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Original Article

Risk Factors for Acute Kidney Injury in ST-Elevation Myocardial Infarction Patients Undergoing Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: Acute kidney injury (AKI) is a frequent complication of ST-Elevation Myocardial Infarction (STEMI). AKI occurs in about 10% to 20% of patients with STEMI, which significantly impacts both short-term and long-term clinical outcomes.
Objectives: We purposed to identify the risk factors for AKI among STEMI patients undergoing Primary PCI.
Methods: This retrospective cohort study analyzed 568 STEMI patients who underwent Primary PCI from 2017 to July 2022 at Saiful Anwar General Hospital Malang. We conducted both univariate and multivariate studies to determine AKI risk factors.
Result: The risk factors for AKI among STEMI patients undergoing primary PCI were identified. The OR values for each were as follows: Shock condition (OR = 1.41; 95% CI = 1.18 – 1.92); Killip ≥3 (OR = 3.54; 95% CI = 2.14 – 4.26); and total contrast volume> 145 ml (OR = 1.61; 95% CI = 1.13 – 1.92). Based on the ROC curve analysis, total contrast volume >145 ml with an area under the curve (AUC) of 0.75 (95% CI = 0.65-0.85) with a specificity of 0.66 (95% CI = 0.61 – 0.71) and a sensitivity of 0.71 (95% CI = 0.65-0.76).
Conclusion: Our study revealed that the risk factors for AKI among STEMI patients undergoing primary PCI were shock condition, Killip class ≥3, and total contrast volume >145 ml.

1. Introduction

Acute kidney injury (AKI) is a frequent complication of Acute Coronary Syndrome (ACS), particularly in ST-Elevation Myocardial Infarction (STEMI). It has the potential role to be a predictor of long-term survival. AKI occurs in about 10% to 20% of patients with STEMI, which significantly impacts both short-term and long-term clinical outcomes.[1] A retrospective study conducted on 386 patients with STEMI by Shacham et al. found that the incidence of AKI following STEMI was 9.7%.[2] Tsai et al. reported that 7.1% of patients with STEMI who underwent PCI developed AKI; of those, 0.3% needed dialysis.[3] In another trial comprising 3810 patients treated with STEMI, AKI was identified in 690 (18%) participants.[4]

One in every five patients develops AKI within the first 72 hours following primary percutaneous coronary intervention (primary PCI). This condition is significantly linked to both short- and long-term outcomes. There was a wide range (15-30%) in the reported incidence of AKI following Primary PCI in STEMI, depending on the study population, methodology, and definition of AKI. Contrast media intake procedures (including PCI) are associated with a much lower risk of AKI in the general population, with estimates ranging from 1% to 2%.5,6 This study was purposed to identify the risk factors for AKI among STEMI patients undergoing Primary PCI.

2. Methods

2.1 Study Design

This retrospective cohort study was conducted at Saiful Anwar General Hospital Malang. 597 STEMI patients who underwent Primary PCI (PPCI) from 2017 to July 2022 were registered. There were 568 patients included, and 29 patients were excluded for the following reasons: incomplete medical record data. Data obtained from medical records include age, gender, vital signs (blood pressure, heart rate), shock conditions, hemoglobin levels, leukocyte count, serum electrolytes (Sodium, Potassium), random blood sugar, Kidney Function Test (Urea, creatinine, eGFR), uric acid, culprit lesion, The Thrombolysis in Myocardial Infarction (TIMI) flow, and total volume contrast.

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Table 1. Characteristics of the group of patients with AKI in STEMI undergoing Primary PCI.

Variable		Non-AKI (n = 499)	AKI (n = 69)	p-value
Age (years) (mean ± SD)		57.4 ± 10.9	58.3 ± 9.4	0.538
Gender	Female (n, %)	106 (18.7%)	10 (1.8%)	0.192
	Male (n, %)	393 (69.2%)	59 (10.4%)	
Risk Factor				
Hypertension (n)		243 (42.8%)	38 (6.7%)	0.369
Diabetes Mellitus (n)		182 (32.0%)	21 (3.7%)	0.351
Dyslipidemia (n)		31 (5.5%)	5 (0.9%)	0.791
Smoker (n)		182 (32.0%)	29 (5.1%)	0.371
Family history of CAD (n)		30 (5.3%)	10 (1.8%)	0.020
Clinical Condition				
Systolic Blood Pressure (mmHg)		125.29 ± 29.02	122.87 ± 31.88	0.527
Diastolic Blood Pressure (mmHg)		76.63 ± 15.83	74.49 ± 17.40	0.38
Heart Rate (beats per minute)		83 ± 22	89 ± 27	0.113
Shock Condition		123 (21.8%)	31 (5.5%)	<0.001
Infarct Location	Anterior MI	145 (25.5%)	13 (2.3%)	0.019
	Anterior Extensive MI	56 (9.9%)	15 (2.6%)	
	Anteroseptal MI	27 (4.8%)	8 (1.4%)	
	Anterolateral MI	34 (6.0%)	3 (0.5%)	
	Inferior MI	103 (18.1%)	11 (1.9%)	
	Inferoposterior MI	75 (13.2%)	12 (2.1%)	
	Inferoposterior + RV MI	22 (3.9%)	6 (1.1%)	
	Inferior + RV MI	33 (5.8%)	1 (0.2%)	
	Posterior MI	4 (0.7%)	0 (0%)	
Killip	I	390 (68.7%)	23 (4.0%)	<0.001
	II	51 (9.0%)	7 (1.2%)	
	III	18 (3.2%)	13 (2.3%)	
	IV	40 (7.0%)	26 (4.6%)	
Laboratory Parameter				
Hemoglobin (g/dL)		13.56 ± 7.33	12.62 ± 2.63	0.289
Leucocyte (sel/UL)		9592 ± 2048	10840 ± 2235	0.246
Creatinine (mg/dL)		0.91 ± 0.21	1.97 ± 0.28	<0.001
Ureum (mg/dL)		37.93 ± 18.4	78.91 ± 13.15	<0.001
eGFR (mL/min/1.73m2)		68.74 ± 31.68	51.08 ± 21.8	<0.001
Uric acid		7.07 ± 3.93	7.24 ± 2.62	0.771
Random Blood Sugar (mg/dL)		165.87 ± 97.0	216.68 ± 141.53	<0.001
Na+ (mEq/L)		136.53 ± 7.55	136.55 ± 4.14	0.966
K+ (mEq/L)		3.94 ± 0.67	3.84 ± 0.59	0.233
Cardiac Catheterization				
Culprit Lesion	Left main	27 (4.8%)	1 (0.2%)	<0.001
	Left anterior descending	161 (28.3%)	44 (7.7%)	
	Left circumflex	159 (28.0%)	21 (3.7%)	
	Right coronary artery	152 (26.8%)	3 (0.5%)	
TIMI Flow	0	3 (0.5%)	1 (0.2%)	<0.001
	1	52 (9.3%)	6 (1.1%)	
	2	165 (29.5%)	45 (8.0%)	
	3	272 (48.6%)	16 (2.9%)	
Total Contrast Volume(ml)		45.66 ± 71.32	85.33 ± 73.12	<0.001

Note. All data were presented by mean SD; AKI = acute kidney injury; CAD = coronary artery disease; eGFR = estimated glomerular filtration rate; MI = myocardial infarction; Primary PCI = primary percutaneous coronary intervention; RV = right ventricle; STEMI = ST-Elevation Myocardial Infarction; TIMI = The Thrombolysis in Myocardial Infarction.

2.2 End Points

Patients were classified as either having AKI or non-AKI. According to the KDIGO 2012 criteria, AKI was diagnosed when the serum creatinine (sCr) level increased by more than 0.3 mg/dL from admission. We used univariate analysis to examine all measured variables between individuals with an increase in sCr of >0.3 mg/dL and those without an increase in sCr to identify which ones were independently related to AKI. To identify potential risk factors for AKI, multivariate analysis was performed on variables with a P-value of 0.05 from univariate analysis. Calculations of the Total Contrast Volumesensitivity and specificity were made.

2.3 Statistical Analysis

For all data, the mean and standard deviation are shown. Comparing categorical variables was done using the Chi-Square (x2). Mean differences for numeric variables were examined using two-sample t-tests. To find potential risk factors, we conducted both univariate and multivariate analyses. All variables with a univariate P-value of 0.05 underwent a multivariate analysis. Using logistic regression, a multivariate analysis was performed. Receiver operating characteristic (ROC) curves for these variables were developed. For each positive variable, receiver operating characteristic curves were compared using the area under the curve (AUC), as well as sensitivity and specificity estimations. In order to analyze the data, SPSS 26.0 was used.

3. Results

Table 1 indicates that among STEMI patients following initial Primary PCI, 69 patients (12.14%) with AKI and 499 patients (87.86%) without AKI. In STEMI patients following Primary PCI, there was a significant difference between the groups of patients with and without AKI. The risk variables for a family history of CAD (p-value = 0.020), the presence of cardiogenic shock (p-value <0.001), infarct location (p-value = 0.019), and Killip class (p-value = <0.001) were significantly different between both groups. The significant laboratory parameters between the two groups to be a risk factor for AKI include Random Blood Sugar (RBS) with a p-value of <0.001. From the cardiac catheterization results analysis, we observed significant differences in Culprit Lesion (p-value <0.001), TIMI Flow (p-value <0.001), and total contrast volume (p-value <0.001) between both groups. Furthermore, multivariate analysis with logistic regression was performed on those variables.

The odds ratio (OR) value of each variable was obtained from multivariate analysis with logistic regression for the incidence of AKI. The OR values for each were as follows: Shock condition (OR = 1.41; 95% CI 1.18 – 1.92); Killip ≥3 (OR = 3.54; 95% CI 2.14 – 4.26); LAD culprit lesions (OR = 0.34; 95% CI 0.27 – 0.68); and Total Contrast Volume> 145 ml (OR = 1.61; 95% CI 1.13 – 1.92). Based on the ROC curve analysis, Total Contrast Volume> 145 ml with an area under the curve (AUC) of 0.75 (95% CI 0.65-0.85) with a specificity of 0.66 (95% CI 0.61 – 0.71) and a sensitivity of 0.71 (95% CI 0.0.65-0.76).

4. Discussion

In this study, 12.14% of STEMI patients following primary percutaneous coronary intervention experienced AKI. According to research by Goriki et al., the incidence of AKI in patients with STEMI ranges from 10 to 20%. The presence of AKI impacts clinical outcomes in hospitals and long-term outcomes.[1]

Shacham et al. conducted a retrospective analysis on 386 STEMI patients and found that the incidence of AKI following STEMI was 9.7%.2 Tsai et al. discovered that 7.1% of patients with STEMI who had to undergo PCI developed AKI, with 0.3% requiring dialysis therapy.[3] In a separate trial with 3,810 patients treated with STEMI, AKI was observed in 690 (18%) patients, according to Schmucker et al. The incidence of AKI was more significant in STEMI patients following primary percutaneous coronary intervention compared to those undergoing elective diagnostic coronary angiography. According to the prior study, acute myocardial infarction might produce kidney damage even in the absence of contrast media.[7]

The baseline characteristics of patients with AKI in STEMI patients who underwent Primary PCI were dominated by male patients (10.4%) with a mean age of 58.3 ± 9.4 years. From research conducted by Sinkovic et al., it was stated that the average age of patients with AKI in STEMI patients who underwent PPCI was 66.3 ± 12.0, with the dominance of male patients being 13.8%.8

Table 2. Multivariate Analysis with Logistic Regression

Variable	OR	95% CI		p-value
		Lower	Upper	
Shock Condition	1.41	1.18	1.92	0.042
Killip ≥ 3	3.54	2.14	4.26	<0.001
Culprit Lesion (LAD)	0.340	0.27	0.68	<0.001
Total Contrast Volume (>145 ml)	1.61	1.13	1.92	0.046

Note. significant if the p-value is 0.05 or lower

In this analysis, shock condition was an independent risk factor that occurred in 5.5% of the population, with an OR of 1.41 (95% CI, 1.18 to 1.92). A previous study revealed that the shock was linked to an increase in acute renal impairment (OR 9.7, CI 8.5-48.5; P 0.001). Shock, volume depletion, heart failure with reduced ejection fraction, venous congestion, hepatorenal syndrome, and hypercalcemia can all result in potentially reversible renal hypoperfusion, which temporarily lowers GFR without causing parenchymal injury.[9] However, as an ischemia condition, ischemia of tubular injury may progress to tubular necrosis. The Renin-Angiotensin-Aldosterone System (RAAS) and stimulation of the systemic nervous system cause afferent vasoconstriction, reduced renal blood flow, and decreased adequate glomerular perfusion pressure in shock patients.[10]

Patients with Killip presentation greater than three were independent risk variables for the incidence of AKI in STEMI patients after Primary PCI, with OR = 3.54 (95% CI 2.14 - 4.26), based on the clinical status of the patient. This is corroborated by a study by Matějka et al., which found that individuals with heart failure have a four times higher risk of developing AKI than patients without heart failure.[7] The most frequent causes of kidney injury are atheroembolic events during catheterization, more significant volumes of contrast media, and renal hypoperfusion circumstances brought on by heart failure or prolonged hypotension.[7,11] The study by Shacham et al. stated that 1656 STEMI patients with decreased left ventricular ejection fraction and congestive heart failure were individual predictors of AKI incidence with p < 0.001 (OR 2.44; (95% CI 1.51 – 3.96).[2]

Acute heart failure is significantly influenced by hemodynamic processes, which leads to a reduction in renal arterial flow and glomerular filtration rate (GFR). In this investigation, the Left Anterior Descending (LAD) artery was a risk factor for AKI in STEMI patients after primary PCI, with an odds ratio (OR) of 0.34 (95% confidence interval [CI]: 0.27 to 0.70).

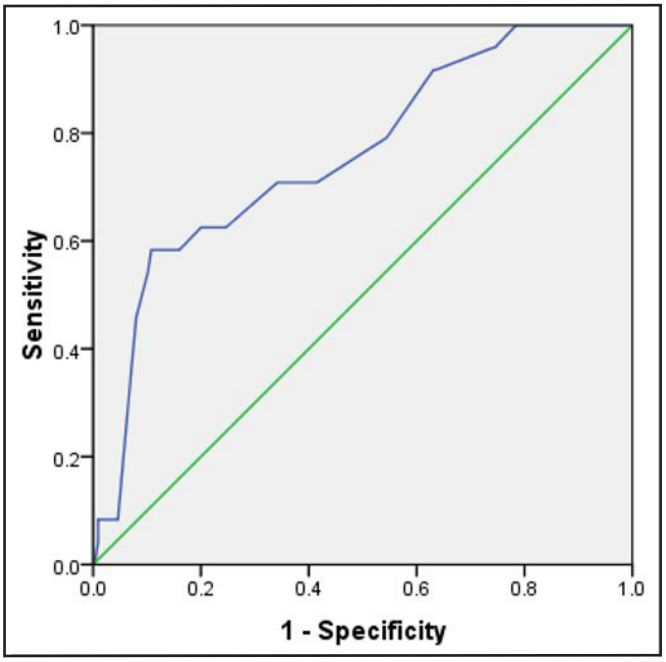

Figure 1. ROC curve to determine the sensitivity and specificity of the amount of contrast to the incidence of acute renal impairment in STEMI patients undergoing primary percutaneous intervention

According to a study by Silvain et al., the Left Anterior Descending (LAD) artery supplies the anterior region, where anterior infarct sites have a greater risk of heart failure and cardiogenic shock.[11] Research conducted by Hayıroğlu et al. In 492 STEMI patients with cardiogenic shock complications, it was found that the left anterior descending artery was a significant culprit lesion in the AKI group with $p < 0.001$.12 A study conducted by Wang et al., it was found that the risk factors that influence the incidence of AKI in STEMI patients include Killip class ≥3 (OR 5.22, 95% CI 3.07–8.87, P = 0.000) and extensive anterior MI (OR 3.02, 95% CI 1.85–4.93, P = 0.000).[13]

Contrast volume greater than 145 ml was also an independent risk factor for AKI, with OR = 1.61 (95% CI: 1.12 to 1.92). According to some views, the mechanism underlying the occurrence of AKI under clinical settings of STEMI and primary percutaneous coronary intervention (PPCI) is influenced by numerous factors. On renal tubular cells, contrast substances are known to have direct cytotoxic effects as well as indirect cytotoxic effects brought on by modifications in renal blood flow that create localized hypoxia. Contrarily, as a result of decreased cardiac output and increased venous congestion, the fundamental pathogenetic cause of AKI is altered systemic and renal hemodynamics.[14,15]

Furthermore, there is a mismatch between endogenous vasodilators and vasoconstrictors. Additional kidney damage could potentially result from severe inflammation and immune activity. AKI consequently occurs directly from the pathophysiological response during the post-AMI period.[1] This study could not differentiate between the impact of administering contrast media and these other hemodynamic abnormalities because sympathetic hyperactivity, renin-angiotensin-activation of the aldosterone system, the release of adenosine, and oxidative stress are known to contribute to the development of AKI in STEMI patients with heart failure. [14,15]

5. Conclusion

We successfully identified the risk factors for AKI among STEMI patients undergoing primary PCI. Our study revealed that the risk factors for AKI among STEMI patients undergoing primary PCI were shock condition, Killip class ≥3, and total contrast volume >145 ml.

6. Declarations

6.1. Ethics Approval and Consent to participate
This study was approved by local Institutional Review Board, and all participants have provided written informed consent prior to involvement in the study.

6.2. Consent for publication
Not applicable.

6.3. Availability of data and materials
Data used in our study were presented in the main text.

6.4. Competing interests
Not applicable.

6.5. Funding source
Not applicable.

6.6. Authors contributions
Idea/concept: YPA, BS. Design: YPA, BS. Control/supervision: BS, SA, IP, AF. Literature search: BS, SA, IP, AF. Data extraction: YPA, BS. Statistical analysis: YPA, BS. Results interpretation: YPA, BS. Critical review/discussion:BS, SA, IP, AF. Writing the article: SW, NK. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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