Predictors of Acute Kidney Injury in Patients Undergoing Left Main Percutaneous Coronary Intervention: Insights from the Taiwan Transcatheter Therapeutics Complex and High-Risk Indicated Patients (TTT-CHIP) Registry

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Abstract

Background: Acute kidney injury (AKI) increases morbidity, prolongs hospital stays, and elevates in-hospital mortality rates in patients undergoing left main coronary artery (LMCA) percutaneous coronary intervention (PCI).

Methods: This study analyzed a cohort of patients undergoing LMCA PCI in the Registry of Taiwan Transcatheter Therapeutics - Complex and High-risk Indicated Patients to identify predictors of AKI.

Results: Univariable logistic regression analysis showed that procedure time, use of inotropic agents and mechanical circulatory support (MCS) devices, including intra-aortic balloon pump, percutaneous cardiopulmonary support, Impella, or extracorporeal membrane oxygenation were significantly associated with AKI ($p < 0.05$ for all comparisons). In the multivariable analysis, procedure time (OR: 1.01 per minute, 95% CI: 1.001-1.02, $p = 0.029$) and use of MCS devices (OR: 6.74, 95% CI: 1.50-30.18, $p = 0.013$) remained significantly associated with the development of AKI. The primary significant factor identified in the decision tree was the use of MCS devices (adjusted $p = 0.000$, chi-square = 14.6). In patients not requiring MCS devices (n = 110), procedure time (> 114 minutes) was the next most significant factor (adjusted $p = 0.034$, chi-square = 9.8).

Conclusion: A decision tree model revealed a straightforward two-step risk stratification in which the use of MCS and a procedure time threshold of 114 minutes represented critical decision points for patients undergoing LMCA PCI. This model may facilitate early identification of high-risk patients and guide the implementation of targeted renal-protective strategies in clinical practice.

Keywords: acute kidney injury, left main coronary artery disease, percutaneous coronary intervention

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Introduction

Acute kidney injury (AKI) is a serious complication, particularly among high-risk patients undergoing complex procedures such as left main coronary artery (LMCA) percutaneous coronary intervention (PCI).[1] AKI increases morbidity, prolongs hospital stays, and elevates in-hospital mortality rates.[2] Understanding the predictors of AKI in this patient population is crucial for improving clinical outcomes and guiding therapeutic strategies.

LMCA disease accounts for approximately 4-6% of all PCI procedures.[3] PCI is considered a revascularization option in the treatment of LMCA disease, comparable to coronary artery bypass grafting (CABG), particularly in patients with a low SYNTAX score (SYNTAX score < 33).[4] Generally, PCI is reserved for patients who are not suitable candidates for CABG or when rapid revascularization is required due to hemodynamic instability or ongoing myocardial ischemia.

The complexity of PCI procedures for LMCA disease often involves high-risk features such as calcified lesions, bifurcations and chronic total occlusions. These cases require advanced techniques, including rotational atherectomy, intravascular imaging, and mechanical circulatory support (MCS).[5-7] Additionally, LMCA PCI typically entails longer procedure times and the use of larger volumes of contrast media to achieve optimal revascularization.[8] Patients undergoing LMCA PCI frequently present with multiple comorbidities, including advanced age, diabetes mellitus (DM), and chronic kidney disease (CKD), which further increase their susceptibility to AKI. These factors collectively contribute to the heightened risk of AKI following LMCA PCI[9-11]

Although previous studies have investigated various risk factors for AKI following PCI, only limited data are available specifically for patients undergoing LMCA PCI. This study aims to address this gap by analyzing a cohort of patients undergoing LMCA PCI to identify significant predictors of AKI. By understanding these risk factors, clinicians can better tailor interventions and improve the overall management of this high-risk patient population.

Methods

This study was a retrospective cohort analysis of patients undergoing complex, high-risk indicated PCI in the Registry of Taiwan Transcatheter Therapeutics - Complex and High-risk Indicated Patients (TTT-CHIP).[12] Between 2014 and 2022, patients who were at least 18 years old were enrolled from several tertiary medical heart centers if they met the TTT-CHIP criteria, which were as follows: (1) SYNTAX score > 32, and (2) undergoing PCI for complex coronary anatomy, such as true LMCA disease or heavily calcified lesions requiring cutting balloon or debulking techniques, or presenting with high-risk clinical features, including cardiogenic shock, acute myocardial infarction, or left ventricular ejection fraction (LVEF) < 40%. This study focused on patients who underwent LMCA PCI. Patients with end-stage renal disease undergoing regular hemodialysis were excluded. Clinical, demographic, and procedural data were collected from electronic medical records to evaluate risk factors associated with AKI following LMCA PCI.

The primary outcome in this study was the incidence of AKI following LMCA PCI. AKI was defined as an increase in serum creatinine of at least 0.3 mg/dL within 48 hours after the procedure or at least 50% increase from baseline within 7 days post-procedure. The incidence of AKI was recorded and analyzed to assess its association with various clinical, demographic and procedural variables.

Continuous variables were initially tested for normality using the Shapiro-Wilk test. Variables with a normal distribution were presented as mean ± standard deviation and compared using independent sample t-tests. Non-normally distributed continuous variables were presented as median with interquartile range

and compared using the Mann-Whitney U test. Categorical dichotomous variables were presented as counts and percentages, and compared across groups using chi-square tests or Fisher's exact tests. Both categorical and continuous variables were included in univariable and multivariable logistic regression models to identify significant predictors of AKI. In the univariable analysis, variables with a p-value < 0.1 were considered potential candidates for the multivariable model. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to estimate the strength of associations with AKI.

A decision tree model was implemented using the Classification and Regression Tree algorithm to identify the key predictors of AKI. The Gini impurity criterion was used to determine the optimal split points for the selected variables. The findings from the decision tree analysis were used to supplement and validate the results obtained from multivariable logistic regression, thereby enhancing the robustness of the study's conclusions regarding the risk factors for AKI following LMCA PCI. All statistical analyses were performed using the SPSS 23 software package (IBM Corp., Armonk, NY, USA).

Results

The TTT-CHIP registry included 454 patients. Of these, 141 patients who underwent LMCA PCI and did not have end-stage kidney disease were included in the study. Among them, 23 (16.3%) developed AKI, while 118 (83.7%) did not. The demographic and clinical characteristics of the two groups are summarized in Table 1.

There was no statistically significant difference in gender distribution between the groups, although a higher proportion of women developed AKI (39.1% vs. 24.6% in the non-AKI group, p = 0.15). The mean age of patients was similar in the AKI and non-AKI groups (67.7 ± 14.3 years vs. 69.5 ± 13.3 years, p = 0.56). Systolic blood pressure was 130.8 ± 22.5 mmHg in the AKI group and 132.1 ± 25.2 mmHg in the non-AKI group (p = 0.82), while diastolic blood pressures were 71.8 ± 14.3 mmHg and 73.8 ± 17.1 mmHg, respectively (p = 0.61). Body weight was also similar in both groups (p = 0.78).

The distribution of admission status, including stable angina, unstable angina, non-ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI), did not differ significantly between the two groups (p = 0.56). There was also no significant difference in the prevalence of DM (72.7% in the AKI group vs. 62.9% in the non-AKI group, p = 0.38) and CKD (40.9% in the AKI group vs. 48.3% in the non-AKI group, p = 0.64).

Pre-procedural creatinine levels were higher in the AKI group (3.0 ± 2.3 mg/dL) than in the non-AKI group (2.5 ± 2.6 mg/dL); however, this difference was not statistically significant (p = 0.49). Similarly, post-PCI troponin-I levels were elevated in the AKI group (78.1 ± 132.0 ng/mL) compared to the non-AKI group (50.2 ± 117.4 ng/mL), but this difference also failed to reach statistical significance (p = 0.51). Pre-PCI hemoglobin levels showed a trend toward significance, with lower levels in the AKI group (11.5 ± 2.5 g/dL) compared to the non-AKI group (12.5 ± 2.6 g/dL, p = 0.08). There were no significant differences in LVEF (47.9 ± 13.5% in the AKI group vs. 49.9 ± 15.6% in the non-AKI group, p = 0.61) or in the prevalence of statin medication at admission (68.2% in the AKI group vs. 66.1% in the non-AKI group, p = 0.85).

There was no significant difference between the two groups in the use of radial arterial access (39.1% in the AKI group vs. 47.5% in the non-AKI group, p = 0.46). The total contrast volume used during PCI was higher in the AKI group, although this difference was not statistically significant (230.6 ± 91.5 mL vs. 202.2 ± 82.1 mL, p = 0.22). Patients who developed AKI had significantly longer procedure times (185.2 ± 52.3 minutes vs. 130.3 ± 61.4 minutes, p = 0.001), but there was no significant difference in the number of stents placed (2.5 ± 1.6 in the AKI group vs. 2.5 ± 1.1 in the non-AKI group, p = 0.93). Complete

Table 1. Demographic and clinical characteristics of the two groups

Variable	Total (n=141)	AKI		p value
		No (n=118)	Yes (n=23)	
Gender				0.15
Male	103 (73.1%)	89 (75.4%)	14 (60.9%)	
Female	38 (27.0%)	29 (24.6%)	9 (39.1%)	
Age, year	69.2 (13.5)	69.5 (13.3)	67.7 (14.3)	0.56
Systolic bp (mmHg)	131.9 (24.7)	132.1 (25.2)	130.8 (22.5)	0.82
Diastolic bp (mmHg)	73.4 (16.6)	73.8 (17.1)	71.8 (14.3)	0.61
Body weight (kg)	66.0 (13.5)	66.2 (13.7)	65.3 (13.1)	0.78
Admission status				0.56
Stable angina	26 (18.8%)	24 (20.7%)	2 (9.1%)	
Unstable angina	29 (21.0%)	25 (21.6%)	4 (18.2%)	
NSTEMI	71 (51.5%)	57 (49.1%)	14 (63.6%)	
STEMI	12 (8.7%)	10 (8.6%)	2 (9.1%)	
DM				0.38
No	49 (35.5%)	43 (37.1%)	6 (27.3%)	
Yes	89 (64.5%)	73 (62.9%)	16 (72.7%)	
CKD				0.64
No	73 (52.9%)	60 (51.7%)	13 (59.1%)	
Yes	65 (47.1%)	56 (48.3%)	9 (40.9%)	
Cr prePCI (mg/dL)	2.6 (2.6)	2.5 (2.6)	3.0 (2.3)	0.49
Troponin-I post-PCI (ng/mL)	55.0 (119.3)	50.2 (117.4)	78.1 (132.0)	0.51
Hb pre-PCI (g/dL)	12.4 (2.6)	12.5 (2.6)	11.5 (2.5)	0.08
LVEF (%)	49.5 (15.2)	49.9 (15.6)	47.9 (13.5)	0.61
Statin medication at admission				0.85
No	47 (33.6%)	40 (33.9%)	7 (31.8%)	
Yes	93 (66.4%)	78 (66.1%)	15 (68.2%)	
Arterial access for coronary intervention Radial				0.46
No	76 (53.9%)	62 (52.5%)	14 (60.9%)	
Yes	65 (46.1%)	56 (47.5%)	9 (39.1%)	

(Continued)

Table 1. *(Continued)*

Variable	Total (n=141)	AKI		p value
		No (n=118)	Yes (n=23)	
Total contrast amount (mL)	209.5 (84.9)	202.2 (82.1)	230.6 (91.5)	0.22
Procedure time (minute)	140.1 (63.3)	130.3 (61.4)	185.2 (52.3)	0.001
Stent amount	2.5 (1.2)	2.5 (1.1)	2.5 (1.6)	0.93
Complete revascularization				0.03
No	52 (36.9%)	48 (40.7%)	4 (17.4%)	
Yes	89 (63.1%)	70 (59.3%)	19 (82.6%)	
IABP or PCPS or Impella or ECMO during PCI /after PCI				<0.001
No	110 (78.0%)	99 (83.9%)	11 (47.8%)	
Yes	31 (21.9%)	19 (16.1%)	12 (52.2%)	
Dopamine, levophed, dobutamine or adrenaline during PCI				0.02
No	115 (81.6%)	101 (85.6%)	14 (60.9%)	
Yes	26 (18.4%)	17 (14.4%)	9 (39.1%)	
Imaging device FFR or iFR				0.42
No	138 (97.9%)	116 (98.3%)	22 (95.7%)	
Yes	3 (2.1%)	2 (1.7%)	1 (4.4%)	
Imaging device IVUS				0.20
No	40 (28.3%)	36 (30.5%)	4 (17.4%)	
Yes	101 (71.6%)	82 (69.5%)	19 (82.6%)	
Imaging device OCT				0.19
No	136 (96.5%)	115 (97.5%)	21 (91.3%)	
Yes	5 (3.6%)	3 (2.5%)	2 (8.7%)	

AKI: acute kidney injury, CKD: chronic kidney disease, Cr: creatinine, DM: diabetes mellitus, ECMO: extracorporeal membrane oxygenation, FFR: fractional flow reserve, Hb: hemoglobin, IABP: intra-aortic balloon pump, IVUS: intra-coronary ultrasound, iFR: instantaneous wave-free ratio, LVEF: left ventricular ejection fraction, NSTEMI: non-ST elevation myocardial infraction, OCT: optical coherence tomography, PCPS: percutaneous cardiopulmonary support, PCI: percutaneous coronary intervention, STEMI: ST elevation myocardial infraction.

revascularization was more common in the AKI group (82.6% vs. 59.3%, p = 0.03). The use of MCS devices, including intra-aortic balloon pump (IABP), percutaneous cardiopulmonary support (PCPS), Impella and extracorporeal membrane oxygenation (ECMO) during or after PCI was significantly more frequent in patients who developed AKI (52.2% vs. 16.1%, p < 0.001). Similarly, the use of inotropic agents such as dopamine, norepinephrine, dobutamine or adrenaline during PCI was more common in the AKI group (39.1% vs. 14.4%, p = 0.02). The use of fractional flow reserve (FFR), instantaneous wave-free ratio (iFR), intravascular ultrasound (IVUS), and optical coherence tomography (OCT) did not differ significantly between the two groups (p > 0.05 for all comparisons).

Table 2 presents the results of both univariable and multivariable logistic regression analyses assessing factors associated with the development of AKI following LMCA PCI. In the univariable analysis, gender, age, systolic blood pressure, diastolic blood pressure, body weight and admission status did not show significant associations with AKI (p > 0.05 for all comparisons).

Neither DM nor CKD were significantly associated with AKI (For DM: OR: 1.57, 95% CI: 0.57-4.32, p = 0.38; For CKD: OR: 0.74, 95% CI: 0.29-1.87, p = 0.53). Other variables, including creatinine (OR: 1.06 per mg/dL, 95% CI: 0.90-1.24, p = 0.49), troponin-I (OR: 1.00 per ng/mL, 95% CI: 0.99-1.01, p = 0.51), hemoglobin (OR: 0.85 per g/dL, 95% CI: 0.71-1.02, p = 0.08), LVEF (OR: 0.99, 95% CI: 0.96-1.02, p = 0.61), and the prevalence of statin use at admission (OR: 1.10, 95% CI: 0.41-2.91, p = 0.85) also did not show statistically significant associations with AKI.

The use of radial arterial access (OR: 0.71, 95% CI: 0.29-1.77, p = 0.47) and the amount of contrast used during the procedure (OR: 1.00 per mL, 95% CI: 0.99-1.01, p = 0.23) were not significantly associated with AKI. However, procedure time was significantly associated with AKI (OR: 1.01 per minute, 95% CI: 1.01-1.02, p = 0.002). The number of stents placed was not significantly associated with AKI (OR: 1.02, 95% CI: 0.70-1.50, p = 0.91), and complete revascularization showed a trend toward significance (OR: 2.86, 95% CI: 0.80-10.24, p = 0.11). The use of MCS devices, including IABP, PCPS, Impella or ECMO during or after PCI, was significantly associated with the development of AKI (OR: 5.68, 95% CI: 2.19-14.75, p < 0.001). Similarly, the use of inotropic agents such as dopamine, norepinephrine, dobutamine or adrenaline during PCI was significantly associated with AKI (OR: 3.82, 95% CI: 1.43-10.20, p = 0.008). The use of FFR or iFR (OR: 2.64, 95% CI: 0.23-30.34, p = 0.44), IVUS (OR: 2.09, 95% CI: 0.66-6.57, p = 0.21) or OCT (OR: 3.65, 95% CI: 0.57-23.18, p = 0.17) was not significantly associated with AKI.

In the multivariable analysis, procedure time remained significantly associated with the development of AKI (OR: 1.01 per minute, 95% CI: 1.001-1.02, p = 0.029). The use of MCS devices also continued to show a strong association with AKI (OR: 6.74, 95% CI: 1.50-30.18, p = 0.013). However, after multivariable adjustment, the use of inotropic agents during PCI was no longer significantly associated (OR: 0.91, 95% CI: 0.15-5.34, p = 0.92). Other variables did not show significant associations with AKI in the multivariable analysis (p > 0.05 for all comparisons).

The results of the decision tree are shown in Figure 1. The primary significant factor identified in the decision tree was the use of MCS devices. Among patients who required MCS, 38.7% (n = 12) developed AKI, compared to only 10.0% (n = 11) among those who did not use these devices (adjusted p-value = 0.000, chi-square = 14.6). Among patients who did not require MCS devices (n = 110), procedure time was identified as the next significant factor. Among patients with procedure times less than or equal to 114 minutes, only 1.7% (n = 1) developed AKI, compared to 19.6% (n = 10) of patients with procedure times

Table 2. Results of both univariable and multivariable logistic regression analyses

Variable	Univariable		Multivariable	
	OR (95% CI)	p value	OR (95% CI)	p value
Gender (Female vs. Male)	1.97 (0.77-5.03)	0.16		
Age, year	0.99 (0.96-1.02)	0.56		
Systolic bp (mmHg)	0.99 (0.98-1.02)	0.82		
Diastolic bp (mmHg)	0.99 (0.97-1.02)	0.60		
Body weight (kg)	0.99 (0.96-1.03)	0.78		
Admission status				
Unstable angina vs. Stable angina	1.92 (0.32-11.46)	0.47		
NSTEMI vs. Stable angina	2.95 (0.62-13.97)	0.17		
STEMI vs. Stable angina	2.40 (0.30-19.48)	0.41		
DM (Yes vs. No)	1.57 (0.57-4.32)	0.38		
CKD (Yes vs. No)	0.74 (0.29-1.87)	0.53		
Cr pre-PCI (mg/dL)	1.06 (0.90-1.24)	0.49		
Troponin-I post-PCI (ng/mL)	1.00 (0.99-1.01)	0.51		
Hb pre-PCI (g/dL)	0.85 (0.71-1.02)	0.08		
LVEF (%)	0.99 (0.96-1.02)	0.61		
Statin medication at admission (Yes vs. No)	1.10 (0.41-2.91)	0.85		
Arterial access for coronary intervention Radial (Yes vs. No)	0.71 (0.29-1.77)	0.47		
Total contrast amount (mL)	1.00 (0.99-1.01)	0.23		
Procedure time (minute)	1.01 (1.01-1.02)	0.002	1.01 (1.001-1.02)	0.029
Stent amount	1.02 (0.70-1.50)	0.91		
Complete revascularization (Yes vs. No)	2.86 (0.80-10.24)	0.11		
IABP or PCPS or Impella or ECMO during PCI /after PCI (Yes vs. No)	5.68 (2.19-14.75)	< 0.001	6.74 (1.50-30.18)	0.013
Dopamine, levophed, dobutamine or adrenaline during PCI (Yes vs. No)	3.82 (1.43-10.20)	0.008	0.91 (0.15-5.34)	0.92
Imaging device FFR or iFR (Yes vs. No)	2.64 (0.23-30.34)	0.44		
Imaging device IVUS (Yes vs. No)	2.09 (0.66-6.57)	0.21		
Imaging device OCT (Yes vs. No)	3.65 (0.57-23.18)	0.17		

AKI: acute kidney injury, CKD: chronic kidney disease, Cr: creatinine, DM: diabetes mellitus, ECMO: extracorporeal membrane oxygenation, FFR: fractional flow reserve, Hb: hemoglobin, IABP: intra-aortic balloon pump, IVUS: intra-coronary ultrasound, iFR: instantaneous wave-free ratio, LVEF: left ventricular ejection fraction, NSTEMI: non-ST elevation myocardial infraction, OCT: optical coherence tomography, PCPS: percutaneous cardiopulmonary support, PCI: percutaneous coronary intervention, STEMI: ST elevation myocardial infraction.

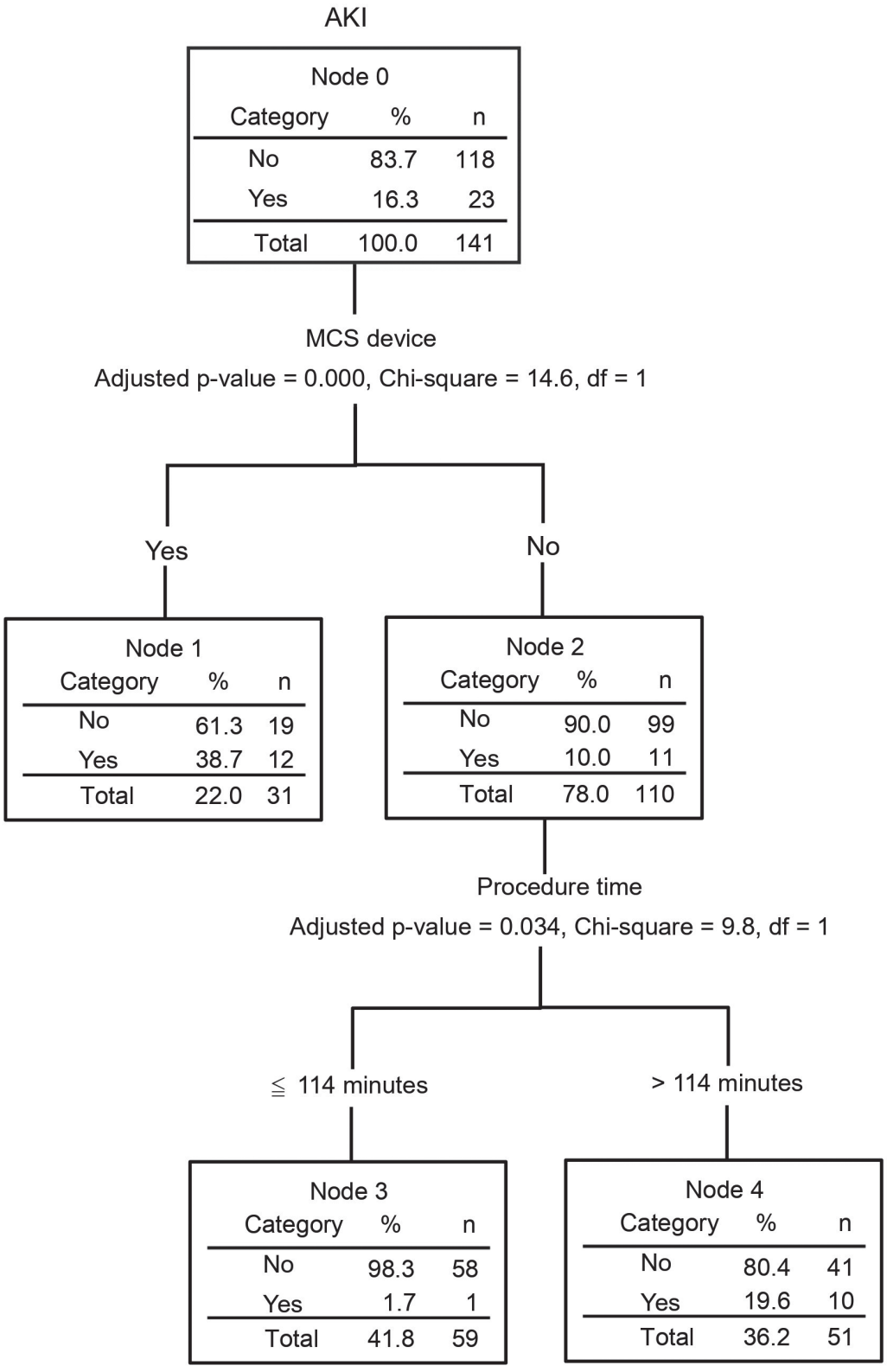

Figure 1. Results of decision tree analysis.

AKI: acute kidney injury; MCS: mechanical circulatory support.

exceeding 114 minutes (adjusted p-value = 0.034, chi-square = 9.8).

Discussion

While previous research has examined AKI risk factors in broader PCI populations, few studies have specifically focused on the subgroup of patients undergoing LMCA PCI. We identified two key factors associated with AKI in these patients: the use of MCS devices and prolonged procedure duration.

The use of MCS devices is typically reserved for patients with severe hemodynamic instability, such as those experiencing cardiogenic shock or presenting with high-risk coronary lesions. While MCS devices are essential for stabilizing cardiac function in these critically ill patients, they can also contribute to the development of AKI through several mechanisms. First, the use of MCS devices likely reflects underlying hemodynamic instability rather than being a direct causal factor for AKI. Second, MCS devices may not always ensure adequate renal perfusion, particularly in patients with pre-existing kidney dysfunction. In patients with fluctuating blood pressure or low cardiac output, renal hypoperfusion can be exacerbated, increasing the risk of AKI.[13-15] Third, devices such as Impella and ECMO can induce hemolysis, releasing free hemoglobin into the bloodstream, which is nephrotoxic. Furthermore, the interaction between blood and the artificial surfaces of MCS circuits often triggers an inflammatory response, leading to systemic oxidative stress that can further contribute to kidney injury.[16-18] Fourth, during MCS use, ischemic periods followed by reperfusion can cause direct damage to renal tissues, increasing the likelihood of AKI.[19] Lastly, patients on MCS frequently require aggressive fluid management to maintain adequate perfusion, which can lead to volume overload, venous congestion, and subsequent renal injury.[20]

Among patients who did not require MCS devices, our results demonstrated that those with prolonged procedure times (approximately 2 hours) had a significantly higher incidence of AKI. This finding suggests that longer, more complex procedures increase the risk of AKI, which may be due to several contributing factors. Extended procedures are often associated with greater use of contrast media, a well-known risk factor for contrast-induced nephropathy.[10,11] Additionally, prolonged procedures may indicate more challenging or high-risk interventions, which can lead to fluctuating hemodynamics, extended periods of hypotension, and increased mechanical stress on the kidneys. These conditions contribute to renal ischemia and subsequently elevate the likelihood of AKI. The present study shows that while the AKI group had longer procedure times, the total contrast volume did not differ significantly between the AKI and the non-AKI groups. This suggests that hemodynamic instability or mechanical stress associated with longer procedure duration might be a more critical factor for AKI in LMCA patients than contrast toxicity itself.

The decision tree model used in this study presents a novel, data-driven approach to predicting AKI in patients undergoing LMCA PCI.[21-24] While the traditional Mehran scoring system has been widely validated and remains a valuable tool for estimating AKI risk in general PCI populations, it relies on a fixed set of clinical and procedural variables and assigns predefined weights based on expert consensus and previous data. Although effective, this method may not fully capture the complex, nonlinear relationships among multiple risk factors, particularly in the context of left main PCI. By contrast, the decision tree model dynamically learns from data and can incorporate a broader range of variables, enabling it to identify complex interactions that conventional scoring methods may overlook. For example, the decision tree can illustrate how specific combinations of risk factors influence AKI risk. This approach enhances the model's flexibility and adaptability across different patient populations and clinical settings, facilitating a more personalized risk assessment.

Moreover, the decision tree model offers clear visual decision pathways, enhancing interpretability for clinicians by illustrating how specific variables and their thresholds contribute to the risk of AKI. In contrast to this, the Mehran score calculates a cumulative risk score but does not visually represent the interactions between risk factors or provide an intuitive understanding of their relative importance.

Clinical Implications and Future Directions

The findings of this study underscore the importance of careful management for patients undergoing LMCA PCI, especially those at increased risk of AKI due to the use of MCS devices or prolonged procedure durations. Future research should focus on exploring renal protection strategies for patients requiring MCS, paying particular attention to how different devices affect renal outcomes. Additionally, studies should investigate methods to optimize procedure times, as shorter procedures may help reduce the risk of AKI. Furthermore, the use of advanced imaging techniques, such as IVUS and OCT, along with physiological assessments like FFR or iFR, should be further evaluated to determine their role in improving PCI outcomes while minimizing AKI risk. Also, given that the threshold of 114 minutes is a very precise cut-off, a discussion on any correlation between this threshold and specific procedural complexities, such as the two-stent strategy for LMCA bifurcations, would be beneficial to provide more context for clinicians. Implementing a multidisciplinary approach involving cardiologists and nephrologists may further enhance AKI prevention and management, particularly in high-risk patient populations.

Limitations

This study has several limitations. First, the limitations of small sample size, lack of validation and residual confounding all apply. Second, the retrospective design may have introduced selection bias, as patients with more severe hemodynamic compromise were more likely to receive MCS devices, inherently increasing their risk of AKI. Additionally, unmeasured confounders may have influenced the observed associations due to the retrospective nature of the analysis. Third, there was a lack of some data on the total contrast volume used during PCI, which could have affected the development of AKI, especially in patients undergoing longer procedures or requiring MCS. This lack of complete contrast volume data limits our ability to fully assess its impact on AKI risk in this cohort. Finally, the use of imaging modalities such as IVUS and OCT, as well as physiological assessments like FFR or iFR, was relatively low in our registry. This limited utilization may have constrained our ability to evaluate the potential benefits of these advanced diagnostic tools in preventing or reducing AKI during PCI. Future studies with broader application of these technologies may provide better insights into their roles in mitigating AKI risk.

Conclusions

In patients undergoing left main PCI, the use of MCS and prolonged procedure time were the two key predictors of AKI. A decision tree model revealed a practical risk stratification pathway: patients requiring MCS had the highest risk, while those not requiring MCS but with procedure times exceeding 114 minutes also faced an elevated risk. This simple two-step model may assist clinicians in identifying high-risk patients in real time and guide the implementation of renal-protective strategies during complex PCI.

Declaration of Conflict of Interest

All authors declare that they have no conflicts of interest.

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