Onset time and prognostic value of acute kidney injury in patients with acute myocardial infarction

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Abstract

Background: The mechanisms and clinical impact of acute kidney injury (AKI) after acute myocardial infarction (AMI) may differ depending on whether AKI develops during the early or late phase after AMI. The present study assessed the timing of AKI onset and the prognostic impact on long-term outcomes in patients hospitalized with AMI.

Methods: The present study enrolled consecutive AMI survivors who had undergone successful percutaneous coronary interventions at admission. AKI was defined as an increase in the serum creatinine level of ≥0.3 mg/dL above the admission value within 7 days of hospitalization. AKI patients were further divided into two subgroups (early-phase AKI: within 3 days vs. late-phase AKI: 4 to 7 days after AMI onset). The primary endpoint was all-cause death.

Results: In total, 506 patients were included in this study, with 385 men and a mean age of 69.5 ± 13.5 years old. The mean follow-up duration was 1289.5 ± 902.8 days. AKI developed in 127 patients (25.1%). Long-term mortality was significantly higher in the AKI group than in the non-AKI group (log-rank p < 0.001). Early-phase AKI developed in 98 patients (19.3%), and late-phase AKI developed in 28 patients (5.5%). In the multivariable analysis, early-phase AKI was significantly associated with all-cause mortality (HR 2.83, 95% CI [1.51–5.29], p = 0.0012), while late-phase AKI was not.

Conclusion: Early-phase AKI but not late-phase AKI was associated with poor long-term mortality. Careful clinical attention and intensive care are needed when AKI is observed within 3 days of AMI onset.

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1. Introduction

Acute kidney injury (AKI), which occurs in a certain proportion of patients hospitalized with acute myocardial infarction (AMI), is independently associated with increased in-hospital and long-term mortality rates up to 10 years after AMI [1–5]. Recent reports have suggested that even small changes in renal function, as measured by serum creatinine levels, are associated with worse short-term outcomes [6–9]. Based on the existing evidence, an increase in the serum creatinine level is defined as acute kidney injury (AKI). However, the definitions used for the timing of AKI onset has not been consistent in the previous studies, with AKI defined as an increase in serum creatinine levels within 7 or 14 days after admission or at discharge.

In patients with AMI, the mechanisms underlying AKI may be different depending on whether its onset occurs in the early- or the late-phase of hospitalization and whether the inciting insult is the AMI or the primary PCI. In addition, clinical conditions and treatment strategies may influence the development of AKI after AMI. Thus, clinical outcomes might vary depending on the timing of AKI onset. Therefore, the present study classified AKI by its timing at onset and determined the impact of this timing on long-term outcomes in patients hospitalized with AMI.

2. Methods

2.1. Study population

The data of the present study is from a prospective registry which included consecutive AMI patients who underwent emergent percutaneous coronary intervention (PCI) in Showa University Hospital.
Hospital. The data was retrospectively analyzed for the purpose of the present study. Patients admitted with a diagnosis of AMI from November 2011 to January 2016 were screened and, if eligible, were prospectively enrolled to the registry. All patients provided written informed consent. The study population included patients who were within 12 h of onset of an AMI and who had successfully undergone a primary PCI. Patients who had experienced cardiopulmonary arrest on arrival or had renal dysfunction on hemodialysis were excluded. In this study, AMIs included both ST-segment elevation myocardial infarctions (STEMI) and non-ST-segment elevation myocardial infarctions (NSTEMI). STEMI and NSTEMI were diagnosed based on general universal definitions [10]. The diagnosis of an AMI was confirmed by coronary angiography in all patients. A successful PCI was defined as a stenosis of less than 25% in the target vessel after reperfusion. When other significant coronary lesions deemed ischemia-inducible were detected, an additional PCI was performed immediately after the primary PCI. The interventional strategy was left to the discretion of the operator. Demographic, clinical, and procedural data, as well as information about in-hospital outcomes, were collected from the medical record and entered into a prospective database. Clinical follow-up data were obtained at office visits or via telephone interviews. This study was approved by the Ethics Committee of Showa University.

2.2. Definitions of AKI and endpoints

We checked serum biomarkers on admission and within 1 h after the primary PCI. We also monitored serum creatinine (Cr) daily during the first week. Based on the observed changes in the serum Cr level, we divided the patients into two groups, including a non-AKI and an AKI group, in the first week. Several definitions of AKI were used in the previous studies, such as serum creatinine increase cut-off: 0.5 mg/dl, 0.3 mg/dl or 25% [11]. The definition of cut-off: 0.3 mg/dl above the value on admission within 7 days was chosen as same as that of “worsening renal function”, because “worsening renal function” was also used in many previous studies [6–9]. AKI patients were further divided into two additional groups, including an early-phase AKI group (onset within 3 days) or a late-phase AKI group (onset within 4 to 7 days after admission). This study’s primary endpoint was all-cause death during the long-term follow-up period, which was compared between patients with or without AKI and between patients with early-phase vs. late-phase AKI.

2.3. Statistical analysis

Continuous variables are presented as mean ± standard deviation (SD) and were compared using the Student’s t-test or the Wilcoxon rank-sum test depending on their distributions. Categorical variables are presented as numbers with percentages and were compared using the chi-squared test or Fisher’s probability test, as appropriate. Hazard ratios (HR) and their 95% confidence intervals (CI) were computed by logistic regression model analysis to clarify the impact of several potentially independent prognostic factors. Variables with p < 0.1 on univariate analysis were entered into the multivariable Cox model to adjust for baseline differences. Multivariable analysis was performed with forward–backward stepwise selection methods. The proportion of patients who survived was plotted using Kaplan-Meier curves, and the significance was examined using the log-rank test. Statistical analyses were performed using JMP software version pro 15.0 (SAS Institute Inc, Cary, NC, USA). Statistical significance was defined as p < 0.05.

3. Results

Of the 606 patients who underwent a primary PCI for AMI during the study period, 100 patients met the exclusion criteria for this study, including out-of-hospital cardiopulmonary arrests in 65 cases and hemodialysis in 36 cases. The remaining 506 patients were included in this study, with 385 men and a mean age of 69.5 ± 13.5 years. The mean follow-up duration was 1289.5 ± 902.8 days (range 2 to 3118 days). The AMI at presentation was the first cardiovascular event in 429 (84.8%) patients. Seventy-seven...
Baseline characteristics between AKI vs. no AKI.

| Baseline Characteristics | no AKI (n = 379) | AKI (n = 127) | p value |
|--------------------------|------------------|---------------|---------|
| Age (y.o.)               | 67.8 ± 13.6      | 74.6 ± 12.0   | <0.0001 |
| Male                     | 289 (76.3)       | 96 (75.6)     | 0.8796  |
| Body Mass Index (kg/m²)  | 23.8 ± 4.3       | 24.0 ± 4.2    | 0.2209  |
| Smoker                   | 227 (59.9)       | 78 (61.4)     | 0.7615  |
| Hypertension°             | 261 (68.9)       | 102 (80.3)    | 0.0131  |
| Diabetes                 | 150 (39.6)       | 61 (48.0)     | 0.0945  |
| Dyslipidemia             | 308 (81.3)       | 98 (77.2)     | 0.1209  |
| Prior MI*                | 42 (11.1)        | 26 (20.5)     | 0.0072  |
| Post CABG                | 4 (1.1)          | 2 (1.6)       | 0.6398  |
| Previous PCI°            | 45 (11.9)        | 30 (23.6)     | 0.0013  |
| History of HF°           | 13 (3.4)         | 12 (9.5)      | 0.0068  |
| History of stroke        | 42 (11.1)        | 22 (17.3)     | 0.0871  |
| CKD                      | 97 (25.6)        | 77 (60.6)     | <0.0001 |
| STEMI                    | 271 (71.5)       | 93 (73.2)     | 0.7982  |
| Multi-vessel disease     | 172 (46.4)       | 58 (49.6)     | 0.5440  |
| Cardiogenic shock        | 21 (5.5)         | 9 (7.1)       | 0.5232  |
| IABP°                    | 47 (12.7)        | 25 (21.2)     | 0.0229  |
| LVEF (%)                 | 50.8 ± 10.4      | 47.1 ± 10.8   | 0.0008  |
| Contrast media volume (ml)| 189.1 ± 60.9  | 163.1 ± 71.8  | 0.0002  |

BNP (pg/ml)°               | 187.5 ± 321.8    | 447.3 ± 667.0 | <0.0001 |
| Cr (mg/dl)                 | 0.79 ± 0.3       | 1.19 ± 0.78   | <0.0001 |
| CRP (mg/dl)                | 1.44 ± 3.8       | 2.06 ± 4.0    | 0.0393  |
| HDL (mg/dl)                | 44.7 ± 11.8      | 42.7 ± 12.2   | 0.1231  |
| LDL (mg/dl)                | 120.4 ± 39.5     | 109.3 ± 35.9  | 0.0063  |
| HbA1c (%)                  | 6.36 ± 1.3       | 6.36 ± 1.3    | 0.674   |
| peak CK (U/l)              | 2103.1 ± 2336.0  | 3319.9 ± 8280.8 | 0.1 |
| peak Cr (mg/dl)°           | 0.94 ± 0.3       | 1.86 ± 1.1    | <0.0001 |

AKI, acute kidney injury; MI, myocardial infarction; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; HF, heart failure; CKD, chronic kidney disease; STEMI, ST-segment elevation myocardial infarction; IABP, intra-aortic balloon pumping; LVEF, left ventricular ejection fraction; BNP, brain natriuretic peptide; Cr; creatinine; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; HbA1c, Hemoglobin A1c; CK, creatine kinase; Values are presented as the mean ± SD or n (%).
° p < 0.05.
in AMI patients [8]. In the present study, the incidence of AKI was approximately 25%. Compared with this previous study, some factors associated with AKI were higher, including the average age, the prevalence and severity of CKD, and the occurrence of a STEMI. It has been reported that patients with transient AKI have a greater risk of death after AMI during long-term follow-up, even if their renal function is completely recovered at discharge [17]. Thus, it is known that AKI is a reliable marker of long-term mortality. However, there is limited information about the clinical implications of the timing of AKI onset in patients with AMI. Therefore, this study determined the effect of the timing of AKI onset on long-term outcomes after AMI.

### 4.2. Impact of early-phase AKI on long-term mortality

The overall 3-year, all-cause mortality rate was 13.2% in this study, which is consistent with the findings of previous reports [18,19]. In order to assess the effects of AKI timing on clinical outcomes, we divided AMI patients with AKI into two groups, including an early-phase AKI group and a late-phase AKI group. The majority of cases of AKI occurred within the first 3 days, or the early-phase, after AMI. Recently, Moriyama et al. have reported that early-phase AKI is associated with higher rates of in-hospital, all-cause death, although they did not assess long-term outcomes [20]. To our best knowledge and research, the present study is the first one to report that early-phase AKI is associated with higher long-term mortality. These patients had a poorer prognosis than those with late-phase AKI. The mechanisms underlying early-phase versus late-phase AKI may be different, and the corresponding prevention and management strategies should likely be different between the two groups as well. Thus, differences in the mechanisms of AKI occurrence in the early- versus late-phase should be explored.
4.3. Mechanism of early-phase AKI

There were no significant differences in baseline variables between patients with early- vs. late-phase AKI, except for in the prevalence of diabetes mellitus and the HbA1c, CRP, and peak serum Cr levels. Thus, these factors may play a role in the differences between the mechanisms underlying early- versus late-phase AKI.

Many studies demonstrated the relation between contrast volume and AKI in AMI patients [21,22], although some studies suggested AKI after AMI was not associated with the volume of contrast. Thus, the impact of contrast volume on AKI in AMI is still controversial. The definition of CIN is generally serum creatinine increase more than 0.5 mg/dl or 25% during 48–72 h after contrast use. It is similar to the definition of early-phase AKI in the present study. Thus, the mechanism of majority of early-phase AKI might be CIN. Early-phase AKI patients more frequently had diabetes mellitus than late-phase AKI. Diabetes mellitus is a strong predictor and one of risk score for CIN that Mehran R et al. advocated. In the early-phase AKI group, diabetes was more frequent, and the HbA1c level was significantly higher. The flow reserve of the kidney may be less in diabetic patients with uncontrolled serum glucose levels. Microvascular dysfunction might reflect the poor prognosis [23]. Furthermore, it is well known that CIN is the predictor for long-term mortality after PCI. It could be the one of explanation why early-phase AKI patients had higher mortality in the present study.

Cosentino et al. demonstrated that admission high-sensitive CRP was closely associated with AKI development and severity, and with in-hospital outcomes in AMI [24]. Early-phase AKI patients had higher CRP on admission than late AKI patients in the present study. In patients with AMI, it has been documented those inflammatory factors and activation of neurohormonal systems, such as the renin-angiotensin-aldosterone system, by AMI might aggravate renal dysfunction, increase catecholamine pro-

### Table 3
Baseline characteristics between early-phase AKI vs. late-phase AKI.

|                  | Early-phase AKI | Late-phase AKI | p value |
|------------------|-----------------|----------------|---------|
| Age (y.o.)       | 74.4 ± 13.6     | 75.5 ± 14.5    | 0.2909  |
| Male             | 75 (75.8)       | 21 (75.0)      | 0.9343  |
| Body Mass Index  | 23.5 ± 3.8      | 23.3 ± 5.0     | 0.7072  |
| Smoker           | 61 (61.6)       | 17 (60.7)      | 0.9310  |
| Hypertension     | 80 (80.8)       | 22 (78.6)      | 0.7927  |
| Diabetes*        | 53 (53.5)       | 8 (28.6)       | 0.0196  |
| Dyslipidemia     | 78 (78.8)       | 20 (71.4)      | 0.4127  |
| Prior MI         | 22 (22.2)       | 4 (14.3)       | 0.3581  |
| Previous CABG    | 2 (2.0)         | 0 (0.0)        | 0.4484  |
| Previous PCI     | 25 (25.3)       | 5 (17.9)       | 0.4100  |
| History of HF    | 11 (11.1)       | 1 (3.6)        | 0.2285  |
| History of stroke| 19 (19.2)       | 3 (10.7)       | 0.2953  |
| CKD              | 60 (60.6)       | 17 (60.7)      | 0.9917  |
| STEMI            | 69 (69.7)       | 24 (85.7)      | 0.0910  |
| Multi-vessel disease | 48 (53.3) | 10 (37.0)      | 0.1374  |
| Cardiogenic shock | 8 (8.1)        | 1 (3.6)        | 0.4116  |
| IABP            | 21 (23.1)       | 4 (14.8)       | 0.3562  |
| LVEF (%)         | 467 ± 10.8      | 483 ± 11.0     | 0.4399  |
| Contrast media volume (ml) | 168.3 ± 76.3 | 146.0 ± 51.5 | 0.1702 |
| BNP (pg/ml)      | 505.3 ± 727.8   | 246.5 ± 325.1  | 0.1041  |
| Cr (mg/dl)       | 1.27 ± 0.9      | 0.93 ± 0.3     | 0.0718  |
| CRP (mg/dl)*     | 2.28 ± 4.5      | 1.30 ± 3.1     | 0.0083  |
| HDL (mg/dl)      | 424 ± 11.2      | 437 ± 15.3     | 0.6562  |
| LDL (mg/dl)      | 109.3 ± 36.6    | 109.5 ± 34.2   | 0.9120  |
| HbA1c (%)        | 6.51 ± 1.4      | 5.86 ± 0.8     | 0.0044  |
| peak CK (U/l)    | 3637.1 ± 954.4  | 2232.1 ± 1771.6| 0.9833 |
| peak Cr (mg/dl)* | 2.00 ± 1.2      | 1.33 ± 0.3     | 0.0025  |

AKI, acute kidney injury; MI, myocardial infarction; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; HF, heart failure; CKD, chronic kidney disease; STEMI, ST-segment elevation myocardial infarction; IABP, intra-aortic balloon pumping; LVEF, left ventricular ejection fraction; BNP, brain natriuretic peptide; Cr; creatinine; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; HbA1c, Hemoglobin A1c; CK, creatine kinase; Values are presented as the mean ± SD or n (%). * P < 0.05.
duction, and elevate endothelin [25–28]. These neurohormonal alterations may lead to renal arteriolar vasoconstriction and a reduction in renal perfusion pressure, which may result in poor clinical outcomes. Although assessing the impact of these factors on AKI onset is difficult with AMI, they may represent key factors. The factor such as hemodynamic abnormalities might be also related with the cause of early-phase AKI. The mechanism of early-phase AKI may be multifactorial. The incidence of late-phase AKI was lower than that of early-phase AKI. The mechanism of late-phase AKI might be drug-induced or transient dehydration, not resulting in serious condition later. Further studies are needed to determine the mechanism of early-phase and late-phase AKI in AMI.

5. Study limitations

This study had several limitations. First, though we tried to adjust for confounding factors via a multivariate logistic regression analysis, we cannot exclude the possibility of residual contributing factors resulting from the presence of an unmeasured confounder or measurement errors in the included factors. Second, because of methodologic limitations in this retrospective analysis, we cannot identify etiologic factors for AKI in patients with MI. Although the predictors for early- and late-phase AKI were individually analyzed, the analyses of predictors for each group may be underpower. Finally, the initial Cr level might have been affected by hemodynamic or metabolic statuses at presentation. Thus, it is difficult to ascertain an approximation of baseline renal function in all patients.

6. Conclusion

Early-phase but not late-phase AKI was associated with poor long-term mortality. Monitoring Cr levels during the first few days after AMI is a simple method to identify this high-risk patient group. Careful clinical monitoring and intensive care are needed in patients with AMI and AKI, particularly for those with early-phase onset.

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Author contributions

None.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcha.2021.100826.

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