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Original Article

Factors Associated with Early Acute Kidney Injury in Patients with Acute Decompensated Heart Failure: A Retrospective Observational Study in Bandung, Indonesia

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Abstract

Background: Acute Kidney Injury (AKI) frequently complicates Acute Decompensated Heart Failure (ADHF) and is associated with adverse clinical outcomes. Early recognition of patients at higher risk is clinically important, particularly during the first 48 hours of hospitalization when decongestive treatment and renal monitoring are actively adjusted.

Methods: This retrospective observational registry-based study analyzed adult patients hospitalized with ADHF at Dr. Hasan Sadikin General Hospital, Bandung, Indonesia, from January 2024 to October 2025. Of 279 screened registry records, 148 were included in the final analysis. AKI was defined as an increase in serum creatinine of at least 0.3 mg/dL within 48 hours after admission. Baseline demographic, clinical, echocardiographic, treatment, and laboratory variables were evaluated using bivariate analysis and multivariable logistic regression.

Results: Among 148 included patients, AKI occurred in 67 patients (45.3%). The cohort was predominantly composed of patients with reduced Left Ventricular Ejection Fraction (LVEF), with 145 patients (98.0%) having LVEF ≤40%. Admission N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) >5000 pg/mL was associated with higher odds of early AKI in the adjusted model (Adjusted Odds Ratio [AOR] 2.04; 95% Confidence Interval [CI] 1.02-4.11; p=0.045). Hypertension and high initial furosemide dose showed nonsignificant trends, whereas other demographic and comorbidity variables did not show statistically significant associations in this cohort.

Conclusions: Elevated admission NT-proBNP was associated with early AKI among patients hospitalized with ADHF. However, these findings should be interpreted as exploratory and hypothesis-generating rather than causal or predictive. Validation in larger and more diverse cohorts is required.

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Keywords: Acute decompensated heart failure; acute kidney injury; associated factors; NT-proBNP; renal function

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Introduction

Acute Decompensated Heart Failure (ADHF) is a leading cause of hospitalization and is associated with substantial morbidity and mortality.[1-2] Renal dysfunction frequently accompanies ADHF and may manifest as Acute Kidney Injury (AKI) early during hospitalization.[3-4] Early AKI can complicate clinical management, limit decongestive treatment strategies, and contribute to worse clinical outcomes.[5-7] The reported prevalence of AKI among hospitalized patients with ADHF varies across studies, commonly ranging from approximately 20% to 40% when serum creatinine-based criteria are used, with some reviews reporting rates approaching 47% depending on case mix, baseline creatinine definition, and observation window.[6,8]

AKI in ADHF arises through multifactorial mechanisms. Reduced effective renal perfusion, neurohormonal activation, venous congestion, and treatment-related changes in intravascular volume may interact during the early phase of hospitalization.[3,9-14] Venous congestion is increasingly recognized as an important pathway in cardiorenal dysfunction because elevated venous and renal interstitial pressures can reduce glomerular filtration even when systemic arterial pressure appears acceptable.[10-11] These mechanisms support the clinical relevance of identifying baseline factors associated with early renal deterioration in ADHF.

Demographic characteristics, cardiovascular comorbidities, diuretic exposure, Left Ventricular Ejection Fraction (LVEF), and congestion-related biomarkers have all been evaluated as potential correlates of renal dysfunction in acute Heart Failure (HF).[12,15-24] Among these markers, N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) reflects myocardial wall stress and congestion severity, and is recommended in heart failure care for diagnostic and prognostic assessment.[2,23-24] However, evidence from low- and middle-income country settings remains limited, and local registry data may help clarify whether routinely available admission variables can identify patients who warrant closer early renal monitoring.

This study aimed to evaluate factors associated with early AKI among adults hospitalized with ADHF at a tertiary referral hospital in Bandung, Indonesia. The analysis focused on AKI occurring within the first 48 hours after admission and was intended to support practical early risk awareness rather than to establish causal relationships or to provide definitive predictions.

Methods

Study Design, Setting, and Ethical Considerations

This retrospective observational study used registry data on adult patients hospitalized with ADHF at Dr. Hasan Sadikin General Hospital, Bandung, Indonesia. The registry period included admissions from January 2024 to October 2025. Baseline admission variables were evaluated in relation to AKI occurring within the first 48 hours of hospitalization. Because this analysis was based on existing registry records and did not involve prospective follow-up beyond the early hospitalization window, the findings were interpreted as associations rather than causal or predictive effects. The study received ethical approval from the Health Research Ethics Committee, Faculty of Medicine, Universitas Padjadjaran (DP.04.03/D. XIV.6.5/591/2025).

Participants and Eligibility Criteria

Records were eligible for inclusion if patients were aged 18 years or older and had a documented diagnosis of ADHF during the early phase of hospitalization. Records were excluded if key variables required for outcome or exposure assessment were incomplete, if patients had end-stage kidney disease receiving chronic maintenance dialysis, including hemodialysis or Continuous Ambulatory Peritoneal Dialysis (CAPD), or if they had a history of kidney transplantation.

Study Variables

Baseline variables evaluated in this study included age category, sex, history of hypertension, coronary artery disease, prior heart failure, diabetes mellitus, initial furosemide therapy dose, LVEF category, and admission NT-proBNP category. These variables were selected because they were clinically plausible and available in the ADHF registry. The term "associated factors" was used throughout the manuscript because the retrospective observational design does not support causal inference.

Baseline Creatinine Definition and Imputation

Baseline serum creatinine was defined as the first available creatinine value recorded in the registry at hospital admission. When the baseline creatinine value was missing, serum creatinine was imputed using the New Linear Equation (New-LE), an age- and sex-based formula: baseline serum creatinine (μmol/L) = 55.2 + (0.525 × age in years) - 15.0 for female patients, whereas no subtraction was applied for male patients. Estimated values in μmol/L were converted to mg/dL by dividing by 88.4. The final

baseline value used for analysis was the measured admission creatinine when available or the New-LE-estimated creatinine when the measured value was missing. This approach was prespecified, but it may introduce classification uncertainty and was therefore considered in the interpretation of findings.

Outcome Definition

The primary outcome was early AKI, defined as an increase in serum creatinine of at least 0.3 mg/dL within 48 hours after admission, consistent with the creatinine component of the Kidney Disease: Improving Global Outcomes (KDIGO) AKI definition.[37] AKI ascertainment was based on the final baseline serum creatinine value, and the creatinine level was measured approximately 48 hours after admission. Urine output criteria were not used because urine output data were not consistently available in the registry.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 27. Categorical variables were summarized as counts and proportions. Continuous variables were summarized as the mean with standard deviation or as the median with minimum and maximum values, as appropriate. Bivariate associations were assessed using the chi-square test or Fisher's exact test. Variables with a bivariate p-value <0.25 were entered into a multivariable logistic regression model to estimate Adjusted Odds Ratios (aORs) with 95% Confidence Intervals (CIs). Statistical significance was defined as a two-sided p-value <0.05. Because several exposure categories contained small numbers of patients, all estimates were interpreted cautiously, with attention to confidence interval width and proximity to the null value.

Results

This study used secondary data from the ADHF registry at Dr. Hasan Sadikin General Hospital, Bandung, Indonesia. A total of 279 registry records from January 2024 to October 2025 were screened. Of these, 122 records were excluded because of incomplete registry data, and 9 records were excluded because of a history of end-stage chronic kidney disease. The final analytic sample comprised 148 records that met the eligibility criteria. The patient selection process is summarized in Figure 1.

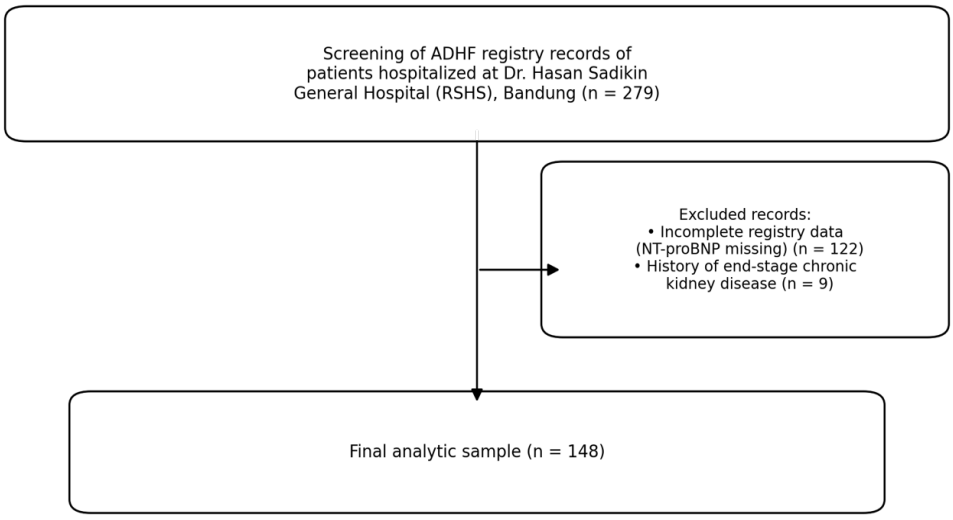

Figure 1. Flow of registry record selection for the study population.

Table 1 shows the baseline characteristics of the included patients. The mean age was 54 ± 13 years. Patients younger than 60 years constituted the majority (98 patients, 66.2%), whereas 50 patients (33.8%) were aged 60 years or older. Male patients predominated (90 patients, 60.8%). Hypertension was present in 72 patients (48.6%), diabetes mellitus in 47 patients (31.8%), coronary artery disease in 50 patients (33.8%), and prior heart failure in 109 patients (73.6%). All patients received furosemide at the beginning of hospitalization. A low initial dose was administered to 136 patients (91.9%), whereas a high initial dose was administered to 12 patients (8.1%). The median LVEF was 24% (range, 12-57%). Most patients had LVEF ≤40% (145 patients, 98.0%), whereas only 3 patients (2.0%) had LVEF >40%. The median NT-proBNP level was 6539.5 pg/mL (range, 16-25000 pg/mL), and 91 patients (61.4%) had NT-proBNP >5000 pg/mL. Overall, this cohort was dominated by patients with Heart

Failure with reduced Ejection Fraction (HFrEF) and substantial biomarker evidence of congestion.

Table 2 shows renal function parameters during the first 48 hours of hospitalization. The median admission serum creatinine was 0.92 mg/dL (range, 0.44-2.95 mg/dL). After 48 hours, the median serum creatinine increased to 1.19 mg/dL (range, 0.56-3.74 mg/dL). The median change in creatinine was 0.25 mg/dL, ranging from a decrease of -0.44 mg/dL to a maximum increase of 2.00 mg/dL. Using the criterion of a serum creatinine increase of at least 0.3 mg/dL, 67 patients (45.3%) developed early AKI, whereas 81 patients (54.7%) did not.

Table 1. Characteristics of study participants with acute decompensated heart failure.

Variable	n = 148
Age (years), mean ± SD	54 ± 13
Age category, n (%)	
60 years or older	50 (33.8)
Younger than 60 years	98 (66.2)
Sex, n (%)	
Female	58 (39.2)
Male	90 (60.8)
History of hypertension, n (%)	
Yes	72 (48.6)
No	76 (51.4)
History of coronary artery disease, n (%)	
Yes	50 (33.8)
No	98 (66.2)
Prior history of heart failure, n (%)	
Yes	109 (73.6)
No	39 (26.4)
History of diabetes mellitus, n (%)	
Yes	47 (31.8)
No	101 (68.2)
Initial furosemide therapy, n (%)	
High dose	12 (8.1)
Low dose	136 (91.9)
LVEF (%), median (min–max)	24 (12–57)
LVEF category, n (%)	
40% or lower	145 (98.0)
Greater than 40%	3 (2.0)
NT-proBNP (pg/mL), median (min–max)	6539.5 (16–25000)
NT-proBNP category, n (%)	
Greater than 5000 pg/mL	91 (61.4)
5000 pg/mL or lower	57 (38.6)

Abbreviations: SD = standard deviation; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide.

Table 3 shows the bivariate analyses of factors associated with early AKI. No statistically significant association was observed between AKI and age category (p=0.899; Odds Ratio [OR] 0.96; 95% CI 0.48-1.89) or sex (p=0.801; OR 1.09; 95% CI 0.56-2.11). Hypertension showed a nonsignificant trend toward higher odds of AKI, with AKI occurring in 52.8% of patients with hypertension and 38.2% of those without hypertension (p=0.074; OR 1.81; 95% CI 0.94-3.49). Histories of coronary artery disease, prior heart failure, and diabetes mellitus did not show statistically significant associations. High initial furosemide dose showed a nonsignificant trend toward lower odds of AKI (16.7% in the high-dose

group versus 47.8% in the low-dose group; p=0.066; OR 0.22; 95% CI 0.05-1.03). LVEF category did not show a statistically significant association, although interpretation was limited by the very small number of patients with LVEF >40%.

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Table 2. Serum creatinine distribution and acute kidney injury incidence in patients with acute decompensated heart failure.

Variable	n = 148
Admission serum creatinine (mg/dL), median (min–max)	0.92 (0.44–2.95)
Serum creatinine at 48 hours (mg/dL), median (min–max)	1.19 (0.56–3.74)
Δ creatinine (mg/dL), median (min–max)	0.25 (−0.44–2.00)
Acute kidney injury (AKI), n (%)	
Yes (serum creatinine increase ≥ 0.3 mg/dL)	67 (45.3)
No	81 (54.7)

Table 3. Bivariate analysis of factors associated with acute kidney injury in patients with acute decompensated heart failure.

Variable	Total (n = 148)	AKI: Yes (n = 67)	AKI: No (n = 81)	p value	OR (95% CI)
Age category, n (%)					
60 years or older	50	23 (46.0)	27 (54.0)	0.899a	0.96 (0.48–1.89)
Younger than 60 years	98	44 (44.9)	54 (55.1)		1.00 (ref)
Sex, n (%)					
Female	58	27 (46.6)	31 (53.4)	0.801a	1.09 (0.56–2.11)
Male	90	40 (44.4)	50 (55.6)		1.00 (ref)
History of hypertension, n (%)					
Yes	72	38 (52.8)	34 (47.2)	0.074a	1.81 (0.94–3.49)
No	76	29 (38.2)	47 (61.8)		1.00 (ref)
History of coronary artery disease, n (%)					
Yes	50	22 (44.0)	28 (56.0)	0.825a	0.93 (0.47–1.84)
No	98	45 (45.9)	53 (54.1)		1.00 (ref)
Prior history of heart failure, n (%)					
Yes	109	47 (43.1)	62 (56.9)	0.379a	0.72 (0.35–1.50)
No	39	20 (51.3)	19 (48.7)		1.00 (ref)
History of diabetes mellitus, n (%)					
Yes	47	22 (46.8)	25 (53.2)	0.798a	1.10 (0.55–2.19)
No	101	45 (44.6)	56 (55.4)		1.00 (ref)
Initial furosemide therapy, n (%)					
High dose	12	2 (16.7)	10 (83.3)	0.066a	0.22 (0.05–1.03)
Low dose	136	65 (47.8)	71 (52.2)		1.00 (ref)

LVEF category, n (%)					
40% or lower	145	65 (44.8)	80 (55.2)	0.452b	0.41 (0.04–4.58)
Greater than 40%	3	2 (66.7)	1 (33.3)		1.00 (ref)
NT-proBNP category, n (%)					
Greater than 5000 pg/mL	91	47 (51.6)	44 (48.4)	0.049a*	1.98 (1.00–3.91)
5000 pg/mL or lower	57	20 (35.1)	37 (64.9)		1.00 (ref)

Notes: p values were calculated using a Chi-square test or b Fisher's exact test; *statistically significant at $p < 0.05$.

Table 4. Multivariable analysis of factors associated with acute kidney injury.

Variable	aOR (95% CI)	p value
Hypertension	1.79 (0.91–3.51)	0.092
High-dose initial furosemide therapy	0.24 (0.05–1.15)	0.075
High NT-proBNP (greater than 5000 pg/mL)	2.04 (1.02–4.11)	0.045*

Abbreviations: aOR = adjusted odds ratio; CI = confidence interval; *statistically significant at $p < 0.05$. Variables entered into the multivariable model were those with bivariate p values < 0.25.

Table 4 shows the multivariable logistic regression analysis. After adjustment for variables with bivariate p-values <0.25, admission NT-proBNP >5000 pg/mL remained associated with higher odds of early AKI (aOR 2.04; 95% CI 1.02-4.11; p=0.045). Hypertension showed a nonsignificant trend toward higher odds of AKI (aOR 1.79; 95% CI 0.91-3.51; p=0.092), whereas high initial furosemide dose showed a nonsignificant trend toward lower odds (aOR 0.24; 95% CI 0.05-1.15; p=0.075). These adjusted estimates should be interpreted as exploratory due of the limited sample size, small numbers in some exposure categories, and CIs close to the null.

Discussion

This retrospective registry-based study found that early AKI occurred in 45.3% of adults hospitalized with ADHF at a tertiary referral hospital in Bandung, Indonesia. The proportion was within the upper range of prior estimates in acute heart failure populations and may reflect the severity of systolic dysfunction and congestion in this cohort.[3,5-6,8] The main finding was that admission NT-proBNP >5000 pg/mL was associated with approximately twofold higher adjusted odds of early AKI. However, the CI was close to unity, and the finding should be interpreted as an exploratory signal rather than as evidence of an independent predictive effect.

The association between elevated NT-proBNP and early AKI is biologically plausible. NT-proBNP reflects myocardial wall stress and congestion severity, and contemporary heart failure literature recognizes venous congestion as a key contributor to kidney dysfunction.[10-11,23-24] Increased central venous pressure, renal venous pressure, and renal interstitial pressure can reduce the glomerular filtration gradient and impair renal function, even when arterial pressure appears adequate.[10,32] These mechanisms are consistent with cardiorenal syndrome frameworks that position congestion as both a marker of disease severity and a contributor to renal vulnerability.[9,33]

These findings also align with current guideline-oriented care, while adding a local exploratory observation. The 2022 AHA/ACC/HFSA Heart Failure Guideline supports measuring natriuretic peptides for diagnostic and prognostic assessment in HF, including in hospitalized patients.[2] The 2024 American Heart Association scientific statement on kidney dysfunction in HF emphasizes that renal function must be interpreted within the broader clinical trajectory of HF and congestion.[4] Meanwhile, KDIGO criteria define AKI using serum creatinine changes and urine output, and the present study used only the creatinine component because registry urine output was incomplete.[37] In this context, NT-proBNP should not be interpreted as a stand-alone predictor of AKI, but it may help clinicians identify patients who require closer creatinine monitoring, careful decongestion assessment, and more cautious interpretation of early renal function changes.

The local setting is relevant because ADHF care in low- and middle-income countries often depends on routinely available registry variables rather than advanced hemodynamic monitoring or

novel renal biomarkers. A simple admission marker such as NT-proBNP may therefore have practical value as a signal for early monitoring. Nevertheless, the present findings should not be used to change therapy on their own. Instead, they support the need for larger local and multicenter studies that integrate biomarkers, fluid balance, diuretic response, blood pressure trajectories, and objective assessment of congestion.

No statistically significant associations were observed for age category, sex, coronary artery disease, prior HF, or diabetes mellitus. These results should not be interpreted as a definitive absence of association. The study was not powered to detect small or moderate effects across multiple variables, and several exposure groups were small. Similarly, hypertension and high initial furosemide dose showed nonsignificant trends, but these estimates may be unstable. The trend toward higher-dose furosemide may reflect treatment selection, differences in the congestion phenotype, or unmeasured clinical factors rather than a protective effect.[12-14] Future analyses should include diuretic response, urine output, fluid balance, and congestion markers to clarify this relationship.

The predominance of HFrEF in this cohort is an important consideration. Almost all included patients had LVEF ≤40%, and only 3 patients had LVEF >40%. Therefore, the findings primarily apply to patients with reduced ejection fraction and should not be generalized to patients with Heart Failure with mildly reduced Ejection Fraction (HFmrEF) or Heart Failure with preserved Ejection Fraction (HFpEF). Prior studies suggest that the relationship between LVEF phenotype and worsening renal function may differ across acute HF subgroups, and broader representation of HFmrEF and HFpEF is needed before conclusions can be extended to those populations.[35-36]

Strengths of this study include the use of real-world registry data from a tertiary referral hospital and the focus on a clinically meaningful early hospitalization window. The study also explicitly described its approach to baseline creatinine ascertainment, which is important because this can substantially influence AKI classification and comparability across cohorts.[28-31]

This study has several limitations. First, its retrospective observational design limits causal inference and creates susceptibility to residual confounding. Second, 131 of 279 screened records were excluded, mainly because of incomplete registry data. This may have introduced selection bias and may limit representativeness. Third, the sample size was modest, and some exposure categories were very small, especially high initial furosemide dose and LVEF >40%, which reduced statistical power and precision of estimates. Fourth, the association between NT-proBNP and AKI was borderline, with the CI close to the null value. Fifth, AKI was defined using serum creatinine within 48 hours and did not include urine output criteria. Sixth, admission creatinine and New-LE imputation were used when measured baseline values were unavailable, which may have led to misclassification, particularly if renal dysfunction had already begun before admission. Finally, the single-center setting and predominance of HFrEF limit generalizability.

Future studies should prospectively evaluate renal trajectories beyond 48 hours and incorporate urine output, fluid balance, congestion measures, hemodynamic variables, and diuretic response. External validation across hospitals and HF phenotypes will be important to determine whether admission NT-proBNP can be incorporated into locally applicable tools for early renal monitoring in ADHF.

Conclusion

Early AKI was common among adults hospitalized with ADHF at Dr. Hasan Sadikin General Hospital, Bandung, occurring in 45.3% of included patients within 48 hours of admission. Admission NT-proBNP >5000 pg/mL was associated with higher adjusted odds of early AKI.

These findings suggest that elevated NT-proBNP may serve as a practical signal for closer early renal monitoring in ADHF, particularly in settings where advanced congestion or renal biomarkers are not routinely available. However, the study should be viewed as exploratory and hypothesis-generating. Therefore, larger prospective, multicenter studies are needed to validate this association and determine whether NT-proBNP adds value to clinical risk assessment for early AKI in ADHF.

List of Abbreviations

ADHF	Acute Decompensated Heart Failure
AKI	Acute Kidney Injury
aOR	Adjusted Odds Ratio
CAPD	Continuous Ambulatory Peritoneal Dialysis
CI	Confidence Interval
HF	Heart Failure

HFmrEF	Heart Failure with mildly reduced Ejection Fraction
HFpEF	Heart Failure with preserved Ejection Fraction
HFrEF	Heart Failure with reduced Ejection Fraction
KDIGO	Kidney Disease: Improving Global Outcomes
LVEF	Left Ventricular Ejection Fraction
NT-proBNP	N-terminal pro-B-type natriuretic peptide
OR	Odds Ratio

Ethical Clearance

This study received ethical approval from the Health Research Ethics Committee, Faculty of Medicine, Universitas Padjadjaran (DP.04.03/D. XIV.6.5/591/2025).

Publication Approval

All authors are consent to the publication of this manuscript.

Author Contributions

HSP and FYR conceptualized the study; HSP, FYR, RA, IW, JWM, and LS contributed to methodology, analysis, interpretation, manuscript drafting, and critical revision.

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Conflict of Interest

The authors declare no conflicts of interest.

Availability of Data and Materials

The data that support the findings of this study are available from the corresponding author upon reasonable request and with permission from Dr. Hasan Sadikin General Hospital.

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Authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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