Research Article

In-Hospital and Six-Month Outcomes in ST-Elevation Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention: Impact of Acute Kidney Injury

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ABSTRACT: Background: Cardiovascular disease remains a leading cause of global morbidity and mortality and often initially presents as Acute Coronary Syndrome (ACS), with renal dysfunction such as Acute Kidney Injury (AKI) frequently occurring and contributing to poorer outcomes.
Aim: This study aimed to compare in-hospital and six-month clinical outcomes among ST-Elevation Myocardial Infarction (STEMI) patients undergoing Percutaneous Coronary Intervention (PCI) with and without AKI.
Methods: A retrospective cohort study was conducted using the 2022 ACS Registry data. A total of 140 STEMI patients treated with PCI were included. Patients were divided into two groups based on the presence or absence of AKI. Clinical outcomes assessed included length of stay (LOS), incidence of heart failure, and Major Adverse Cardiovascular and Cerebrovascular Events (MACCE) during hospitalization and six months after discharge.
Results: A total of 140 patients were included (AKI, $n = 35$; non-AKI, $n = 105$). The AKI group was older and had lower systolic and diastolic blood pressures, with higher TIMI risk scores (all $p < 0.05$), while the sex distribution was similar. Random blood glucose and left ventricular ejection fraction were numerically worse but not statistically significant. Urea, creatinine, and potassium levels were significantly elevated in the AKI group (all $p < 0.001$). Length of stay was longer in the AKI group (8.77 ± 3.86 vs. 6.25 ± 2.53 days, $p < 0.001$), with more patients hospitalized for more than 8 days (53.3% vs. 24.2%, $p = 0.003$). In-hospital MACCE was not significantly different (34.3% vs. 45.7%, $p = 0.237$). However, at 6 months, the incidence of MACCE was significantly higher in the AKI group (25.8% vs. 4.2%, $p < 0.001$). Overall MACCE rates were comparable. At 6 months, heart failure occurred more frequently in AKI patients (22.6% vs. 3.2%, $p = 0.001$), while reinfarction and mortality rates were similar between the groups.
Conclusions: AKI in STEMI patients undergoing PCI is associated with worse clinical outcomes. Early detection and intervention are essential to improve prognosis.

Keywords: STEMI, PCI, acute kidney injury, clinical outcomes, MACCE

I. INTRODUCTION

Cardiovascular disease is the leading cause of morbidity and mortality worldwide, accounting for an estimated 19 million deaths (37%) in 2020. Acute Coronary Syndrome (ACS) is often the first clinical manifestation, with around 5.8 million new cases of ischemic heart disease reported in 2019 across 57 ESC countries. In the United States, one person experiences Acute Myocardial Infarction (AMI) every 40 seconds. Ischemic heart disease contributes to 38% of cardiovascular deaths in women and 44% in men. According to the One ACS multicenter registry, 48.8% of ACS cases are ST-elevation myocardial infarction (STEMI), with 65.2% receiving reperfusion therapy. STEMI has a higher mortality rate than non-STEMI (11.7% vs. 6.2%) [1], [2].

STEMI commonly occurs due to the rupture of atherosclerotic plaques, which initiates platelet aggregation and activates the coagulation cascade. This process leads to thrombus formation that can partially or completely block the coronary arteries, restrict blood flow, and result in myocardial ischemia. If the oxygen supply remains disrupted for approximately 20 minutes, it can cause irreversible damage to the heart muscle, known as myocardial necrosis. To restore blood flow, revascularization using primary percutaneous coronary intervention (PCI) or fibrinolytic therapy is recommended. Among these, PCI is particularly effective in limiting heart muscle injury, reducing the risk of severe complications such as arrhythmias and heart failure, and improving overall clinical outcomes [2, 3].

Acute Kidney Injury (AKI) is a frequent complication in hospitalized patients, often resulting from decreased cardiac output, altered renal hemodynamics, and the use of contrast agents during PCI. AKI can trigger systemic inflammation, worsen clinical outcomes, and increase treatment complexity and costs. The reported incidence in ACS patients ranges from 5–55%, influenced by diagnostic criteria and patient populations. Although previous studies have associated AKI with poor long-term outcomes in AMI patients, limited data exist regarding short- and medium-term outcomes in STEMI patients undergoing PCI [4, 5, 6, 7]. Therefore, this study aims to compare short- and medium-term clinical outcomes in STEMI patients undergoing PCI reperfusion, with or without AKI, who were treated at Dr. Wahidin Sudirohusodo Hospital.

II. METHODS

A. Study design and setting

This study was an observational study with a retrospective cohort design. The study analyzed data collected from the ACS Registry between January and December 2022. The population included all patients recorded in the ACS Registry at the Integrated Heart Center of Dr. Wahidin Sudirohusodo Hospital, Makassar. We consecutively enrolled all patients diagnosed with STEMI who underwent PCI.

The inclusion criteria were male and female patients aged 18–60 years, diagnosed with STEMI, who underwent PCI reperfusion and had serum creatinine measurements. Patients were excluded if they had chronic kidney disease, were treated with thrombolytic agents, or suffered from severe infections such as sepsis or systemic inflammatory response syndrome (SIRS). Ethical approval was granted under protocol UH24060390, number 506/UN4.6.4.5 .31/PP36/2024. Written informed consent was obtained from all participants prior to inclusion in the study.

B. Data collection

Data for this study were collected retrospectively from the ACS Registry at Dr. Wahidin Sudirohusodo General Hospital, including patients diagnosed with STEMI who underwent PCI between January and December 2022. Variables collected included demographic data (age, gender), cardiovascular risk factors (hypertension, diabetes mellitus, smoking status), clinical features (Killip class, onset of symptoms), laboratory results (serum creatinine, troponin levels), echocardiographic data (left ventricular ejection fraction), and in-hospital treatments. Serum creatinine was measured at admission to determine the presence of AKI using the KDIGO criteria. Clinical outcomes assessed included short-term outcomes such as heart failure, recurrent myocardial infarction, in-hospital mortality, and length of stay, as well as six-month follow-up outcomes. Major Adverse Cerebrovascular and Cardiovascular Events (MACCE), including cardiovascular death, stroke, reinfarction, and rehospitalization, were evaluated to compare mid-term outcomes between patients with and without AKI. Data were extracted and recorded using standardized forms and cross-checked for consistency before analysis.

C. Statistical analysis

Data were processed and analyzed using SPSS version 24. Categorical variables are presented as frequencies and percentages, while numerical variables are presented as mean ± standard deviation (SD) for normally distributed data, or median with interquartile range (Q1–Q3) for non-normally distributed data. Normality was assessed using the Kolmogorov–Smirnov test for sample sizes ≥ 50 and the Shapiro–Wilk test for smaller groups.

Bivariate analysis was conducted to compare the groups with and without acute kidney injury. For numerical variables, independent t-tests were used when parametric assumptions were met; otherwise, the Mann–Whitney test was applied. Categorical variables were compared using the Chi-square test or Fisher's exact test, depending on the data distribution and sample size.

III. RESULTS

A total of 140 patients met the inclusion and exclusion criteria and were included in the study. The sample was divided into two groups: the AKI group (n = 35) and the non-AKI group (n = 105). The characteristics of the study participants are summarized in Table 1.

Table 1. Baseline and clinical characteristics of the study participants

Variable	AKI (n = 105)	Non-AKI (n =105)	p-value
Age	60.5 ± 10.9	56.2 ± 10.3	0.034*
Body Mass Index (BMI)	24.6 ± 2.9	24.8 ± 3.0	0.794
Gender			
Male	29 (82.9%)	86 (81.9%)	0.899
Female	6 (17.1%)	19 (18.1%)	
Smoking Status			
Yes	24 (68.6%)	70 (66.7%)	0.835
No	11 (31.4%)	35 (33.3%)	
Hypertension			
Yes	19 (54.3%)	56 (53.3%)	0.922
No	16 (45.7%)	49 (46.7%)	
Diabetes Mellitus			
Yes	13 (37.1%)	26 (24.8%)	0.157
No	22 (62.9%)	79 (75.2%)	
Hypercholesterolemia			
Yes	2 (5.7%)	8 (7.6%)	0.705
No	33 (94.3%)	97 (92.4%)	
History of Myocardial Infarction			
Yes	3 (8.6%)	4 (3.8%)	0.263
No	32 (91.4%)	101 (96.2%)	
Systolic Blood Pressure	116.17 ± 30.83	128.87 ± 22.90	0.011*
Diastolic Blood Pressure	73.03 ± 17.16	81.34 ± 14.11	0.005*
Heart Rate	79.80 ± 26.24	79.99 ± 16.91	0.96
TIMI Score	5.60 ± 2.89	3.77 ± 1.90	<0.001*
Blood Glucose	205.51 ± 134.81	171.11 ± 89.50	0.03*
Hyperglycemia			
Yes	10 (28.6%)	26 (24.8%)	0.655
No	25 (71.4%)	79 (75.2%)	
Ejection Fraction	40.62 ± 7.59	42.95 ± 7.51	0.13
Hemoglobin	13,42 ± 3,96	13,96 ± 1,89	0,283
Hematocrit	38,30 ± 8,46	41,28 ± 5,62	0,019*
Leukocyte Count	15,24 ± 5,91	13,28 ± 3,93	0,028*
Cholesterol	188,58 ± 67,89	201,41 ± 57,48	0,300
Triglycerides	105 (43-334)	106 (48-400)	0,704
Low-Density Lipoprotein (LDL)	118,59 ± 38,95	120,87 ± 37,76	0,769
High-Density Lipoprotein (HDL)	45,22 ± 15,71	45,86 ± 10,47	0,792
Urea	68,89 ± 35,28	29,39 ± 14,05	<0,001*
Creatinine	1,98 ± 0,93	0,86 ± 0,24	<0,001*
Sodium	134,11 ± 4,79	133,65 ± 4,37	0,600
Potassium	4,67 ± 1,14	3,97 ± 0,42	<0,001*
Troponin I	26348 (1.5-40.000)	22520 (1.5-40.000)	0,432

Description: Values are n (%) or mean ± SD, unless stated otherwise. Continuous variables were compared using independent-samples t-test, categorical variables with Pearson Chi-square test. Non-normally distributed data were analyzed with Mann-Whitney U test and presented as median.*p < 0.05

The AKI group had a significantly higher mean age (60.5 ± 10.9 vs. 56.2 ± 10.3 years, $p = 0.034$), and lower systolic and diastolic blood pressures (116.17 ± 30.83 vs. 128.87 ± 22.90 mmHg, $p = 0.011$; 73.03 ± 17.16 vs. 81.34 ± 14.11 mmHg, $p = 0.005$). TIMI risk scores were also higher (5.60 ± 2.89 vs. 3.77 ± 1.90, $p < 0.001$). Gender distribution was similar (male: 82.9% vs. 81.9%, $p = 0.899$). Random blood glucose was higher in the AKI group, although the difference was not significant (205.51 ± 134.81 vs. 171.11 ± 89.50 mg/dL, $p = 0.088$), and the prevalence of hyperglycemia was comparable (28.6% vs. 24.8%, $p = 0.655$). Ejection fraction tended to be lower (40.62 ±

7.59% vs. 42.95 ± 7.51%, p = 0.130). Hematocrit was significantly lower in the AKI group (38.30 ± 8.46% vs. 41.28 ± 5.62%, p = 0.019), while hemoglobin showed no difference. Leukocyte count was higher (15.24 ± 5.91 vs. 13.28 ± 3.93 $\times 10^3$/mm^3, p = 0.028).

AKI patients had markedly higher urea (68.89 vs. 29.39 mmol/L), creatinine (1.98 vs. 0.86 μmol/L), and potassium levels (4.67 vs. 3.97 mmol/L; all $p < 0.001$). Sodium and troponin I levels were similar between the groups.

Based on Table 2, the mean length of hospital stay was significantly longer in the AKI group (8.77 ± 3.86 days) compared to the non-AKI group (6.25 ± 2.53 days) ($p < 0.001$). Additionally, the proportion of patients with a length of stay exceeding 8 days was higher in the AKI group (53.3%) than in the non-AKI group (24.2%), which was statistically significant (p = 0.003). During hospitalization, the incidence of Major Adverse Cardio-Cerebrovascular Events (MACCE) was lower in the AKI group (34.3%) than in the non-AKI group (45.7%), although this difference was not statistically significant (p = 0.237). However, at 6 months post-discharge, the AKI group demonstrated a significantly higher incidence of MACCE (25.8%) compared to the non-AKI group (4.2%) ($p < 0.001$).

Table 2. Comparative Analysis of Clinical Outcomes Between Patients With and Without Acute Kidney Injury (AKI)

Variables	AKI (n = 105)	Non-AKI (n =105))	p-value
Length of Stay	8.77 ± 3.86	6.25 ± 2.53	<0.001*
Length of Stay >8 days	16 (53.3%)	22 (24.2%)	0.003*
MACCE During Hopitalization			0.237
Yes	12 (34.3%)	48 (45.7%)	
No	23 (65.7%)	57 (54.3%)	
MACCE at 6 Months Post-Discharge			<0.001*
Yes	8 (25.8%)	4 (4.2%)	
No	23 (74.2%)	91 (95.8%)	
Total MACCE			0.329
Yes	20 (57.1%)	50 (47.6%)	
No	15 (42.9%)	55 (52.4%)	
Description: Values are n (%) or mean ± SD, unless stated otherwise. Continuous variables were compared using independent-samples t-test, categorical variables with Pearson Chi-square test. Non-normally distributed data were analyzed with Mann-Whitney U test and presented as median. *p < 0.05			

When considering the total incidence of MACCE (during hospitalization and post-discharge combined), no statistically significant difference was observed between the AKI and non-AKI groups, with event rates of 57.1% and 47.6%, respectively (p = 0.329).

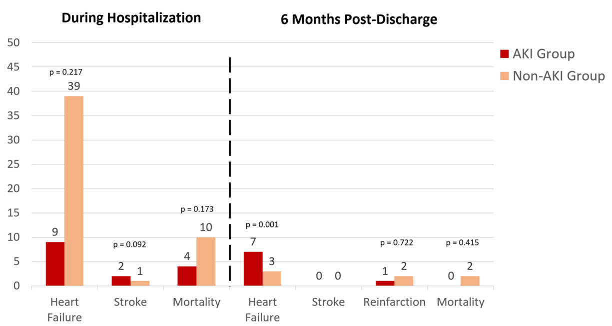

Figure 1. Comparison of clinical outcomes of major adverse cardio-cerebrovascular events (MACCE) during hospitalization and 6 months post-discharge

The distribution of MACCE components during hospitalization and six months post-discharge between patients with and without acute kidney injury (AKI) is shown in Figure 1. During hospitalization, heart failure was observed in 25.7% of patients with AKI and 37.1% of those without AKI, although this difference was not statistically significant (p = 0.217). Stroke occurred more frequently in the AKI group (5.7%) compared to the non-AKI group (0.5%), yet this difference did not reach statistical significance (p = 0.092). In-hospital mortality was slightly higher in the AKI group (11.4%) than in the non-AKI group (9.5%), but the difference was not statistically significant (p = 0.173).

At six months post-discharge, heart failure remained significantly more common in the AKI group (22.6%) compared to the non-AKI group (3.2%) (p = 0.001). No cases of stroke were reported in either group during this period. Reinfarction rates were comparable between the two groups (3.2% in the AKI group vs. 2.1% in the non-AKI group; p = 0.722). There were no reported deaths in the AKI group, while mortality in the non-AKI group was 2.1%; however, this difference was not statistically significant (p = 0.415).

IV. DISCUSSION

This study aimed to compare the short-term outcomes of NSTEMI patients undergoing reperfusion, with or without impaired renal function, who were treated at the Integrated Heart Center of Dr. Wahidin Sudirohusodo Hospital. The sample was divided into two groups: the group with AKI (n = 41) and the group without AKI (non-AKI) (n = 117). Data on the basic characteristics and clinical profiles of the research subjects were collected upon hospital admission. There were no significant differences in age, sex, smoking status, prevalence of hypertension, diabetes mellitus, hypercholesterolemia, and history of infarction between the AKI and non-AKI groups.

Basic subject characteristics were collected upon hospital admission. A significant difference was found in age, with a higher mean age in the AKI group compared to the non-AKI group. This result is consistent with the study by Kaur et al. [8], which showed that older patients are more susceptible to AKI. A study conducted in a cardiac care unit in Tripoli, Libya, reported similar findings, with a mean age of 61.6 ± 1.5 years in AKI patients compared to 54.7 ± 13 years in non-AKI patients [9]. Mezhonov et al. [10] also reported that the mean age of AKI patients (66.4 ± 13.08 years) was higher than that of those without AKI (61.2 ± 9.86 years) (p = 0.001). According to Coca et al. [11], age is an important factor that increases vulnerability to AKI due to age-related decline in renal function, a high prevalence of comorbidities, and increased exposure to nephrotoxic agents. Older adults have a lower baseline GFR and reduced renal reserve, making them more vulnerable to acute stress and AKI [8, 9, 10, 11].

No significant differences were found in other variables, such as BMI, gender, smoking status, history of hypertension, diabetes mellitus, hypercholesterolemia, and myocardial infarction. These findings align with studies by Jing et al. [12] and Mezhonov et al. [10], which also found no significant differences in hypertension, diabetes mellitus, and infarction history between AKI and non-AKI patients with STEMI [10].

In our study, clinical data were collected at admission. Systolic blood pressure was significantly lower in the AKI group compared to the non-AKI group. Diastolic blood pressure was also lower in the AKI group. This finding is consistent with a study by Sun et al. [13], which found that patients with AKI had lower systolic and diastolic blood pressures at admission than those without AKI. In their study, the median systolic blood pressure decreased from 135.0 mmHg in the non-AKI group to 122.5 mmHg in stage 2 and 3 AKI groups ($p = 0.001$), while the median diastolic pressure decreased from 80 mmHg to 72 mmHg ($p < 0.001$). The drop in blood pressure may reflect hemodynamic disturbances contributing to renal dysfunction [13].

Blood glucose levels were significantly higher in the AKI group than in the non-AKI group. This is in line with Jing et al. [12], who reported elevated fasting glucose levels in the AKI group (8.8 ± 4.8 vs. 7.7 ± 3.5 mmol/L; $p = 0.005$). From a pathophysiological perspective, acute hyperglycemia in STEMI patients, even in the absence of pre-existing diabetes, has been recognized as an independent risk factor for the development of congestive heart failure and increased in-hospital mortality. Additionally, it contributes to a greater likelihood of contrast-induced nephropathy through several mechanisms, including heightened oxidative stress, medullary hypoxia due to impaired vasodilation, and osmotic diuresis, which can lead to volume depletion and prerenal azotemia, thereby worsening kidney injury [12], [14].

TIMI scores were significantly higher in the AKI group compared to the non-AKI group. This is consistent with findings by Cinar et al. [15], who reported higher TIMI Risk Index scores in patients with contrast-induced AKI (CI-AKI) (24.2 [19.3–32.2]) compared to non-CI-AKI patients (17.5 [12.9–24.3]) ($p < 0.001$). Multivariate analysis found that the TRI was an independent predictor of CI-AKI. These results support the predictive value of TIMI-based risk scores in identifying AKI development in acute coronary syndrome patients [15].

Hematocrit levels were significantly lower in AKI patients compared to those without AKI. This is consistent with Tan et al. [12], who reported a decreasing trend in hematocrit among AKI patients, although it was not statistically significant. Kosaki et al. [16] also reported lower hemoglobin levels in AKI patients (10.9 ± 2.6 g/dL vs. 12.2 ± 2.1 g/dL; $p < 0.001$) [12], [16].

White blood cell count was significantly higher in the AKI group, similar to Anzai et al. (2010), who found significantly higher WBC counts at admission in AKI patients (12,300 ± 3,400 vs. 10,300 ± 3,000 /mm^3; $p = 0.004$) and also reported higher peak CRP levels (12.6 ± 7.0 vs. 8.4 ± 5.8 mg/dL; $p = 0.0009$). Kosaki et al. [16] likewise found significantly higher peak creatinine and CRP levels in AKI patients, indicating more severe systemic inflammation. Jing et al. [12] also reported elevated lymphocyte counts in AKI patients, further supporting the presence of systemic inflammation.

Our study showed elevated urea and creatinine levels in AKI patients. These results are in line with Jing et al. [12], who reported a higher proportion of reduced eGFR (< 60 mL/min/1.73 m^2) in AKI patients. Kosaki et al. [16] also found significantly higher creatinine levels in AKI patients (2.0 ± 1.5 vs. 0.9 ± 0.5 mg/dL; $p < 0.001$). Additionally, potassium levels were significantly higher in the AKI group, consistent with Zhang et al. [17], who found serum potassium levels of 4.1 (3.8–4.4) in AKI patients versus 3.9 (3.7–4.2) in non-AKI patients ($p = 0.011$), indicating impaired potassium excretion and an increased risk of hyperkalemia [12], [16], [17].

Hospital stay was significantly longer in AKI patients (8.77 ± 3.86 days) than in non-AKI patients. This is consistent with Kim et al. [18], who reported prolonged hospitalization in both transient and persistent AKI. In their study, persistent AKI patients had a mean stay of 25 ± 39 days, compared to 9 ± 8 days in non-AKI patients ($p < 0.001$), likely due to higher complication rates in severe AKI [18].

Although MACCE events during hospitalization were slightly lower in AKI patients, the difference was not significant. However, at the 6-month follow-up, the incidence of MACE was significantly higher in the AKI group. Zhang et al. [17] also reported a significantly higher 18-month MACE incidence in AKI patients (41.7% vs. 28.3%; $p = 0.022$; HR 1.55; 95% CI: 1.06–2.26) [17].

Anzai et al. [19] also found that AKI was associated with increased MACE during the follow-up period, particularly due to higher in-hospital cardiac mortality and readmission for heart failure within the first 12 months post-MI. Multivariate analysis identified AKI, baseline eGFR < 60 mL/min/1.73 m^2, and diabetes as independent predictors of MACE, emphasizing AKI as a potential risk factor for adverse cardiovascular outcomes [19].

Our study found no significant differences in mortality during hospitalization or the 6-month follow-up. We also found no significant differences in reinfarction or reintervention between AKI and non-AKI patients, consistent with Zhang et al. [17], Kim et al. [18], and Anzai et al. [19], who all reported non-significant differences in recurrent myocardial infarction and re-PCI rates.

However, the incidence of heart failure within 6 months after discharge was significantly higher in AKI patients. This aligns with Anzai et al. [19], who found higher readmission rates for heart failure in AKI patients (13% vs. 1%; $p = 0.006$), and with Zhang et al. [17], who also observed higher rehospitalization rates for heart failure in AKI patients.

V. CONCLUSIONS

This study reveals that the occurrence of AKI in STEMI patients undergoing PCI is linked to unfavorable clinical outcomes, particularly an increased risk of 6-month heart failure and MACE. AKI serves as an important indicator of poor medium-term prognosis, highlighting the urgency of prompt detection and appropriate management in affected individuals. To broaden the relevance of these findings, future studies should be conducted across multiple healthcare centers with varied patient groups and include longer follow-up durations.

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CONFLICT OF INTERESTS

The authors declare no competing interests.

ETHICS APPROVAL AND INFORMED CONSENT

Written informed consent was obtained from all individual participants included in this study. All procedures performed in this study involving human participants were in accordance with the regulations and ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethical approval was granted under protocol UH24060390, number 506/UN4 .6.4.5.31/PP36/2024. Written informed consent was obtained from all participants prior to inclusion in the study.

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