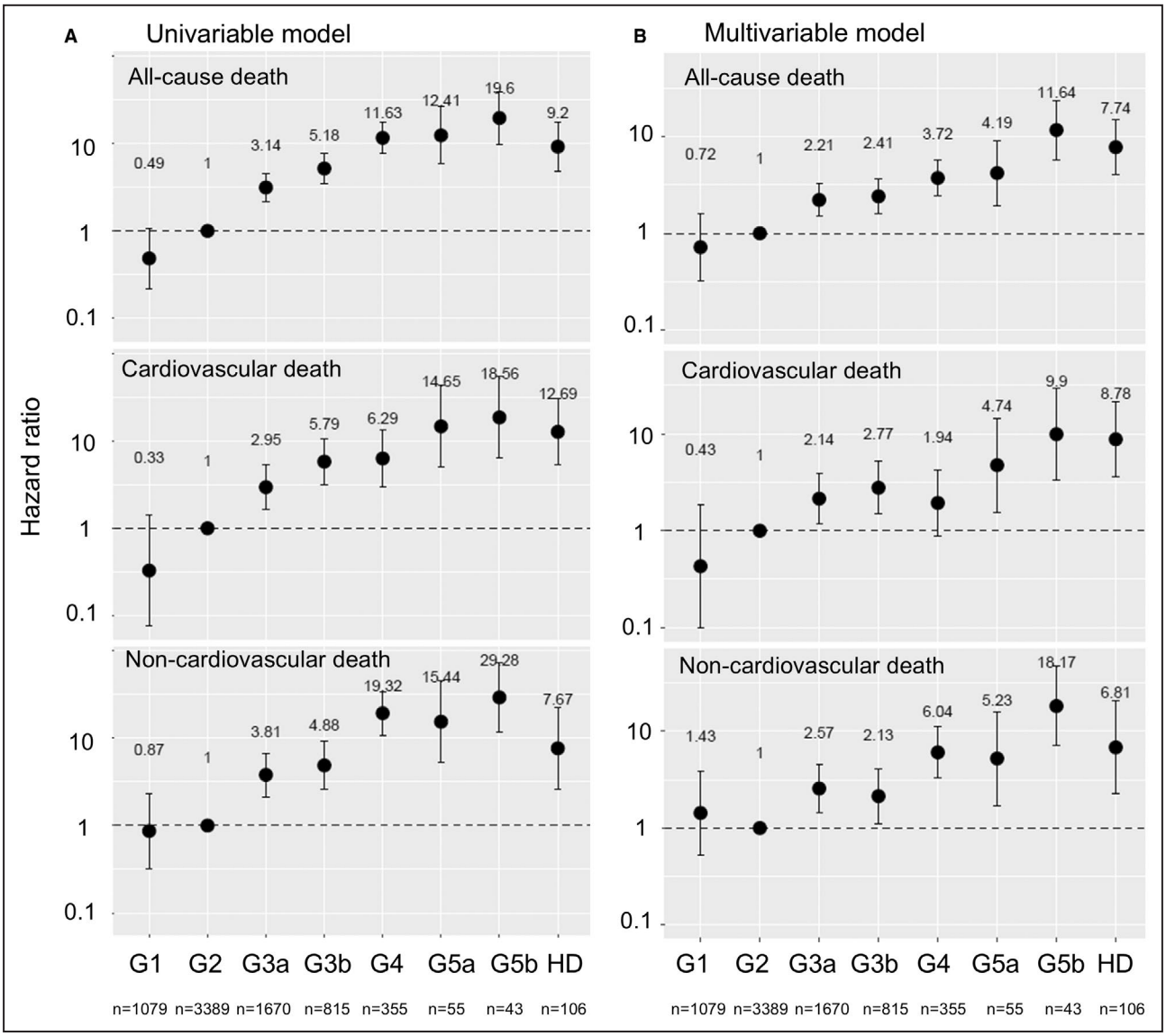

Figure 7. Impact of CKD grade on 6-month all-cause death using landmark analysis at 30 days.
We used the Cox proportional hazard model to evaluate the impact of CKD grade on all-cause mortality (6 months) using landmark analysis at 30 days after hospital admission. Panel (**A**) indicates the univariable model. Panel (**B**) indicates the multivariable model. In each panel, the top panel shows hazard ratios for all-cause death, the middle panel for cardiovascular death, and the bottom panel for noncardiovascular death. In the multivariable model (**B**), the impact of CKD was adjusted for age, sex, diabetes, hypertension, dyslipidemia, peak creatinine kinase >3000 IU/L, and medication at discharge: β-blockers, angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, diuretics, and statins. CKD indicates chronic kidney disease; and HD, hemodialysis.

cardiovascular mortality observed (Figure 3). Although our multivariable adjustment models included primary PCI as a covariate, G5b patients showed better short-term clinical outcomes. Presumably, in G5b patients, primary PCI might have been performed without restrictions on contrast agent, potentially leading to better achievement of complete revascularization. Because the precise information on the complete revascularization is unfortunately unavailable in our data set, this needs to be investigated in future research. Our data set does not include specific data on the use of hemodialysis in the acute phase. Nonetheless, it is reasonable to infer that both G5b and patients with hemodialysis underwent hemodialysis during the periprocedural period. Given that prophylactic hemodialysis does not prevent contrast-induced nephropathy in patients with chronic renal insufficiency undergoing cardiac catheterization, it can be inferred that the improvement in prognosis of G5b patients is more likely due to the proactive revascularization efforts without renal concerns.[20] Our findings lend support to the strategy of implementing aggressive, temporary artificial support of renal function to facilitate early

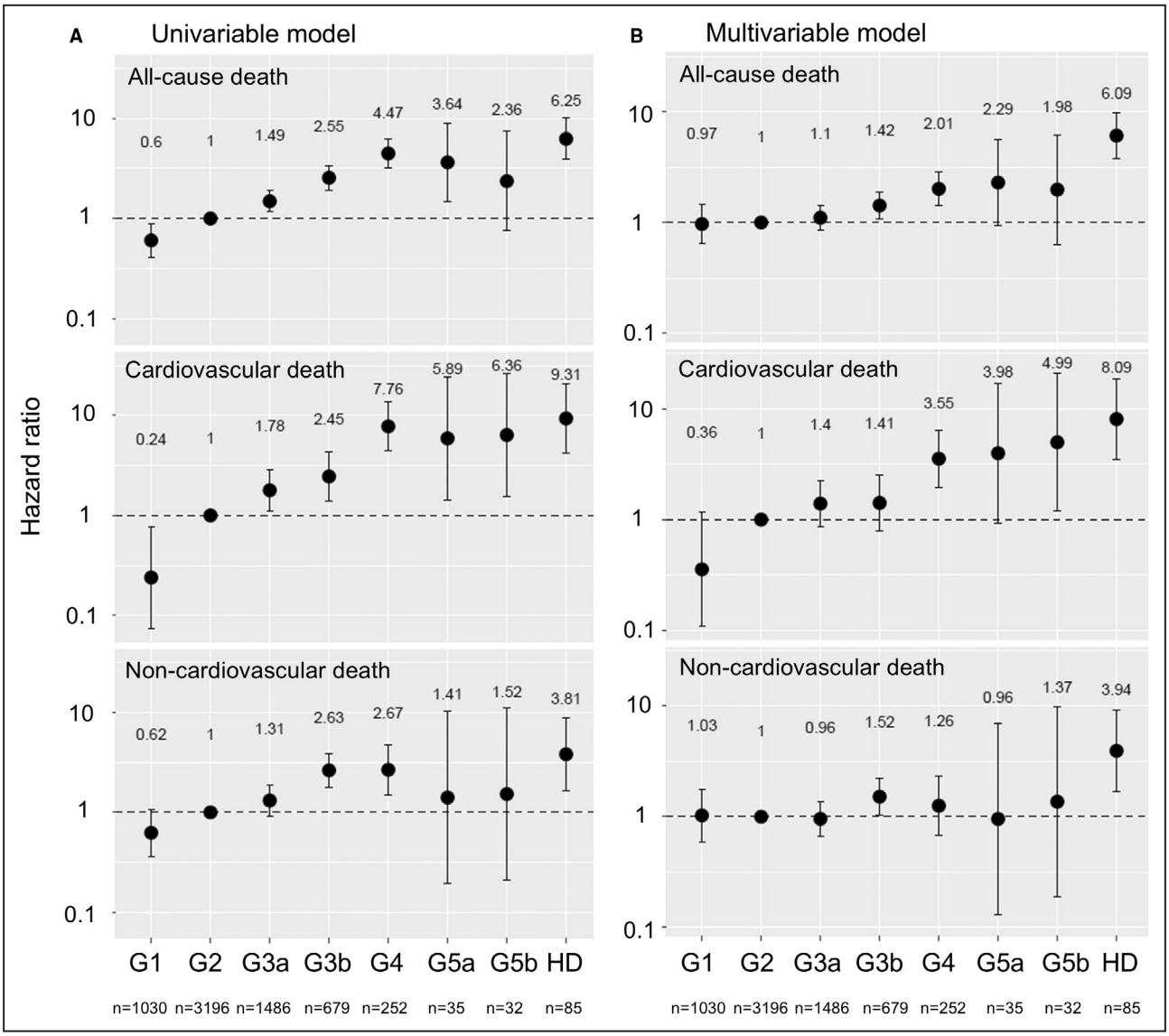

Figure 8. Impact of CKD grade on 5-year all-cause death using landmark analysis at 6 months.
We used the Cox proportional hazard model to evaluate the impact of CKD grade on all-cause mortality (5 years) using landmark analysis at 6 months after hospital admission. Panel (**A**) indicates the univariable model. Panel (**B**) indicates the multivariable model. In each panel, the top panel shows hazard ratios for all-cause death, the middle panel for cardiovascular death, and the bottom panel for noncardiovascular death. In the multivariable model (**B**), the impact of CKD was adjusted for age, sex, diabetes, hypertension, dyslipidemia, peak creatinine kinase >3000 IU/L, and medication at discharge: β-blockers, angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, diuretics, and statins. CKD indicates chronic kidney disease; and HD, hemodialysis.

revascularization, which may improve the short-term outcomes in patients with grade 4 and grade 5 CKD. This hypothesis necessitates further validation through a prospective randomized trial. The improvement in the short-term outcomes observed in the G5b group did not translate into better long-term outcomes. The increased coronary complexity and more pronounced systemic atherosclerosis in this group may explain their long-term poor prognosis comparable to patients with hemodialysis. Other approaches including precise management of basic comorbidities would be necessary to improve long-term outcomes. Future investigations need to focus on identifying predictors for short-term prognostic enhancement in these patients. Furthermore, the mechanisms underlying long-term prognostic outcomes require comprehensive elucidation.

Short-Term and Long-Term Effect of Hemodialysis

It is conceivable that patients with hemodialysis presented with a more deleterious systemic health status before the AMI event, culminating in a worse short-term prognosis. A meticulous review of computed

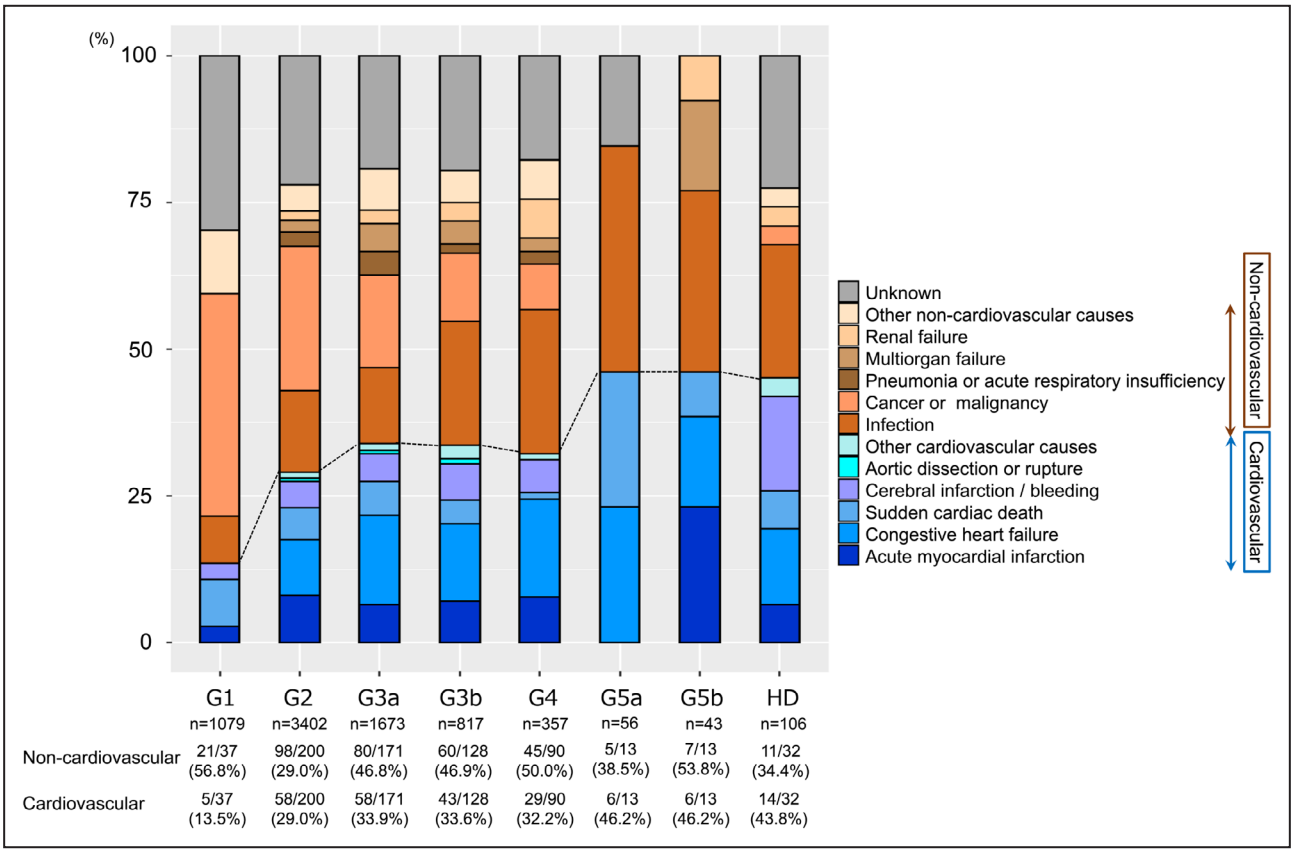

Figure 9. Cause of long-term mortality (5 years).
Bar chart indicates the distribution of cause of long-term death stratified by CKD grade. CKD indicates chronic kidney disease.

tomography–guided evaluations of coronary artery calcification in patients with hemodialysis revealed a pronounced prevalence of calcifications, which were characterized by their progressive nature.[21] Furthermore, patients with hemodialysis inherently possess a heightened susceptibility to hemorrhagic events, because uremia precipitates platelet dysfunction alongside aberrant platelet–vascular wall interactions.[22] The regulation of electrolytes, including serum potassium levels in patients with hemodialysis, presents significant challenges, with both hyperkalemia and hypokalemia recognized as pivotal risk elements for sudden cardiac death.[23,24] In our study population, hemodialysis patients exhibited the highest rate of prior PCI across all CKD grades. Within the initial week of hospitalization, a notable 10.7% of patients with hemodialysis experienced major bleeding events, alongside the highest recorded incidence of ventricular arrhythmias, including ventricular fibrillation and ventricular tachycardia. These findings may highlight the severe systemic health decline and the complexities of managing patients with hemodialysis in the acute phase following AMI.

As to the long-term prognosis, several possible explanations are postulated. First, patients with hemodialysis manifested the highest incidence rates of diabetes and hypertension upon admission across all CKD categories, indicating a significant contribution towards worse long-term cardiovascular incidents.[25] Second, in our research, patients with hemodialysis demonstrated the highest frequency of stroke as a mortality cause across all CKD grades. It is reported that patients with hemodialysis frequently experience cerebral microbleeds (asymptomatic bleeding) during hemodialysis sessions due to the routine anticoagulation.[26] This could precipitate major hemorrhagic events leading to stroke. Lastly, individuals with CKD experience an augmented rate of complications from infections, attributed to compromised immune functionality via diverse mechanisms, with the risk of infection escalating in tandem with CKD severity. Specifically, patients with hemodialysis were found to be significantly more susceptible to sepsis compared with the general populace, as evidenced in a study utilizing Japanese national Vital Statistics data because of the elevated risk of bloodstream infections linked to vascular access arteriovenous fistulas.[27] In our investigation, infections emerged as the predominant long-term mortality cause in patients with hemodialysis after myocardial infarction.

Clinical Implications

In patients with AMI with severe CKD, the proactive revascularization without concern for worsening renal function, even with the aggressive use of mechanical support, may potentially improve short-term clinical outcomes. However, the proactive revascularization during the acute phase appears to eventually have no substantial effect on long-term survival rates. This distinction suggests that while such support can be critical for managing acute hemodynamic status in AMI and patients with CKD, its benefits do not alter the overall progression of underlying chronic conditions. Therefore, although temporary mechanical support is valuable for immediate patient stabilization, it should not be viewed as a solution for long-term disease management. This insight is essential for guiding clinical decision-making, emphasizing the necessity for further investigative trials to explore additional therapeutic options that may enhance long-term survival and quality of life.

Limitations

Several limitations should be acknowledged. First, changes in renal function over time after hospital discharge were not available in our database, which did not allow us to investigate its impact on long-term prognosis. Second, we had access to data on medication prescribed only at the time of discharge but not during the long-term follow-up. Some medications may have changed during the follow-up period. Third, the performance rate of temporary hemodialysis during the periprocedural period and initiation of chronic maintenance hemodialysis during hospitalization or after discharge are unknown in our data set. Fourth, details of the PCI procedure including the achievement of complete revascularization and the amount of contrast medium were not accessible. Recent advancement of the PCI procedure and device technologies possibly provides differing results. The amount of contrast media may strongly influence renal function.[28] Fifth, while CKD grades are basically categorized using both eGFR and albuminuria levels, our database lacked albuminuria data, which may impact the comprehensiveness and accuracy of CKD classification in our study. Sixth, although there was a relatively large-scale sample size in the overall cohort, the sample size of the patients with severe CKD (especially G5a, G5b) and hemodialysis was relatively limited, raising some concern for fragility of inferences about these CKD subgroups. Seventh, Martingale and Deviance residuals in each Cox proportional hazard model exhibited positive skewedness, suggesting a potential underestimation of risk by the model. Eighth, the percutaneous left ventricular assist device may have a potential impact on renal perfusion by unloading the left ventricle and improving kidney blood flow. Our registry comprises data collected before the introduction of the percutaneous left ventricular assist device, thereby precluding an assessment of its impact. Finally, the present study is the East-Asian registry, which would limit the generalizability of the current findings to other races.

CONCLUSIONS

This study demonstrated differential impacts of CKD severity on short- and long-term clinical outcomes in the context of patients with AMI.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Appendix. The Osaka Acute Coronary Insufficiency Study (OACIS) Group
Tables S1–S4
Figures S1–S10

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