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RESEARCH ARTICLE

FREQUENCY AND OUTCOMES OF ACUTE KIDNEY INJURY IN PATIENTS PRESENTING WITH ACUTE HEART FAILURE AT A CARDIAC CENTRE IN KARACHI

Cheena Kumari, Khursheed Hassan, Naimat Ullah Junejo, Abdul Sami, Awais Shafi, Muhammad Amin

Cardiology Department, Tabba Heart Institute, Karachi, Pakistan

ABSTRACT

Objectives: *To determine the frequency of acute kidney injury (AKI) in patients with acute heart failure (AHF) and evaluate its association with in-hospital mortality.*

Patients and Methods: *This cross-sectional study was conducted over six months at Tabba Heart Institute, Karachi. A total of 121 consecutive patients aged 40–80 years admitted with AHF were enrolled. Patients with acute coronary syndromes, sepsis, arrhythmias, or major non-cardiac comorbidities were excluded. AKI was defined as a ⩾ 0.3 mg/dL increase in serum creatinine within 48 hours of admission. Demographic, clinical, and socioeconomic variables were recorded. Primary outcomes were the frequency of AKI and in-hospital mortality.*

Results: *Nineteen patients (15.7%) developed AKI. Patients with AKI had a higher prevalence of urban residence (100% vs 80.4%, p=0.035), dyslipidemia (73.7% vs 44.1%, p=0.018), and reported lower family income (73.7% vs 34.3%, p=0.001) compared to non-AKI patients. In-hospital mortality was higher in the AKI group (42.1% vs 20.6%, p=0.044). Univariate logistic regression showed an odds ratio (OR) for mortality with AKI of 3.05 (95% CI 1.03–9.05, p=0.044). After adjustment, the adjusted OR was 3.21 (95% CI 0.73–14.18, p=0.124)*

Conclusion: *AKI occurred in approximately one-sixth of patients hospitalized with AHF and was associated with higher unadjusted in-hospital mortality. Socioeconomic factors, particularly low income and urban residence, were linked to AKI risk, underscoring the need for targeted preventive strategies in high-risk subgroups.*

Keywords: *Acute heart failure, Acute kidney injury, In-hospital mortality, Pakistan, Socioeconomic status.*

INTRODUCTION

Heart failure (HF) is a rapidly growing public health issue, with rising prevalence driven by an aging population and improved post–myocardial infarction and HF survival.[1] This burden is intensified by the bidirectional relationship between cardiac and renal dysfunction, in which acute or chronic impairment in one organ precipitates dysfunction in the other, a phenomenon known as cardiorenal syndrome.[2,3] AKI, in particular, is a critical event in this interplay, as it can accelerate progression to end-stage renal disease and is a powerful predictor of poor outcomes.[4]

Correspondence:
Dr. Cheena Kumari
Cardiology Department
Tabba Heart Institute, Karachi, Pakistan
Email: cheenakarwa2017@gmail.com
Received: 19 Aug 2025; revision received: 8 Dec 2025; accepted: 26 Jan 2026
DOI: https://doi.org/10.33897/fumj.v8i1.247

How to Cite: Kumari C, Hassan K, Junejo NU, Sami A, Shafi A, Amin M. Frequency and outcomes of acute kidney injury in patients presenting with acute heart failure at a cardiac centre in Karachi. Foundation University Medical Journal. 2026; 8(1): 16-22

The reported incidence of AKI in AHF varies widely, from 9% to 43%, with consistent evidence linking it to higher in-hospital and one-year mortality rates.[5-7] For AHF cases with AKI, hospital death stands at about 10.8%, with extended stays and higher expenses.[8] Studies from Spain noted 13% AKI in AHF inpatients, while in a Pakistani study, Bhatti et al. observed an in-hospital mortality rate of 7.4% in HF patients who developed AKI.[9,10]

Despite growing evidence on worsening renal function in AHF, the precise frequency of acute AKI and its impact on in-hospital mortality remain underexplored in local settings. Understanding the local burden of AKI in acute heart failure is important because it may complicate fluid management, worsen hemodynamic instability, and limit treatment options, thereby worsening patient outcomes.[11] This study aims to determine the frequency of AKI in AHF and assess its association with in-hospital mortality, with the goal of informing targeted prevention strategies, optimizing care quality, and guiding resource allocation for high-risk patients.

PATIENTS AND METHODS

This cross-sectional study was conducted at the Coronary Care Unit of Tabba Heart Institute, Karachi, over a six-month period from 1st March 2025 to 1st August 2025, following approval from the Institutional Review Board of Tabba Heart Institute (OHRP Reg. No. IORG000786; Approval No. THI/IRB/SQ/12-03-2025/119). The required sample size was calculated to be 121 patients using the WHO sample size calculator, assuming a 13% prevalence of AKI among patients with AHF, a 6% margin of error, and a 95% confidence level. Participants were recruited using a non-probability consecutive sampling technique. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki, and written informed consent was obtained from all participants prior to data collection.[12]

Inclusion and exclusion criteria

Patients aged 40–80 years, of either gender, presenting with new-onset or acutely decompensated heart failure requiring hospitalization were eligible. We did not restrict enrollment based on the etiology of cardiomyopathy. Patients with stable or chronic coronary artery disease presenting with acute heart failure not triggered by an acute ischemic event were included.

Patients were excluded if they presented with acute coronary syndromes [ST-Elevation Myocardial Infarction (STEMI), non-ST-Elevation Myocardial Infarction (NSTEMI)], or unstable angina). Additional exclusion criteria were: sepsis, diarrhea, pulmonary hypertension, pericardial disease, significant arrhythmias (bradyarrhythmias or tachyarrhythmias), stroke, asthma, and chronic obstructive pulmonary disease.

Data collection

Data were extracted from the hospital's electronic health records. All patients were informed about the risk, benefit and purpose of the study prior to taking written informed consent. Demographic information (age, gender, residence, education level, occupation, family income), comorbidities (diabetes mellitus type II, hypertension, dyslipidemia), smoking status, laboratory findings, and hospital course were recorded. Serum creatinine and urine output were noted, and AKI was defined according to KDIGO criteria as an increase in serum creatinine of $\geqslant$ 0.3 mg/dL within 48 hours of admission or from the most recent available baseline.[13] The primary outcome was in-hospital mortality.

Data analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 27. Age was categorized into two groups (40–60 years and 61–80 years). All variables including age group, gender, residence, comorbidities, smoking status, socioeconomic indicators, presence of AKI, and in-hospital mortality were summarized as frequencies and percentages.

Group comparisons between AKI and non-AKI patients were performed using the chi-square test or Fisher's exact test. Variables with $p \leqslant 0.05$ in univariate analysis were entered into logistic regression to assess the association between AKI and in-hospital mortality, with results expressed as odds ratios (ORs) and 95% confidence intervals (CIs). Multivariate regression was not performed due to quasi-complete data separation. A two-tailed *p*-value of $\leqslant 0.05$ was considered statistically significant.

RESULTS

A total of 121 patients with AHF were included in the analysis. Most were aged 61–80 years (68.6%) and male (50.4%). The majority resided in urban areas (83.5%). The most common comorbidities were dyslipidemia (48.8%), diabetes mellitus type II (27.3%), hypertension (26.4%), and current smoking (37.2%). AKI occurred in 19 patients (15.7%). The in-hospital mortality rate was 24.0% (Table I).

Comparison between AKI and non-AKI groups

Patients with AKI were more likely to reside in urban areas compared to those without AKI (100% vs. 80.4%; p = 0.035). Dyslipidemia was more frequent in the AKI group than in the non-AKI group (73.7% vs. 44.1%; p = 0.018). A lower family income (< PKR 75,000) was observed more commonly in patients with AKI (73.7% vs. 34.3%; p = 0.001). Interestingly, none of the patients in the AKI group had diabetes mellitus or hypertension. This unexpected finding may reflect the small sample size, selection pattern, or chance rather than a true absence of association. Compared to 31.4% and 28.4% in the non-AKI group, respectively (both p = 0.004). In-hospital mortality was higher in the AKI group (42.1% vs. 20.6%; p = 0.044) (Table II).

Association between AKI and in-hospital mortality

In univariate logistic regression, AKI was associated with in-hospital mortality (OR 3.05; 95% CI 1.03–9.05; p = 0.044). After adjusting for age, sex, comorbidities,

and socioeconomic variables, the association was not statistically significant (adjusted OR 3.21; 95% CI 0.73–14.18; p = 0.124) (Table III). Multivariate logistic regression could not be performed as the model did not converge due to quasi-complete separation of data; therefore, only univariate results are presented. Quasi-complete separation occurs when a predictor variable perfectly (or almost perfectly) predicts the outcome, resulting in estimation instability and preventing the model from generating reliable coefficients.

DISCUSSION

In our study, AKI developed in 15.7% of patients hospitalized with AHF, a frequency comparable to previous reports (13–27%).[14,15] AKI was associated with higher in-hospital mortality in our cohort (42.1% vs 20.6%), underscoring the prognostic weight of renal dysfunction in this setting. Interestingly, the mortality observed in our AKI subgroup was substantially higher than that described in both regional and international cohorts. For example, a recent Pakistani study reported an in-hospital mortality of 7.4% among AHF patients with AKI,[10] suggesting that population differences, variations in disease severity at presentation, or disparities in clinical management may partly explain this discrepancy. It may also reflect differences in level of care across tertiary, secondary, private, and public facilities in Pakistan. The adverse prognostic impact of renal impairment is also supported by long-term data, where patients with worsening kidney function consistently experience higher mortality compared with those without impairment.[16] In our analysis, AKI tripled the odds of in-hospital death (OR 3.05, 95% CI 1.03–9.05); however, this association did not remain significant after adjustment, likely reflecting the relatively small number of AKI cases and the confounding influence of comorbidities and socioeconomic status. Nevertheless, the direction and magnitude of the effect support the clinical importance of vigilant renal monitoring and deliberate hemodynamic optimization in this high-risk group, particularly during aggressive decongestion.

One unexpected finding was the complete absence of diabetes mellitus and hypertension among patients who developed AKI, despite these comorbidities being widely recognized risk factors for renal dysfunction in heart failure.[17-18], In contrast, Mullens et al. reported that 44% of patients with worsening renal function had diabetes and 48% had hypertension, compared with 34% and 40%, respectively, among those without renal impairment, although these differences did not reach statistical significance.[17] Several factors may explain this discrepancy. First, the small number of AKI cases limits statistical inference. Second, our exclusion criteria created a complex selection pattern: removing younger adults (18–40 years) likely enriched the cohort for chronic comorbidities, whereas excluding Acute Coronary Syndrome (ACS) patients may have reduced the prevalence of such comorbidities among those presenting with acute hemodynamic compromise. Third, all patients in the AKI group were urban dwellers, whereas roughly one-fifth of non-AKI patients lived in non-urban areas, suggesting potential environmental or healthcare-access differences that merit further investigation. Finally, contextual factors, including heightened medical awareness and health-seeking behavior in our urban cohort, may have contributed to the observed pattern. In sum, institutional selection, sampling characteristics, and comorbidity prevalence likely influenced this unexpected finding.

Socioeconomic disadvantage also correlated with AKI in our cohort, as patients with lower monthly income (< PKR 75,000) were significantly more likely to had AKI or experienced AKI (73.7% vs 34.3%, p = 0.001). This finding is consistent with population-based data from the UK, where social deprivation was linked to higher AKI incidence (adjusted OR up to 1.79) and poorer survival after first AKI (adjusted HR 1.20).[19] These results suggest that socioeconomic inequities may not only increase AKI risk but also worsen outcomes, underscoring the importance of targeted preventive strategies in resource-limited settings.

We observed a higher prevalence of dyslipidemia among patients with AKI. Although direct evidence in AHF is limited, data from CKD populations provide relevant insights. Khandelwal et al. reported dyslipidemia in 73.8% of pediatric CKD patients, associated with endothelial dysfunction and vascular injury, while Malla et al. highlighted similar pro-atherogenic lipid changes as a common complication of CKD. These findings suggest that dyslipidemia-related vascular injury may also contribute to renal dysfunction in AHF.[20,21]

Our findings also have implications for the management of AHF. The clinical challenge lies in balancing the need for aggressive decongestion against the risk of renal hypoperfusion and electrolyte imbalance. In our setting, where late presentation and advanced disease at admission are common, this balance may be even harder

to achieve, potentially contributing to the observed AKI incidence. Importantly, emerging data suggest that targeted pharmacologic strategies may help break this vicious cycle. Sodium-Glucose Cotransporter-2 inhibitors, even when combined with Renin-Angiotensin-Aldosterone System(RAAS) blockers, have not been shown to increase AKI risk and may confer renal protection by improving hemodynamic stability.[22] Similarly, RAAS antagonism in sepsis-induced AKI has been associated with reduced renal injury and improved survival, while sacubitril/valsartan has demonstrated favorable renal and cardiovascular outcomes in CKD without compromising safety.[23-24] Combination approaches that address hemodynamic, metabolic, and inflammatory pathways are increasingly recognized as synergistic.[25] Yet, despite consistent benefits of Guideline-Directed Medical Therapy(GDMT) in HF patients with and without CKD, real-world data show that CKD patients remain undertreated, including in our local context where SGLT2 inhibitor use is limited.[26-27] Together, these findings highlight how AKI not only complicates therapy but also perpetuates suboptimal GDMT use, reinforcing a cycle of worse HF and CKD outcomes.

There are several limitations to our study. It was conducted at a single cardiac center, which limits generalizability to other settings, especially rural or less-resourced hospitals. The cross-sectional design prevented assessment of long-term outcomes, and the use of non-probability sampling may have introduced selection bias. AKI was defined solely by serum creatinine which may have underestimated its incidence, and although our sample size was calculated to capture the expected AKI prevalence adequately, the low event rate still precluded multivariate analysis and may have limited our ability to identify independent predictors with confidence. Moreover, our cohort included only hospitalized patients with AHF, which may have excluded individuals managed in the outpatient setting with milder disease, potentially limiting the applicability of our findings across the full spectrum of HF severity. Although acute coronary syndromes were excluded, thereby reducing the likelihood of patients undergoing coronary angiography, the potential contribution of contrast-induced nephropathy cannot be entirely ruled out. Finally, by excluding patients with overlapping acute conditions such as myocardial infarction and severe arrhythmias, our cohort may not fully represent the spectrum of real-world AHF presentations.

Our findings emphasize the need for early identification and prevention of AKI in AHF, particularly among socioeconomically disadvantaged patients, and highlight the importance of developing integrated, protocol-driven strategies that mitigate renal complications while optimizing heart failure therapy. In clinical practice, this approach includes optimizing volume status, closely monitoring renal function during HF treatment, and tailoring therapy to individual risk profiles to reduce mortality. Future multicenter studies with larger and more inclusive samples, broader AKI definitions, and longitudinal follow-up could further clarify the complex interplay between cardiac and renal dysfunction in acute settings.

CONCLUSION

In this study, AKI occurred in approximately one in six patients hospitalized with acute heart failure and was associated with significantly higher in-hospital mortality. AKI was more common among patients with dyslipidemia and lower socioeconomic status, highlighting the interplay between metabolic and social determinants of health in acute cardiac care.

ACKNOWLEDGMENTS

We express our gratitude to the staff of Tabba Heart Institute, Karachi, for their support in facilitating data collection and patient care during this study. We also thank the patients who participated in this research, whose cooperation made this study possible.

DISCLAIMER: None

Conflict Of Interest

The authors declare no conflicts of interest related to this study. No financial or personal relationships with other individuals or organizations have influenced the conduct or reporting of this research.

ETHICAL STATEMENT

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.[12] The study protocol was approved by the Institutional Review Board of Tabba Heart Institute (OHRP Reg. No. IORG000786; Approval No. THI/IRB/SQ/12-03-2025/119). Written informed consent was obtained from all participants prior to data collection.

Funding Disclosure: No external funding was received for this study

All authors approved the final version of the manuscript.

Table I: Baseline characteristics of the study cohort (N = 121)

Variable	n (%)
Age group	
40–60 years	38 (31.4)
61–80 years	83 (68.6)
Gender	
Male	61 (50.4)
Female	60 (49.6)
Residence	
Urban	101 (83.5)
Rural	20 (16.5)
Comorbidities	
Diabetes mellitus	33 (27.3)
Hypertension	32 (26.4)
Dyslipidemia	59 (48.8)
Smoking	45 (37.2)
Socioeconomic characteristics	
Family monthly income < PKR 75,000	49 (40.5)
Family monthly income ⩾ PKR 75,000	72 (59.5)
Employed	53 (43.8)
Unemployed	68 (56.2)
Educational status	
Illiterate	6 (5.0)
Primary	48 (39.7)
Secondary	37 (30.6)
Higher	30 (24.8)
Clinical outcomes	
Acute kidney injury (AKI)	19 (15.7)
In-hospital mortality	29 (24.0)

Abbreviations: AKI, acute kidney injury; PKR, Pakistani Rupees.

Table II: Baseline characteristics and outcomes according to AKI status (N = 121)

Characteristic	AKI (n = 19)	No AKI (n = 102)	*p*-value
Age ⩾ 61 years	78.9%	66.7%	0.290
Male gender	47.4%	51.0%	0.772
Urban residence	100.0%	80.4%	0.035*
Diabetes mellitus	0.0%	32.4%	0.004*
Hypertension	0.0%	31.4%	0.004*
Dyslipidemia	73.7%	44.1%	0.018*
Smoking	31.6%	38.2%	0.582
Income < 75,000 PKR	73.7%	34.3%	0.001*
Employed	47.4%	43.1%	0.733
In-hospital mortality	42.1%	20.6%	0.044*

**Statistically significant at $p < 0.05$.*
Abbreviations: AKI, acute kidney injury; PKR, Pakistani Rupees.

Table III: Unadjusted and adjusted predictors of in-hospital mortality among patients with AHF (N = 121)

Variable	Unadjusted OR (95% CI)	*p*-value	Adjusted OR (95% CI)	*p*-value
Acute kidney injury	3.05 (1.03–9.05)	0.044*	3.21 (0.73–14.18)	0.124
Age group (61–80 years vs 40–60 years)	1.02 (0.99–1.05)	0.172	1.03 (0.99–1.06)	0.154
Male sex	0.82 (0.35–1.93)	0.648	0.73 (0.23–2.28)	0.591
Diabetes mellitus	1.51 (0.66–3.44)	0.331	0.89 (0.30–2.65)	0.838
Hypertension	0.88 (0.39–1.96)	0.753	0.62 (0.20–1.95)	0.417
Dyslipidemia	0.76 (0.27–2.11)	0.597	0.47 (0.12–1.79)	0.270
Smoking	0.98 (0.38–2.53)	0.963	0.91 (0.28–2.91)	0.876
Occupational status (employed vs unemployed)	7.12 (2.39–21.21)	<0.001**	7.91 (2.38–26.28)	0.001**
Income category (less than vs more than PKR. 75000)	1.11 (0.63–1.94)	0.720	0.87 (0.42–1.79)	0.705
Education level (illiterate vs primary vs secondary vs higher education)	1.04 (0.62–1.75)	0.888	1.22 (0.65–2.29)	0.532

**Statistically significant at $p < 0.05$., **Statistically significant at $p < 0.01$.*
Abbreviations: AHF, acute heart failure; AKI, acute kidney injury; CI, confidence interval; OR, odds ratio; PKR, Pakistani Rupees.

Authors' Contributions:

Cheena Kumari: Conception of study / Designing / Planning, Experimentation / Study Conduction, Analysis / Interpretation / Discussion, Manuscript Writing

Khursheed Hassan: Conception of study / Designing / Planning, Critical Review

Naimat Ullah Junejo: Experimentation / Study Conduction, Analysis / Interpretation / Discussion, Manuscript Writing

Abdul Sami: Experimentation / Study Conduction, Manuscript Writing

Awais Shafi: Experimentation / Study Conduction, Analysis / Interpretation / Discussion

Muhammad Amin: Conception of study / Designing / Planning, Critical Review

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