

**Relationship Between Killip Class and Acute Kidney Injury in Acute Myocardial Infarction: A Prospective Observational Study****Shani Patel<sup>1</sup>, Pramod Kumar Agrawal<sup>2</sup>, Taskeen Ahmad Reza<sup>3</sup>, Zooneyed Akhtar<sup>4</sup>, Rishav Kumar<sup>5</sup>**<sup>1</sup>3<sup>rd</sup> Year PG Resident, Department of General Medicine, Katihar Medical College, Bihar, India<sup>2</sup>HOD & Professor, Department of General Medicine, Katihar Medical College, Bihar, India<sup>3</sup>Professor, Department of General Medicine, Katihar Medical College, Bihar, India<sup>4</sup>Associate Professor, Department of General Medicine, Katihar Medical College, Bihar, India<sup>5</sup>3<sup>rd</sup> Year, PG Resident, Department of General Medicine, Katihar Medical College, Bihar, India

Received: 26-05-2026 / Revised: 25-06-2026 / Accepted: 27-07-2026

Corresponding Author: Dr. Shani Patel

Conflict of interest: Nil

**Abstract:****Background:** Acute kidney injury (AKI) is an important complication of acute myocardial infarction (AMI).

Hemodynamic deterioration, reduced cardiac output, systemic hypoperfusion, venous congestion, inflammation, and exposure to iodinated contrast media may contribute to renal dysfunction. Killip classification provides a rapid bedside assessment of heart failure severity in AMI and may therefore be related to the development of AKI.

**Objectives:** To evaluate the relationship between Killip class at presentation and development of AKI among patients with acute myocardial infarction (AMI), including STEMI and NSTEMI, and to describe associated clinical and renal characteristics.**Materials and Methods:** This hospital-based prospective observational study included 80 adult patients with confirmed AMI admitted to the Department of General Medicine, Katihar Medical College and Hospital, Katihar, Bihar, between March 2024 and August 2025. Both STEMI and NSTEMI were included. Patients with pre-existing chronic kidney disease or structural renal disease were excluded. AKI was defined according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Serum creatinine was recorded at admission and reassessed at approximately 48 hours, with urine output assessed where documented. Killip class was documented at presentation. Associations were evaluated using the Pearson chi-square test, with  $p < 0.05$  considered statistically significant.**Results:** Of 80 patients, 44 developed AKI, giving an incidence of 55.0%. The mean age of the overall population was 58.46 years, and 46 (57.5%) were male. Killip Class I was present in 40 patients, Class II in 16, Class III in 9, and Class IV in 15. AKI occurred in 25/40 (62.5%) patients in Class I, 6/16 (37.5%) in Class II, 6/9 (66.7%) in Class III, and 7/15 (46.7%) in Class IV. Recalculation from the study contingency table demonstrated no statistically significant overall association between Killip class and AKI ( $\chi^2 = 3.805$ ,  $df = 3$ ,  $p = 0.283$ ). Thus, the  $\chi^2 = 44.000$  and  $p < 0.001$  reported elsewhere in the study are not mathematically compatible with the displayed Killip-by-AKI counts.**Conclusion:** AKI occurred in more than half of patients with AMI in this cohort. Although cardiac dysfunction remains biologically relevant to renal injury, the observed Killip-class distribution did not demonstrate a statistically significant relationship with AKI. Larger studies with adequate representation across Killip classes are required.**Keywords:** Acute Kidney Injury; Acute Myocardial Infarction; Killip Class; STEMI; NSTEMI; Cardiorenal Syndrome.**DOI:** 10.25258/ijcpr.18.7.261This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Acute coronary syndrome encompasses acute myocardial ischemic conditions ranging from unstable angina to myocardial infarction. In the present study, the enrolled population consisted specifically of patients with STEMI and NSTEMI. Acute myocardial infarction remains a major cause of morbidity and mortality and may be complicated

by heart failure, cardiogenic shock, arrhythmias, and multiorgan dysfunction. [1,2]

Acute kidney injury is a particularly important complication following myocardial infarction. Its pathogenesis is multifactorial and includes reduced renal perfusion secondary to diminished cardiac

output, systemic hypotension, neurohormonal activation, venous congestion, inflammation, oxidative stress, and exposure to iodinated contrast media. [3-8] Even relatively small increases in serum creatinine after myocardial infarction have been associated with adverse short- and long-term outcomes. [3-5]

The Killip classification is a simple clinical system used to categorize the severity of heart failure following myocardial infarction. Class I denotes absence of clinical heart failure, Class II indicates mild-to-moderate heart failure, Class III represents pulmonary edema, and Class IV represents cardiogenic shock. Because increasing hemodynamic compromise may reduce renal perfusion, higher Killip class would theoretically be expected to increase the likelihood of AKI. Previous studies have identified cardiogenic shock, reduced left ventricular ejection fraction, impaired baseline renal function, and severe cardiac dysfunction as important predictors of AKI. [3,5-7]

The present study evaluated the incidence of AKI among patients with AMI and examined whether Killip class at presentation was associated with the development of AKI. Both STEMI and NSTEMI patients were included, while patients with established chronic kidney disease or structural renal disease were excluded.

**Aim:** To evaluate the relationship between Killip class and acute kidney injury among patients with acute myocardial infarction (AMI).

### Objectives

The primary objective was to compare the incidence of AKI across Killip Classes I–IV. The secondary objectives were:

1. To determine the overall incidence of AKI among patients with AMI.
2. To describe renal-function parameters and major clinical characteristics.
3. To evaluate the relationship between Killip class and development of AKI.
4. To assess short-term in-hospital mortality among patients with and without AKI.

### Materials and Methods

**Study Design and Setting:** This was a hospital-based prospective observational study conducted in the Department of General Medicine, Katihar Medical College and Hospital, Katihar, Bihar, India. Patients were recruited from the Emergency Department, Intensive Care Unit, Medicine wards, and inpatient services of the Department of General Medicine.

**Study Duration:** The study was conducted over 18 months, from March 2024 to August 2025.

**Study Population:** Adult patients diagnosed with acute myocardial infarction, including STEMI and NSTEMI, were prospectively enrolled. Diagnosis was established based on the clinical presentation, 12-lead electrocardiography, and cardiac biomarkers, including troponin-I and creatine kinase–myocardial band, according to the diagnostic approach described in the study.

**Sample Size:** A total of 80 patients were included in the study.

**Sample Size Calculation:** The required sample size was estimated using a previously reported incidence of approximately 25.1% for AKI following AMI. Using the formula  $n = Z^2 pq / d^2$ , where  $Z = 1.96$  for a 95% confidence level,  $p = 0.251$ ,  $q = 0.749$ , and absolute precision ( $d$ ) = 0.10, the calculated minimum sample size was approximately 72.2 participants. Allowing approximately 10% for incomplete observations or attrition resulted in an estimated sample size of approximately 79.4 participants, which was rounded to 80 participants. Thus, 80 patients were included in the present study.

**Inclusion Criteria:** Patients were included if they fulfilled all of the following criteria:

1. Age >18 years.
2. Patients of either sex.
3. Admission with acute myocardial infarction.
4. Diagnosis of either STEMI or NSTEMI.
5. Admission to the Emergency Department, ICU, or Medicine wards.
6. Provision of written informed consent.

### Exclusion Criteria

Patients were excluded if they had:

1. Age <18 years.
2. Pre-existing chronic kidney disease.
3. Structural kidney disease.
4. Refusal to provide consent.
5. Incomplete clinical or laboratory records.

**Clinical Assessment:** Detailed demographic and clinical information was recorded, including age, sex, duration of symptoms, diabetes mellitus, hypertension, clinical evidence of heart failure, and cardiogenic shock.

All patients underwent physical examination at presentation and were assigned a Killip class based on clinical findings.

Killip Class I was defined as the absence of clinical evidence of heart failure. Class II represented mild-to-moderate heart failure, Class III represented pulmonary edema, and Class IV represented cardiogenic shock.

Left ventricular function was evaluated using two-dimensional echocardiography and reported as left ventricular ejection fraction (LVEF, %).

**Assessment of Renal Function:** Serum creatinine was measured in mg/dL at admission and again at approximately 48 hours. Blood urea was recorded in mg/dL, while estimated glomerular filtration rate (eGFR) was expressed in mL/min/1.73 m<sup>2</sup>.

**Definition of Acute Kidney Injury:** AKI was defined according to KDIGO criteria as an increase in serum creatinine by  $\geq 0.3$  mg/dL within 48 hours, or an increase in serum creatinine to  $\geq 1.5$  times baseline, which is known or presumed to have occurred within the prior 7 days, or urine volume  $< 0.5$  mL/kg/h for 6 hours. The diagnosis of AKI was therefore based on the available serum creatinine and urine-output data during hospitalization.

**Outcome Measures:** The primary outcome was the development of AKI during hospitalization. The principal exposure variable was Killip class at presentation.

Secondary outcomes included:

- Renal-function characteristics
- Cardiac dysfunction
- LVEF

- Five-day in-hospital mortality
- Other clinically documented complications

**Statistical Analysis:** Categorical variables were expressed as frequencies and percentages. Continuous variables available from the study were summarized as mean values with appropriate units. The relationship between Killip class and AKI was evaluated using the Pearson chi-square test based on the reported 4×2 contingency table. A two-sided p value  $< 0.05$  was considered statistically significant. The Killip-class counts were independently recalculated from the displayed contingency table rather than reproducing the inconsistent summary statistic reported elsewhere in the source material. The recalculated Pearson chi-square statistic was  $\chi^2 = 3.805$  with 3 degrees of freedom and  $p = 0.283$ .

**Ethical Considerations:** The study was initiated after approval from the Institutional Ethics Committee of Katihar Medical College and Hospital. Written informed consent was obtained from all participants.

## Result

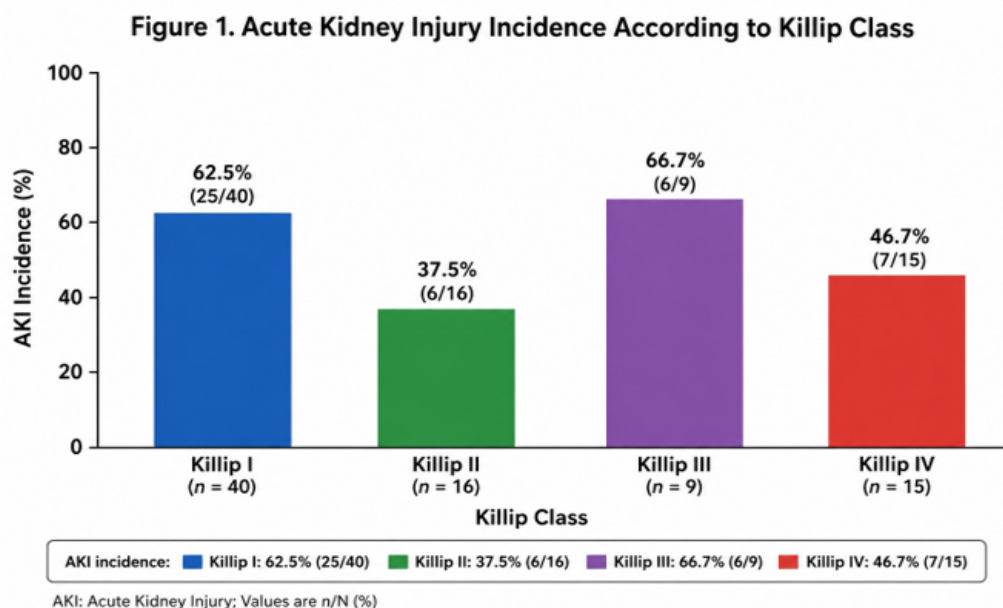


Figure 1. Acute Kidney Injury Incidence According to Killip Class. Bar graph showing the incidence of AKI across Killip Classes I–IV. AKI occurred in

25/40 (62.5%) patients with Class I, 6/16 (37.5%) with Class II, 6/9 (66.7%) with Class III, and 7/15 (46.7%) with Class IV.



Figure 2. Distribution of Killip Class According to Acute Kidney Injury Status. Grouped bar graph showing the distribution of Killip Classes I–IV among patients who developed AKI (n=44) and those who did not develop AKI (n=36). The corresponding frequencies were 25, 6, 6, and 7 in the AKI group and 15, 10, 3, and 8 in the non-AKI group, respectively. Pearson  $\chi^2=3.805$ , df=3, p=0.283, indicating no statistically significant association between Killip class and AKI.

**Baseline Characteristics:** A total of 80 patients with confirmed AMI were included in the study. Of

these, 44 developed AKI, corresponding to an overall AKI incidence of 55.0%, while 36 (45.0%) did not develop AKI.

The mean age of the study population was 58.46 years. There were 46 males (57.5%) and 34 females (42.5%).

Diabetes mellitus was present in 22/44 (50.0%) patients in the AKI group and 16/36 (44.4%) patients in the non-AKI group. Hypertension was present in 19/44 (43.2%) and 16/36 (44.4%), respectively.

**Table 1: Selected Characteristics of the Study Population**

| Variable                      | Finding       |
|-------------------------------|---------------|
| Total participants            | 80            |
| Mean age                      | 58.46 years   |
| Male                          | 46 (57.5%)    |
| Female                        | 34 (42.5%)    |
| AKI developed                 | 44 (55.0%)    |
| AKI not developed             | 36 (45.0%)    |
| Diabetes in AKI group         | 22/44 (50.0%) |
| Diabetes in non-AKI group     | 16/36 (44.4%) |
| Hypertension in AKI group     | 19/44 (43.2%) |
| Hypertension in non-AKI group | 16/36 (44.4%) |

**Renal Function Parameters:** The mean admission serum creatinine was 1.132 mg/dL in the AKI group and 1.254 mg/dL in the non-AKI group. At approximately 48 hours, the corresponding mean serum creatinine values were 1.481 mg/dL and 1.716 mg/dL, respectively.

Mean blood urea was 57.59 mg/dL in the AKI group and 61.28 mg/dL in the non-AKI group, while mean eGFR was 67.82 and 58.22 mL/min/1.73 m<sup>2</sup>, respectively.

These reported values have been retained from the source study. However, the higher 48-hour mean serum creatinine in the group labelled “non-AKI” should be verified against the original patient-level data before journal submission because AKI classification is based on change from baseline rather than the absolute creatinine concentration alone.

**Table 2: Renal Function Parameters According to AKI Status**

| Parameter                            | AKI group (n=44) | Non-AKI group (n=36) |
|--------------------------------------|------------------|----------------------|
| Serum creatinine at admission, mg/dL | 1.132            | 1.254                |
| Serum creatinine at 48 h, mg/dL      | 1.481            | 1.716                |
| Blood urea, mg/dL                    | 57.59            | 61.28                |
| eGFR, mL/min/1.73 m <sup>2</sup>     | 67.82            | 58.22                |

**Relationship Between Killip Class and Acute Kidney Injury:** Among the 44 patients who developed AKI, 25 were Killip Class I, 6 were Class II, 6 were Class III, and 7 were Class IV.

Among the 36 patients who did not develop AKI, 15 were Killip Class I, 10 were Class II, 3 were Class III, and 8 were Class IV.

**Table 3: Relationship Between Killip Class and Development of AKI**

| Killip class | AKI developed n (%) | AKI not developed n (%) | Total     | AKI incidence within class |
|--------------|---------------------|-------------------------|-----------|----------------------------|
| I            | 25 (56.8)           | 15 (41.7)               | 40        | 62.5%                      |
| II           | 6 (13.6)            | 10 (27.8)               | 16        | 37.5%                      |
| III          | 6 (13.6)            | 3 (8.3)                 | 9         | 66.7%                      |
| IV           | 7 (15.9)            | 8 (22.2)                | 15        | 46.7%                      |
| <b>Total</b> | <b>44 (100)</b>     | <b>36 (100)</b>         | <b>80</b> | <b>55.0%</b>               |

Pearson  $\chi^2=3.805$ ; df=3; p=0.283.

There was no statistically significant relationship between Killip class and development of AKI in this cohort. The pattern was not monotonic. AKI incidence was 62.5% in Class I, 37.5% in Class II, 66.7% in Class III, and 46.7% in Class IV. Therefore, the available data did not demonstrate a progressive increase in AKI incidence with worsening Killip class.

**Short-Term Mortality:** Nine deaths occurred among the 44 patients with AKI and six deaths occurred among the 36 patients without AKI. Thus, five-day mortality was 20.5% in the AKI group compared with 16.7% in the non-AKI group. The difference was modest and should not be interpreted as statistically significant without an appropriate statistical test.

## Discussion

The present prospective observational study evaluated the relationship between Killip class and AKI in 80 patients with AMI. AKI developed in 44 patients, yielding an overall incidence of 55.0%.

The incidence of AKI observed in this study was substantial. Previous investigations have demonstrated that AKI is a clinically important complication of AMI, although reported incidence varies according to patient characteristics, baseline renal function, diagnostic criteria, timing of creatinine assessment, hemodynamic status, and treatment-related factors. [3-5,13-15]

The principal finding of the present study was that Killip class was not significantly associated with AKI after correct reanalysis of the reported contingency table. The Pearson chi-square statistic was 3.805 with 3 degrees of freedom and a p value of 0.283.

From a pathophysiological perspective, an association between severe cardiac dysfunction and AKI is plausible. Cardiogenic shock and acute heart failure may reduce renal perfusion pressure, increase venous congestion, activate the sympathetic nervous system and renin-angiotensin-aldosterone system, and contribute to renal ischemia. [3,5-7] Reinstadler et al. reported an association between AKI following myocardial infarction and myocardial microvascular injury. [3] Other studies have also identified severe cardiac dysfunction and hemodynamic compromise as important contributors to renal injury following AMI. [5-7]

Nevertheless, a biologically plausible relationship does not necessarily result in a statistically significant association in every cohort. In the present sample, Killip Class III had the highest within-class AKI incidence at 66.7%, whereas Class IV had an incidence of 46.7%. Interestingly, Class I also demonstrated a relatively high AKI incidence of 62.5%.

This irregular distribution prevented demonstration of a dose-response relationship between increasing Killip class and AKI.

Several factors may explain this finding. First, the overall sample size was relatively small, with only nine patients in Killip Class III and 15 in Class IV. The small numbers within individual Killip categories reduce statistical power to identify differences between groups.

Second, AKI following AMI is multifactorial. Factors such as age, diabetes mellitus, baseline renal reserve, volume status, contrast exposure, nephrotoxic medications, inflammation, reperfusion injury, and hemodynamic instability may all contribute to renal dysfunction. [4-8]

Third, Killip class represents the clinical cardiovascular status at a specific point in time and may change during hospitalization. Consequently, a single Killip-class assessment may not completely capture subsequent hemodynamic changes.

The overall AKI incidence of 55.0% in the present study was clinically important. Previous studies have consistently demonstrated that AKI following AMI is associated with adverse short-and long-term outcomes. [4,5,13-15] Skalsky et al. reported that recovery from AKI following AMI has important implications for long-term prognosis, while Kosaki et al. demonstrated the prognostic importance of the timing of AKI onset. [13,14] Chalikias et al. similarly reported an adverse long-term impact of AKI following myocardial infarction. [15]

The study recorded five-day mortality in 9 of 44 patients with AKI compared with 6 of 36 patients without AKI. Although mortality was numerically higher among patients with AKI, the difference was modest and the available sample was insufficient to establish a statistically significant association.

The renal biochemical findings warrant careful interpretation. The reported mean serum creatinine was higher in the non-AKI group at both admission and 48 hours. Because AKI classification is based on a rise from baseline and/or reduced urine output rather than solely on an absolute serum creatinine value, this pattern is theoretically possible. Nevertheless, the reported values and group labels should be verified against the original dataset before submission to ensure that no data-entry or classification error is present.

The present findings also emphasize that renal monitoring should not be restricted to patients with advanced heart failure. The relatively high AKI incidence observed even among Killip Class I patients indicates that patients without overt clinical heart failure may also develop AKI after AMI.

**Strengths:** The prospective observational design allowed serial assessment of renal function following AMI.

The study included both STEMI and NSTEMI patients encountered in routine tertiary-care practice.

AKI was defined using recognized KDIGO criteria, incorporating both serum creatinine and urine-output criteria.

The principal statistical analysis was recalculated directly from the reported Killip-by-AKI contingency table rather than reproducing an internally inconsistent summary statistic.

**Limitations:** The study was conducted at a single tertiary-care center and included only 80 participants. The relatively small numbers in Killip

Classes II–IV limited statistical power and precision.

The study was conducted exclusively in patients with acute myocardial infarction (STEMI and NSTEMI); therefore, the findings should not be generalized to the entire spectrum of acute coronary syndrome, including unstable angina.

Other potential determinants of AKI, including baseline renal reserve, contrast volume, detailed hemodynamic parameters, nephrotoxic drug exposure, and changes in Killip class during hospitalization, were not comprehensively evaluated.

The renal-function data also require verification against the original patient-level dataset, particularly the reported 48-hour serum creatinine values in the AKI and non-AKI groups.

### Conclusion

Acute kidney injury occurred in 44 of 80 patients (55.0%) with acute myocardial infarction in this prospective observational study at Katihar Medical College and Hospital.

Based on the reported Killip-by-AKI contingency table, Killip class was not significantly associated with the development of AKI ( $\chi^2=3.805$ ,  $df=3$ ,  $p=0.283$ ).

AKI incidence was 62.5%, 37.5%, 66.7%, and 46.7% in Killip Classes I, II, III, and IV, respectively, demonstrating no consistent increase in AKI with worsening Killip class.

Therefore, the study does not support Killip class as an independent or progressively increasing predictor of AKI based on the available data. Renal injury after AMI is likely multifactorial, and larger adequately powered studies incorporating baseline renal function, LVEF, hemodynamic variables, contrast exposure, and treatment characteristics are required.

**Clinical Implications:** Killip classification remains valuable for bedside assessment of hemodynamic severity after AMI, but it should not be used alone to determine AKI risk.

Renal-function monitoring should be performed across all Killip classes, including patients classified as Killip Class I.

Serial serum creatinine measurement and monitoring of urine output, optimization of intravascular volume and hemodynamics, avoidance of unnecessary nephrotoxic medications, and careful use of contrast media remain important components of AKI prevention and management in patients with AMI.

**Declarations Ethics Approval:** Institutional Ethics Committee approval was obtained before commencement of the study.

**Consent to Participate:** Written informed consent was obtained from all participants.

**Conflict of Interest:** The authors declare no conflict of interest.

**Funding:** No external funding is reported in the available material.

**Data Availability:** Data may be made available in accordance with institutional policy and ethical requirements.

### Abbreviations

AKI – Acute Kidney Injury

AMI – Acute Myocardial Infarction

CK-MB – Creatine Kinase–Myocardial Band

ECG – Electrocardiogram

eGFR – Estimated Glomerular Filtration Rate

ICU – Intensive Care Unit

KDIGO – Kidney Disease: Improving Global Outcomes

LVEF – Left Ventricular Ejection Fraction NSTEMI – Non-ST-Elevation Myocardial Infarction PCI – Percutaneous Coronary Intervention

STEMI – ST-Elevation Myocardial Infarction

### References

1. Koester M, Dangl M, Albosta M, Grant J, Maning J, Colombo R. US trends of in-hospital morbidity and mortality for acute myocardial infarctions complicated by cardiogenic shock. *Cardiovasc Revasc Med.* 2024;64:44-51. doi:10.1016/j.carrev.2024.02.007.
2. Salari N, Morddarvanjoghi F, Abdolmaleki A, Rasoulpoor S, Khaleghi AA, Hezarkhani LA, et al. The global prevalence of myocardial infarction: a systematic review and meta-analysis. *BMC Cardiovasc Disord.* 2023;23:206. doi:10.1186/s12872-023-03231-w.
3. Reinstadler SJ, Kronbichler A, Reindl M, Feistritzer HJ, Innerhofer V, Mayr A, et al. Acute kidney injury is associated with microvascular myocardial damage following myocardial infarction. *Kidney Int.* 2017;92(3):743-750. doi:10.1016/j.kint.2017.02.016.
4. Skalsky K, Shiyovich A, Shechter A, Gilutz H, Plakht Y. Recovery from acute kidney injury and long-term prognosis following acute myocardial infarction. *Biomedicines.* 2024;12(7). doi:10.3390/biomedicines12071490.
5. Guo P, Tao F, Du L, Yang H, Wang W, Ma C, et al. Acute kidney injury after myocardial infarction: prognostic implications via dual robust methods. *Front Med.* 2025;12. doi:10.3389/fmed.2025.1555478.
6. Rigattieri S, Cristiano E, Tempestini F, Pittorino L, Cesario V, Casenghi M, et al. Acute kidney injury in patients with acute myocardial infarction undergoing percutaneous coronary intervention: the role of vascular access site. *J Clin Med.* 2024;13(8):2367. doi:10.3390/jcm13082367.
7. Zhang XY, Fan ZG, Xu HM, Xu K, Tian NL, Zhang XY, et al. Clinical outcomes for acute kidney injury in acute myocardial infarction patients after intra-aortic balloon pump implantation: a single-center observational study. *Rev Cardiovasc Med.* 2023;24(6). doi:10.31083/j.rcm2406172.
8. Huang X, Bai S, Luo Y. Advances in research on biomarkers associated with acute myocardial infarction: a review. *Medicine (Baltimore).* 2024;103(15):e37793. doi:10.1097/MD.00000000000037793.
9. Aldous SJ. Cardiac biomarkers in acute myocardial infarction. *Int J Cardiol.* 2013;164(3):282-294. doi:10.1016/j.ijcard.2012.01.081.
10. Wang XY, Zhang F, Zhang C, Zheng LR, Yang J. The biomarkers for acute myocardial infarction and heart failure. *Biomed Res Int.* 2020;2020:2018035. doi:10.1155/2020/2018035.
11. Zhang X, Zhang H, Liu X, Yu C. Serum angiotensinogen as a biomarker for renal outcomes in patients with acute myocardial infarction: prognostic significance and clinical implications. *BMC Nephrol.* 2025;26(1):651. doi:10.1186/s12882-025-04573-7.
12. Yadav A, Jha S, Pokhrel S, Koirala M, Sah R, Mishra A, et al. Incidence and risk factors of acute kidney injury after myocardial infarction; a prospective cohort study with impact of timing of presentation on other risk factors. *BMC Nephrol.* 2026;27(1):150. doi:10.1186/s12882-026-04745-z.
13. Kosaki R, Wakabayashi K, Sato S, Tanaka H, Ogura K, Oishi Y, et al. Onset time and prognostic value of acute kidney injury in patients with acute myocardial infarction. *Int J Cardiol Heart Vasc.* 2021;35:100826. doi:10.1016/j.ijcha.2021.100826.
14. Chalikias G, Serif L, Kikas P, Thomaidis A, Stakos D, Makrygiannis D, et al. Long-term impact of acute kidney injury on prognosis in patients with acute myocardial infarction. *Int J Cardiol.* 2019;283:48-54. doi:10.1016/j.ijcard.2019.01.070.
15. Rodrigues FB, Bruetto RG, Torres US, Otaviano AP, Zanetta DMT, Burdmann EA.

Incidence and mortality of acute kidney injury after myocardial infarction: a comparison between KDIGO and RIFLE criteria.

PLoS One. 2013;8(7):e69998.  
doi:10.1371/journal.pone.0069998.