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Original Article

ASSOCIATION BETWEEN ADMISSION MEAN ARTERIAL PRESSURE AND THE DEVELOPMENT OF CONTRAST-ASSOCIATED ACUTE KIDNEY INJURY WITHIN THE FIRST 72 HOURS IN PATIENTS WITH NSTEMI

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ABSTRACT

Introduction: Contrast-associated acute kidney injury (CI-AKI) is a frequent and clinically important complication after percutaneous coronary intervention (PCI), particularly in hemodynamically unstable patients with non–ST-elevation myocardial infarction (NSTEMI). Mean arterial pressure (MAP) is a key determinant of renal perfusion, yet the impact of admission MAP on CI-AKI development in NSTEMI patients undergoing PCI has not been clearly established. To investigate the relationship between admission MAP and the development of CI-AKI within the first 72 hours after PCI in patients with NSTEMI.

Methods: This single-center, retrospective observational study included 150 consecutive patients hospitalized with NSTEMI who underwent PCI between January and December 2025. Admission systolic and diastolic blood pressures were obtained from emergency department triage records and MAP was calculated using the standard formula (MAP = [SBP + 2×DBP]/3). CI-AKI was defined according to KDIGO criteria as an increase in serum creatinine ≥0.3 mg/dL or ≥50% from baseline within 72 hours after contrast exposure. Patients were divided into two groups: those who developed CI-AKI (n=62, 41.3%) and those who did not (n=88, 58.7%). Non-parametric tests and Spearman correlation analysis were used.

Results: The overall mean MAP was 88.99±27.44 mmHg. Patients who developed CI-AKI had significantly lower admission MAP compared with those without CI-AKI (60.33±3.95 vs. 109.18±16.76 mmHg; p<0.001). Lower MAP was positively correlated with body mass index and heart rate. Chronic kidney disease and hyperlipidemia were more frequent among patients with CI-AKI and were also associated with lower MAP values (both p<0.001).

Conclusion: In NSTEMI patients undergoing PCI, lower admission MAP is strongly associated with the development of CI-AKI within 72 hours. Given that MAP is a simple, rapid, and noninvasive parameter, its routine assessment at presentation may help identify high-risk patients and support clinical awareness regarding preventive care.

Keywords: Contrast-Associated Acute Kidney Injury, Non–ST-Elevation Myocardial Infarction, Mean Arterial Pressure

INTRODUCTION

Cardiovascular diseases are the leading cause of death worldwide (1). Ischemic heart disease is the leading type of cardiovascular disease (2, 3). Patients with non–ST-elevation myocardial infarction (NSTEMI), a subgroup of ischemic heart disease, require urgent treatment due to their high mortality and morbidity rates. In these patients, coronary arteries causing subendocardial ischemia are present. A diagnosis can be made based on the patient's medical history, pain characteristics, ECG and laboratory tests (4, 5). In patients with NSTEMI, cardiac output may cause a decrease in blood pressure, depending on the area of the heart supplied by the artery causing ischemia. This may be associated with an increased risk of acute kidney injury through reduced renal perfusion (6). Additionally, patients with NSTEMI may experience excessive activation of the renin-angiotensin-aldosterone system due to increased sympathetic activation. This results in increased angiotensin II, which causes vasoconstriction in the efferent arterioles. This decreases the glomerular filtration rate and reduces renal blood flow (7).

Another mechanism is that NSTEMI causes renal microvascular dysfunction by increasing inflammation and oxidative stress (8). In patients with NSTEMI scheduled for invasive procedures, iodinated contrast agents used during angiography can induce vasoconstriction in the afferent and efferent arterioles, resulting in nephrotoxic effects (9, 10).

Treatment with anti-ischemic agents can have a nephrotoxic effect. Several risk scoring systems are used to determine treatment strategies for patients with NSTEMI and to predict various adverse events. In most of these risk scores, the patient's serum creatinine level or estimated glomerular filtration rate (eGFR) is included directly in the model, contributing to the determination of both the short-term and long-term prognosis (11). Mean arterial pressure (MAP) is one of the most important indicators used to assess the perfusion of systemic organs. It is the average of the systolic and diastolic pressures during a cardiac cycle. The most common method of calculating MAP is the formula MAP = diastolic blood pressure + 1/3(systolic blood pressure – diastolic blood pressure) (12). The current literature

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suggests that an SBP below 65 mmHg can significantly reduce renal perfusion (13).

Contrast-associated acute kidney injury (CI-AKI) is most commonly defined in the current literature according to KDIGO (Kidney Disease: Improving Global Outcomes). CI-AKI is defined as an increase in serum creatinine of at least 0.3 mg/dL, or a 1.5-fold increase in serum creatinine, within the first 72 hours after exposure to contrast agents (14). We examined the relationship between MAP values at admission and CI-AKI after 72 hours in 150 patients diagnosed with NSTEMI and undergoing Percutaneous Coronary Intervention (PCI).

METHODS

This study was designed as a single-center, retrospective, observational study. A total of 150 consecutive patients diagnosed with NSTEMI and admitted to the PCI unit at Fırat University's Cardiology Department between 1 January and 1 December 2025 were included in the study.

The patient selection and inclusion criteria were as follows: patients were diagnosed with NSTEMI based on clinical, biochemical and electrocardiographic findings; they underwent PCI during the same hospital admission; they had complete blood pressure measurements at presentation; and they had available serum creatinine values at the 72-hour follow-up.

The exclusion criteria included patients with chronic kidney disease stage ≥4. patients receiving dialysis, patients with a prior history of contrast-induced nephropathy, patients presenting with acute infection, sepsis or hemodynamic instability, and patients with incomplete or missing data.

The patients' systolic and diastolic blood pressure values at the time of admission were obtained from the emergency department's triage records. MAP was calculated as previously described.

Admission MAP was defined as the first blood pressure measurement obtained at emergency department triage before the initiation of any medical treatment.

Serum creatinine levels 72 hours after PCI were recorded for all patients, as required by the follow-up protocol.

In accordance with the KDIGO criteria for AKI: defined as an increase in creatinine of at least 0.3 mg/dL from baseline, or a relative increase of over 50%, Patients were accordingly divided into two groups: those who developed CI-AKI, and those who did not.

The study population was divided into two equal groups based on whether or not they developed CI-AKI:

Group 1 comprised 62 patients who developed contrast-associated CI-AKI.

Group 2 comprised 88 patients who did not develop CI-AKI.

Statistical analysis

When summarizing the data obtained from the study, descriptive statistics were examined as the mean ± standard deviation or the median, minimum and maximum, depending on the distribution of the continuous (numerical) variables.

Categorical variables were expressed as counts and percentages, while the normality of numerical variables was assessed using the Shapiro–Wilk test, since n > 30. It was observed that the numerical variable did not exhibit a normal distribution. Accordingly, non-parametric techniques were employed for the data analysis.

Although the overall sample size was relatively large, the choice of non-parametric statistical methods was based on the distributional characteristics of the data rather than sample size alone. The normality assessment demonstrated significant deviation from normal distribution for the primary variable of interest (mean arterial pressure) as well as several secondary variables. Therefore, non-parametric methods were considered more appropriate and robust for group comparisons and correlation analyses.

In addition, multivariable regression models were not constructed in order to avoid potential overfitting and unstable estimates, given the non-normal distribution of the data and the limited number of outcome events. As a sensitivity check, the consistency of the main findings was evaluated using median-based comparisons, which yielded similar results and supported the robustness of the observed associations.

The non-parametric Spearman's rho correlation coefficient technique was used to compare continuous variables. This is the equivalent of the parametric Pearson product-moment correlation coefficient technique. The Mann-Whitney U test was used to compare two groups and the Kruskal–Wallis H test was used to compare three or more groups. When the Kruskal–Wallis H test yielded significant results, the Mann–Whitney U test was used to determine which groups showed significant differences.

When a significant difference was found between the two groups, Bonferroni correction was applied. The statistical analyses were performed using the SPSS 22.0 software package and a significance level of $p < 0.05$ was accepted. Multivariate regression analysis was not performed in order to avoid the risk of overfitting, given the non-normal distribution of the data and the small sample size.

RESULTS

The results of the normality analysis are presented in Table 1. According to the Shapiro–Wilk test, the distribution of MAP values was not normal ($p < 0.001$); therefore, non-parametric statistical methods were employed.

Table 1. Results of the Normality Test for the Variables Used in the Study

Variables	z	r
Mean Arterial Pressure	*0.909*	*0.001*

Statistical Method Used: Shapiro–Wilk Test

The demographic and clinical characteristics of the study population are summarized in Table 2. A total of 150 patients were included, 119 (79.3%) of whom were male and 31 (20.7%) of whom were female. The mean age was 62.44 ± 12.02 years and the mean body mass index was 26.86 ± 3.15 kg/m². The mean systolic and diastolic blood pressure values at admission were 124.51 ± 33.67 mmHg and 71.24 ± 25.47 mmHg, respectively. The mean heart rate was 75.97 ± 17.77 beats per minute and the mean admission MAP was 88.99 ± 27.44 mmHg.

In terms of clinical characteristics, 64.0% of patients had no prior history of coronary artery disease; 23.3% had experienced a previous myocardial infarction; 10.7% had stable coronary artery disease; and 2.0% had undergone coronary artery bypass grafting. Chronic kidney disease was present in 34.0% of patients and hyperlipidemia in 48.0%. Current smoking was reported by 46.7% of the study population. During follow-up, CI-AKI

Table 2. Distribution of Descriptive Characteristics (n=150)

Variables	N (Number)	% (Percent age)
Sex		
Female	*31*	*20.7*
Male	*119*	*79.3*
Age; *Mean±Sd:62.44±12.,02– Medyan:61.5 - Ranj: 53.00- Min: 37.0- Max: 90.0*		
Weight; *Mean±Sd:79.00±10.60 – Medyan: 79.00 - Ranj: 68.00 - Min: 51.0- Max: 119.0*		
Height; *Mean±Sd:171.82±7.85 – Medyan: 173.50 - Ranj: 36.00 - Min: 152.0 – Max: 182.0*		
BMI; *Mean±Sd:26.86±3.15– Medyan: 26.55 - Ranj: 18.89 - Min: 19.41 – Max: 38.30*		
SBP; *Mean±Sd:124.51±33.67– Medyan:130.0 - Ranj:170.00 - Min:60.0 – Max: 230.0*		
DBP; *Mean±Sd:71.24±25.47– Medyan: 73.5- Ranj:95.0 - Min: 35.0 – Max: 130.0*		
Heart Rate; Mean*±Sd:75.97±17.77–Medyan:76.00-Ranj:118.00 - Min: 33.0 – Max: 151.0*		
Coronary Artery Disease Status		
No	*96*	*64.0*
Previous MI	*35*	*23.3*
Previous CABG	*3*	*2.0*
CAD stable	*16*	*10.7*
Chronic Kidney Disease Status		
No	*99*	*66.0*
Yes	*51*	*34.0*
Mean Arterial Pressure;*Mean±Sd:88.99±27.44–Median:95.66- Ranj:117.00-Min:43.0– Max: 160.0*		
Hyperlipidemia Status		
No	*78*	*52.0*
Yes	*72*	*48.0*
Smoking Status		
No	*80*	*53.3*
Yes	*70*	*46.7*
CI-AKI Status		
No	*88*	*58.7*
Yes	*62*	*41.3*
Ejection fraction*; Mean±Sd:50.80±8.31– Medyan:55.0 -Ranj:40.00 - Min:25.0–Max:65.0*		

Abb. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MI, myocardial infarction; CABG, coronary artery bypass grafting; CAD, coronary artery disease; CKD, chronic kidney disease; MAP, mean arterial pressure; CI-AKI: Contrast-associated acute kidney injury; EF, ejection fraction; SD, standard deviation; Min, minimum; Max, maximum.

developed in 62 patients (41.3%). The mean left ventricular ejection fraction was 50.80±8.31%.

For patients, a statistically significant positive linear correlation ($r = 0.193$, $p < 0.05$) was found between MAP and BMI values. A statistically low level of positive linear correlation ($r = 0.210$, $p < 0.05$) was also found between MAP and heart rate values (Table 3). Although the correlations between MAP, body mass index and heart rate are statistically significant, they are weak. Therefore, these relationships should be considered supportive and secondary observations, rather than strong clinical indicators.

Table 3. Correlation Results for Mean Arterial Pressure Values and Certain Variables/Values

Variables	1.	2.	3.	4.	5.
1.Mean Arterial Pressure	*1.0*				
2. Age	*-.122***	*1.0*			
3.Body Mass Index	*.193**	*-.140**	*1.0*		
4.Heart Rate	*.210***	*.072*	*.047*	*1.0*	
5. Ejection fraction	*-.073*	*-.152**	*.049*	*-.199***	*1.0*

Statistical Method Used: Spearman rho Correlation Technique,*=p<0.05 - **=p< 0.001

The distribution of clinical variables between patients with and without CI-AKI is summarized below, and the results of the comparison are presented in Table 4.

It was found that 34.7% of patients with CI-AKI were male, compared to 44.7% of patients without CI-AKI. This difference was not found to be statistically significant ($p>0.05$).

Furthermore, 27.3% of patients with CI-AKI did not have CAD, compared to 36.7% of patients without CI-AKI. This distribution was also not found to be statistically significant ($p>0.05$).

It was found that 28.0% of patients with CI-AKI had chronic kidney disease (CKD), compared to 6.0% of patients without CI-AKI. This difference in distribution was found to be statistically significant ($p < 0.001$). In other words, CI-AKI is more prevalent among patients with CKD than among those without.

Furthermore, 30.7% of patients with CI-AKI were found to have hyperlipidemia, compared to 41.3% of patients without CI-AKI. This difference was also found to be statistically significant ($p < 0.001$). In other words, CI-AKI is more prevalent among patients with hyperlipidemia than among those without.

It was found that 23.3% of patients with CI-AKI were non-smokers, compared to 30.0% of patients without CI-AKI. This difference was not found to be statistically significant ($p>0.05$).

The difference in rank means between groups with regard to MAP values and CI-AKI status was found to be statistically significant (U: 1953.0, $p<0.05$). According to these results, patients with CI-AKI had lower MAP values than patients without CI-AKI ($p<0.001$) (Table 5).

A summary of the comparison of patients' MAP values according to various clinical variables is provided below, with the results presented in Table 6.

No statistically significant difference was found in the rank means of MAP values according to gender (M:1606.5 – $p>0.05$).

Table 4. Analysis Results Regarding the Presence or Absence of CI-AKI in Terms of Certain Variables (n:150)

Variables	Patients without CI-AKI		Patient with CI-AKI		Analysis result	
	n	%	n	%	X^2	p
Sex						
Female	*21*	*14.0*	*10*	*6.7*	1.32[a]	0.249*
Male	*67*	*44.7*	*52*	*34.7*		
Coronary Artery Disease Status						
NO	*55*	*36.7*	*41*	*27.3*		
Previous MI	*22*	*14.7*	*13*	*8.7*	.446[b]	0.931*
Previous CABG	*2*	*1.3*	*1*	*0.7*		
CAD stable	*9*	*6.0*	*7*	*4.7*		
Chronic Kidney Disease Status						
No	*79*	*52.7*	*20*	*13.3*	53.61[a]	0.001**
Yes	*9*	*6.0*	*42*	*28.0*		
Hyperlipidemia Status						
No	*62*	*41.3*	*16*	*10.7*	29.05[a]	0.001**
Yes	*26*	*17.3*	*46*	*30.7*		
Smoking Status						
No	*45*	*30.0*	*35*	*23.3*	.413[a]	0.521*
Yes	*43*	*28.7*	*27*	*18.0*		

Statistical Method Used:: a=Chi-Square Test, b=Fisher-Freeman-Halton Exact,*=p>0.05 - **=p< 0.001 level. MI: myocardial infarction; CABG :coronary artery bypass grafting; CAD: coronary artery disease.

The difference in rank means between groups with different MAP values and CKD status was found to be statistically significant (U:906.5, p<0.05). According to these results, patients with CKD have lower MAP values than those without CKD (p< 0.001).

The difference in the median MAP values between hyperlipidemia status groups was also found to be statistically significant (U:1784.5, p<0.05). According to these results, the MAP values of patients with hyperlipidemia were lower than those of patients without hyperlipidemia (p< 0.001).

DISCUSSION

CI-AKI is a serious complication of PCI procedures that increases mortality and morbidity. The current literature indicates that the main risk factors for the development of CI-AKI are advanced age, chronic renal failure, diabetes mellitus, hemodynamic instability, low ejection fraction, anemia and hyperlipidemia (15, 16). However, studies examining the effect of hemodynamic status at presentation on CI-AKI in NSTEMI patients undergoing PCI are limited.

Table 5. Comparison of Mean Arterial Pressure Values According to CI-AKI Status (n:150)

Variables	Mean Arterial Pressure Value					Statistical Analysis
	n	Mean	SD	Median	S.O.	
CI-AKI Status						*U:1953.0* *p:0.001**
No	*88*	*109.18*	*16.76*	*106.67*	*106.50*	
Yes	*62*	*60.33*	*3.95*	*60.00*	*31.50*	

Statistical Method Used: Mann Whitney U Test - *= p<0.001 level

The kidneys' autoregulatory mechanism can maintain a constant renal blood flow despite average pressure changes in the range of 80–180 mmHg, thanks to the vasomotor tone of the afferent and efferent arterioles (17). However, increased oxidative stress, as occurs in NSTEMI, increases activation of the sympathetic system and excessive activation of the renin-angiotensin-aldosterone system (RAAS) negatively affects autoregulatory mechanisms (6, 7). Studies have shown that MAP plays a decisive role in renal perfusion, and that low perfusion pressure (below 60 mmHg) increases the risk of renal ischemia (13). Our findings are consistent with the hypothesis that impaired renal autoregulation may be present in patients with lower MAP values.

Table 6. Comparison Results for Mean Arterial Pressure Values with Some Variables (n:150)

Variables	Mean Arterial Pressure Value					Statistical Analysis
	n	Mean	SD	Median	S.O.	
Sex						*U:1606.5* *p:0.268*
Female	*31*	*94.48*	*27.23*	*96.67*	*83.18*	
Male	*119*	*87.56*	*27.42*	*95.33*	*73.50*	
Chronic Kidney Disease Status						*U:906.5* *p:0.001**
No	*99*	*99.66*	*24.91*	*103.33*	*91.84*	
Yes	*51*	*68.28*	*19.09*	*63.33*	*43.77*	
Hyperlipidemia Status						*U:1784.5* *p:0.001**
No	*78*	*98.06*	*25.02*	*98.67*	*88.62*	
Yes	*72*	*79.17*	*26.70*	*63.33*	*61.28*	

Statistical Method Used: Mann Whitney U Test - *= p< 0.001 level

In our study, the relationship between MAP at the time of admission and CI-AKI developing at 72 hours was evaluated in 150 patients who underwent PCI with a diagnosis of NSTEMI. The results showed that MAP values were significantly lower in patients who developed CI-AKI. Hemodynamic instability was associated with a higher occurrence of CI-AKI, likely reflecting impaired renal perfusion in this vulnerable population. This result highlights the adverse effect of hemodynamic instability on renal perfusion and is consistent with the existing literature. The lack of a strong association between ejection fraction and MAP suggests that MAP reflects global perfusion status rather than isolated left ventricular systolic function.

Yoshizawa and colleagues demonstrated that CI-AKI risk is increased in hypotensive patients undergoing contrast-enhanced computed tomography (18). Similarly, in a study by Liu et al., low preoperative MAP was shown to be an independent risk factor for the development of CI-AKI (19).

Previous studies have shown that CKD is the most important risk factor for the development of CI-AKI (20). It has been demonstrated that CKD patients are more sensitive to contrast agents due to their reduced kidney function (20, 21). In line with these findings, our study showed that the presence of CKD increases the likelihood of developing CI-AKI. In patients with CKD, impaired renal autoregulation and increased susceptibility to hypoperfusion may explain the strong association between low admission MAP and the development of CI-AKI.

CKD and hyperlipidemia were more frequent in patients who developed CI-AKI, which is consistent with previous studies (22). Hyperlipidemia increases the toxicity of contrast media by causing endothelial dysfunction. However, these conditions are closely related to hemodynamic instability, vascular stiffness, and impaired autoregulation, and the observed associations may reflect a shared vulnerability rather than independent causal pathways. Our study shows that patients with hyperlipidemia are more likely to develop CI-AKI, which is consistent with existing literature.

In patients with low MAP values, providing adequate hydration before the procedure, avoiding nephrotoxic agents, and minimizing contrast volume may be relevant for identifying hemodynamically fragile NSTEMI patients who require closer monitoring. However, this study was unable to fully evaluate the effects of potential confounding factors such as contrast volume, periprocedural hydration protocols, and the use of nephrotoxic drugs, which should be considered when interpreting the results.

Limitations: This study has several limitations that should be acknowledged. Firstly, the single-center, retrospective design limits the ability to establish a causal relationship and may restrict the generalizability of the findings. Secondly, the relatively small sample size may have reduced the statistical power, particularly for subgroup analyses. Thirdly, MAP was evaluated only at the time of admission, and dynamic hemodynamic changes during hospitalization were not assessed. Therefore, the potential impact of blood pressure variability on the development of contrast-associated acute kidney injury could not be evaluated. Additionally, procedural factors such as contrast volume, contrast type, and variations in periprocedural hydration protocols were not analyzed in detail. Detailed data regarding vasopressors, antihypertensive agents, and other hemodynamically active medications were not available; therefore, these variables could not be included in the analysis. Finally, some potential confounding factors that may influence the development of contrast-associated acute kidney injury, such as inflammatory markers and the dose or duration of nephrotoxic medications, could not be evaluated. As a result, residual confounding related to procedural and periprocedural characteristics cannot be excluded. These findings should be interpreted with caution and confirmed in larger, multicenter, prospective studies.

CONCLUSION

Our study demonstrated that low admission MAP is significantly associated with the development of contrast-associated acute kidney injury in patients with NSTEMI undergoing PCI. Given that MAP can be assessed rapidly and noninvasively, it may serve as a practical parameter for early risk stratification, particularly in high-risk patients. In addition, admission MAP may complement existing CI-AKI risk assessment tools by providing rapid bedside hemodynamic information and increasing clinical awareness regarding patients who may require closer monitoring and preventive care. However, given the observational design, these findings should be interpreted as associations rather than causal relationships.

Ethics Committee Approval: This study was approved by the Non-Interventional Research Ethics Committee of Fırat University(Session No: 2025/18-35, Date: December 11, 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki.

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Informed Consent: It was not required as the study was conducted retrospectively.

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