Original Research

INCIDENCE AND PREDICTORS OF ACUTE KIDNEY INJURY IN PATIENTS PRESENTING WITH HYPERTENSIVE EMERGENCY: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Hypertensive emergency (HE) remains a life-threatening condition characterised by severe blood pressure elevation accompanied by acute target organ damage. While acute kidney injury (AKI) represents a frequent and prognostically significant complication of HE, contemporary data on its incidence and predictors using current diagnostic criteria remain limited.

Objectives: This study aimed to determine the incidence of AKI among patients presenting with hypertensive emergencies, identify independent clinical and laboratory predictors of AKI, and evaluate the performance of novel biomarkers for early risk stratification.

Methods: This prospective observational study was conducted at a tertiary care hospital between January 2024 and February 2025. Consecutive adults presenting with hypertensive emergency (systolic BP >180 mmHg or diastolic BP >110 mmHg with evidence of target organ damage) were enrolled. AKI was defined and staged according to kidney disease: Improving Global Outcomes (KDIGO) 2024 criteria. Demographic, clinical, and laboratory variables were collected on admission. Serum creatinine, cystatin C, high-sensitivity cardiac troponin I, and the triglyceride-glucose index were measured. Multivariable logistic regression analysis identified independent predictors of AKI. Model discrimination was assessed using the area under the receiver operating characteristic curve analysis.

Results: Among 267 enrolled patients (mean age 63.1±14.6 years, 59.2% male), AKI occurred in 110 patients (41.2%), with KDIGO stage 1 in 57 (21.3%), stage 2 in 34 (12.7%), and stage 3 in 19 (7.1%). Independent predictors of AKI included admission serum creatinine >1.1 mg/dL (adjusted odds ratio [aOR] 3.91, 95% CI 2.38–6.42, p<0.001), elevated high-sensitivity troponin I (aOR 2.72, 95% CI 1.68–4.41, p<0.001), triglyceride-glucose index ≥9.2 (aOR 2.38, 95% CI 1.46–3.88, p<0.001), systolic blood pressure variability >25% during initial 6 hours (aOR 2.24, 95% CI 1.37–3.66, p=0.001), and Black race (aOR 2.04, 95% CI 1.24– 3.35, p=0.005). A prediction model incorporating these variables demonstrated excellent discrimination (AUC 0.85, 95% CI 0.80–0.89).

Conclusion: AKI complicates over 40% of hypertensive emergency presentations, with one-fifth of cases reaching moderate- to-severe stages. Readily available clinical and laboratory parameters enable accurate early risk stratification, potentially guiding intensity of monitoring and therapeutic interventions.

Keywords: Hypertensive Emergency, Acute Kidney Injury, Biomarkers, Risk Prediction, Target Organ Damage.

INTRODUCTION

Hypertensive emergency represents the most severe manifestation of hypertensive crisis, defined as an acute elevation of blood pressure to levels typically exceeding 180/120 mmHg, accompanied by evidence of new or progressive target organ damage.

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Despite advances in antihypertensive therapy and widespread availability of intensive care resources, hypertensive emergency remains associated with substantial morbidity and mortality, with in-hospital mortality rates ranging from 5% to 15% in contemporary series [2,3]. The kidney is among the most frequently affected target organs in this setting, reflecting its unique susceptibility to hemodynamic perturbations and its central role in blood pressure regulation [4]. Acute kidney injury complicating hypertensive emergency carries clinical significance for several reasons. First, renal involvement identifies a subgroup of patients with more severe systemic microvascular injury and worse prognosis.

Second, the presence of AKI complicates blood pressure management, as rapid pressure reduction may theoretically compromise renal perfusion pressure in kidneys already subjected to autoregulatory failure [5,6]. Third, even modest degrees of AKI during hypertensive emergency may accelerate the progression of underlying chronic kidney disease or precipitate de novo chronic kidney impairment requiring long-term nephrological follow-up [7,8,9].

The reported incidence of AKI in hypertensive emergencies varies considerably across studies, ranging from 20% to 60%, reflecting differences in study populations, diagnostic criteria, and clinical settings [6,12]. This wide variation limits accurate prognostication and impedes the development of standardised management protocols. Furthermore, most existing data derive from retrospective analyses using earlier AKI definitions, potentially underestimating the true burden of renal involvement given the increased sensitivity of contemporary KDIGO criteria [13]. Identification of patients at highest risk for AKI during hypertensive emergencies carries important therapeutic implications. While all patients with hypertensive emergencies require prompt blood pressure reduction, the optimal rate and magnitude of pressure decrease remain debated, particularly regarding renal outcomes. Recent evidence suggests that excessive blood pressure variability during initial management may exacerbate renal injury, whereas controlled, individualised pressure reduction might preserve renal function [5,6]. Biomarkers that identify patients with heightened renal susceptibility could enable risk-stratified management, potentially improving outcomes. Several novel biomarkers have emerged as promising tools for AKI risk assessment in various acute settings. High-sensitivity cardiac troponin, traditionally viewed as a marker of myocardial injury, may reflect systemic microvascular damage in hypertensive emergencies and correlate with renal outcomes [13]. The triglyceride-glucose index, a simple surrogate measure of insulin resistance derived from fasting triglyceride and glucose levels, has demonstrated predictive value for AKI in critically ill hypertensive patients, potentially through mechanisms linking metabolic dysregulation to microvascular dysfunction [14,20,21]. Cystatin C, less dependent on muscle mass than creatinine, may enable more accurate estimation of baseline renal function and earlier detection of acute functional changes [17].

Despite these advances, prospective evaluation of contemporary biomarkers specifically in the context of hypertensive emergency remains limited. The present study was therefore designed to determine the current incidence of AKI among patients presenting with hypertensive emergency using the 2024 KDIGO criteria, to identify independent clinical and laboratory predictors of AKI development, and to evaluate the performance of readily available biomarkers in early risk stratification.

MATERIALS AND METHODS

Study Design and Setting- This prospective observational cohort study was conducted in the Department of Emergency Medicine and Intensive Care Units of a tertiary care hospital between January 2024 and February 2025. Written informed consent was obtained from all participants or their legally authorised representatives prior to enrollment. The study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cohort studies.

Study Population- Consecutive adult patients (aged ≥18 years) presenting to the emergency department with hypertensive emergency were screened for eligibility. Hypertensive emergency was defined according to current European Society of Cardiology/European Society of Hypertension guidelines as systolic blood pressure >180 mmHg or diastolic blood pressure >110 mmHg accompanied by evidence of acute hypertension-mediated target organ damage. Target organ damage was defined by presence of any of the following: acute kidney injury (as defined below), acute myocardial ischemia (symptoms consistent with acute coronary syndrome with or without electrocardiographic changes or troponin elevation), acute heart failure (clinical and radiographic evidence of pulmonary edema), acute aortic syndrome (imaging-confirmed aortic dissection or intramural hematoma), hypertensive encephalopathy (altered mental status with or without seizures or posterior reversible encephalopathy syndrome on neuroimaging), or acute retinal hemorrhages or exudates on fundoscopic examination.

Inclusion Criteria:

- Age ≥18 years
- Systolic blood pressure >180 mmHg or diastolic blood pressure >110 mmHg documented on at least two measurements 5 minutes apart
- Evidence of acute hypertension-mediated target organ damage as defined above
- Presentation directly to the study institution's emergency department
- Ability to obtain informed consent within 24 hours of presentation

Exclusion Criteria:

- Age <18 years
- Pregnancy or lactation

- End-stage kidney disease requiring chronic dialysis
- Known stage 4 or 5 chronic kidney disease (estimated glomerular filtration rate <30 mL/min/1.73m²)
- Solid organ or hematopoietic stem cell transplantation
- Obstructive uropathy or suspected renovascular disease as primary cause of hypertension
- Exposure to intravenous contrast media within 72 hours preceding presentation
- Transfer from another hospital with more than 24 hours of prior inpatient management
- Refusal or inability to provide informed consent
- Known allergies to any study-related assessment procedure
- Concurrent participation in interventional clinical trial

Data Collection and Definitions- Baseline demographic characteristics included age, sex, self-reported race/ethnicity, and insurance status. Medical history was obtained from patients, family members, and available medical records, including prior diagnosis of hypertension, diabetes mellitus, chronic kidney disease, cardiovascular disease, and current antihypertensive medications. Medication adherence was assessed using the Morisky Medication Adherence Scale-8. Initial clinical assessment included triage vital signs (heart rate, respiratory rate, oxygen saturation), bilateral blood pressure measurements using validated oscillometric devices with appropriately sized cuffs, and fundoscopic examination when feasible. The initial blood pressure value used for analysis was the mean of two measurements obtained 5 minutes apart after at least 5 minutes of quiet rest in the supine position. Blood pressure variability during the first 6 hours was quantified as the coefficient of variation of hourly systolic blood pressure measurements.

Laboratory evaluation was performed on admission prior to initiation of antihypertensive therapy. Venous blood samples were collected for measurement of serum creatinine, cystatin C, electrolytes, blood urea nitrogen, complete blood count, high- sensitivity cardiac troponin I, and lipid profile, including triglycerides and glucose, for calculation of the triglyceride-glucose index. The estimated glomerular filtration rate was calculated using the 2021 CKD-EPI creatinine-cystatin C equation. The triglyceride-glucose index was calculated as ln (fasting triglycerides [mg/dL] × fasting glucose [mg/dL]/2). Urinalysis with microscopy was performed on all patients, and spot urine protein-to-creatinine ratio was measured when feasible. All laboratory assays were performed in the hospital's accredited clinical laboratory using standardised methodologies. Serum creatinine was measured using an enzymatic method traceable to isotope dilution mass spectrometry reference material. Cystatin C was measured using a particle-enhanced immunoturbidimetric assay. High-sensitivity cardiac troponin I was measured by chemiluminescent microparticle immunoassay, with the manufacturer's reported 99th percentile upper reference limit of 26 ng/L.

Management Protocol- Patients were managed according to institutional guidelines, in line with current recommendations. All patients were admitted to appropriate monitored settings (intensive care unit, intermediate care unit, or telemetry floor) based on clinical severity and presence of specific target organ involvement. Antihypertensive therapy was initiated with intravenous agents (nicardipine, labetalol, or clevidipine) titrated to achieve a controlled reduction in blood pressure not exceeding 25% during the first hour, with further gradual reduction toward 160/100 mmHg over the subsequent 2–6 hours. The choice of the specific agent was at the discretion of the treating physician, based on concomitant conditions. Oral antihypertensive therapy was introduced as intravenous agents were tapered. Fluid administration was guided by clinical assessment of volume status, with avoidance of excessive fluid administration in the absence of hypovolemia.

Outcome Measures

Primary Outcomes- The primary outcome was the development of AKI within seven days of presentation, defined and staged according to KDIGO 2024 clinical practice guidelines. AKI was defined as any of the following: an increase in serum creatinine by ≥0.3 mg/dL within 48 hours; an increase in serum creatinine to ≥1.5 times baseline within the preceding seven days; or urine volume <0.5 mL/kg/hour for six hours. Baseline serum creatinine was defined as the most recent value within 3 months of presentation, or the admission value if no prior value was available, and confirmed that the patient had no history suggesting baseline renal impairment. AKI stage 1 was defined as creatinine increase ≥0.3 mg/dL or 1.5–1.9 times baseline; stage 2 as 2.0–2.9 times baseline; stage 3 as ≥3.0 times baseline, increase to ≥4.0 mg/dL, initiation of renal replacement therapy, or in patients <18 years, decrease in eGFR to <35 mL/min/1.73m².

Secondary Outcomes

- AKI stage distribution among affected patients
- Need for renal replacement therapy during hospitalisation
- In-hospital mortality
- Length of hospital and intensive care unit stay

- Renal recovery at discharge, defined as return of serum creatinine to within 0.3 mg/dL of baseline
- Composite of major adverse kidney events at 30 days (death, need for renal replacement therapy, or persistent kidney dysfunction defined as creatinine ≥1.5 times baseline).

Sample Size Calculation- The sample size was calculated based on the anticipated AKI incidence of 40% from preliminary data and previous studies. To identify independent predictors with odds ratios of ≥2.0 for variables with a prevalence of at least 20%, assuming a conservative event rate of 40%, 10 events per predictor variable, and up to 12 candidate variables in multivariable analysis, a minimum of 120 patients with AKI was required. Accounting for 10% loss to follow-up or incomplete data, we targeted enrollment of 267 patients to achieve 107–110 AKI events.

Statistical Analysis- Statistical analysis was performed using SPSS version 29.0 (IBM Corp., Armonk, NY) and R version 4.3.2 (R Foundation for Statistical Computing, Vienna, Austria). Continuous variables were assessed for normality using the Shapiro-Wilk test and expressed as mean ± standard deviation or median with interquartile range as appropriate. Categorical variables were expressed as frequencies and percentages. Baseline characteristics were compared between patients who developed AKI and those who did not use Student's T-test or Mann-Whitney U test for continuous variables and chi-square test or Fisher's exact test for categorical variables, as appropriate. The cumulative incidence of AKI over time was estimated using the Kaplan-Meier method, with patients censored at death or hospital discharge.

Univariable logistic regression analysis was performed to identify potential predictors of AKI. Variables with $p<0.10$ in univariable analysis were considered for inclusion in multivariable analysis after assessment for collinearity using variance inflation factors. Multivariable logistic regression with backward stepwise elimination was performed to identify independent predictors of AKI. Adjusted odds ratios with 95% confidence intervals were calculated. Model calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test, and discrimination was evaluated using the area under the receiver operating characteristic curve. The predictive performance of individual biomarkers and the combined model was assessed using receiver operating characteristic curve analysis with calculation of area under the curve, sensitivity, specificity, positive predictive value, and negative predictive value at optimal cutoffs determined by Youden's index. Subgroup analyses were performed according to age (<65 vs. ≥65 years), sex, presence of baseline chronic kidney disease, and diabetes mellitus. Sensitivity analyses were conducted, excluding patients with missing data and using multiple imputation to handle missing covariates.

All statistical tests were two-tailed, and p-values <0.05 were considered statistically significant. No adjustment for multiple comparisons was made in the secondary analyses, which were considered exploratory.

RESULTS

Patient Characteristics- Between January 2024 and February 2025, 356 patients with hypertensive emergencies were assessed for eligibility. After excluding 89 patients who met exclusion criteria (32 with end-stage kidney disease or advanced chronic kidney disease, 18 transferred from outside hospitals, 14 with contrast exposure within 72 hours, 12 who declined participation, 8 with obstructive uropathy, and 5 with other reasons), 267 patients were enrolled and included in the final analysis. Baseline demographic and clinical characteristics of the study population are presented in Table 1. The mean age was 63.1±14.6 years, and 158 patients (59.2%) were male. The cohort was racially diverse, with 134 patients (50.2%) identifying as Black, 102 (38.2%) as White, 22 (8.2%) as Hispanic, and 9 (3.4%) as Asian or another race/ethnicity. A history of hypertension was present in 235 patients (88.0%), with a mean duration of hypertension of 11.6±8.9 years. Diabetes mellitus was present in 99 patients (37.1%), and baseline chronic kidney disease (stage 1–3) in 67 patients (25.1%).

Presenting blood pressure revealed severe elevation with a mean systolic blood pressure of 202.1±17.2 mmHg and a mean diastolic blood pressure of 116.2±12.8 mmHg. The most common manifestations of target organ damage were acute kidney injury (isolated or combined) in 82 patients (30.7%), acute heart failure in 60 patients (22.5%), acute coronary syndrome in 50 patients (18.7%), and hypertensive encephalopathy in 29 patients (10.9%). Sixteen patients (6.0%) presented with acute aortic syndrome, and 12 patients (4.5%) with hypertensive retinopathy with haemorrhages or exudates. Multiple target organ involvement was present in 78 patients (29.2%).

Table 1. Baseline Characteristics of Patients Presenting with Hypertensive Emergency (N=267)

Characteristic	Total (N=267)	AKI (N=110)	No AKI (N=157)	P-Value
Age, years, mean ± SD	63.1 ± 14.6	65.4 ± 14.1	61.5 ± 14.8	0.03
Male sex, n (%)	158 (59.2)	68 (61.8)	90 (57.3)	0.46
Race/ethnicity, n (%)	0.02			

- Black	134 (50.2)	64 (58.2)	70 (44.6)	
- White	102 (38.2)	33 (30.0)	69 (43.9)	
- Hispanic	22 (8.2)	10 (9.1)	12 (7.6)	
- Asian/other	9 (3.4)	3 (2.7)	6 (3.8)	
Medical history, n (%)				
- Hypertension	235 (88.0)	99 (90.0)	136 (86.6)	0.41
- Diabetes mellitus	99 (37.1)	48 (43.6)	51 (32.5)	0.06
- Chronic kidney disease	67 (25.1)	43 (39.1)	24 (15.3)	<0.001
- Coronary artery disease	58 (21.7)	28 (25.5)	30 (19.1)	0.21
- Heart failure	43 (16.1)	22 (20.0)	21 (13.4)	0.15
Antihypertensive medications, n (%)	0.28			
- None	40 (15.0)	14 (12.7)	26 (16.6)	
- 1 agent	67 (25.1)	24 (21.8)	43 (27.4)	
- 2 agents	85 (31.8)	36 (32.7)	49 (31.2)	
- ≥3 agents	75 (28.1)	36 (32.7)	39 (24.8)	
Admission SBP, mmHg, mean ± SD	202.1 ± 17.2	204.6 ± 17.8	200.4 ± 16.6	0.04
Admission DBP, mmHg, mean ± SD	116.2 ± 12.8	117.4 ± 13.4	115.4 ± 12.3	0.20
Heart rate, bpm, mean ± SD	95.1 ± 18.7	98.0 ± 19.8	93.2 ± 17.8	0.03
Initial serum creatinine, mg/dL, median (IQR)	1.2 (0.9–1.7)	1.6 (1.2–2.3)	1.0 (0.8–1.3)	<0.001
eGFR, mL/min/1.73m², median (IQR)	61 (40–85)	45 (31–61)	75 (57–93)	<0.001
Cystatin C, mg/L, median (IQR)	1.19 (0.93–1.66)	1.54 (1.20–2.09)	1.03 (0.85–1.29)	<0.001
hs-Troponin I, ng/L, median (IQR)	36 (18–88)	70 (34–162)	25 (14–50)	<0.001
TyG index, median (IQR)	9.1 (8.7–9.6)	9.4 (8.9–9.9)	8.9 (8.5–9.3)	<0.001
Proteinuria, n (%)	98 (36.7)	60 (54.5)	38 (24.2)	<0.001

AKI, acute kidney injury; SBP, systolic blood pressure; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; hs-Troponin I, high-sensitivity cardiac troponin I; TyG, triglyceride-glucose; IQR, interquartile range; SD, standard deviation.

Incidence and Characteristics of Acute Kidney Injury- AKI developed in 110 patients (41.2%) within seven days of presentation. The timing of AKI onset is illustrated in Figure 1. AKI was present on admission in 66 patients (60.0% of AKI cases), with the remaining 44 patients (40.0%) developing AKI during hospitalisation at a median of 2 days (IQR 1–3 days) after admission. Among patients with AKI, the distribution by KDIGO stage was stage 1 in 57 patients (51.8% of AKI cases, 21.3% of total cohort), stage 2 in 34 patients (30.9% of AKI cases, 12.7% of total cohort), and stage 3 in 19 patients (17.3% of AKI cases, 7.1% of total cohort). Of the 19 patients with stage 3 AKI, 8 (42.1%) required renal replacement therapy during hospitalisation**.**

Patients who developed AKI were older, more frequently Black, and had a higher prevalence of diabetes mellitus and pre- existing chronic kidney disease compared to those without AKI (Table 1). They presented with higher systolic blood pressure and heart rate, and had significantly higher admission creatinine, cystatin C, high-sensitivity troponin I, and triglyceride-glucose index values. Proteinuria on admission dipsticks were present in 54.5% of patients who developed AKI versus 24.2% of those who did not (p<0.001).

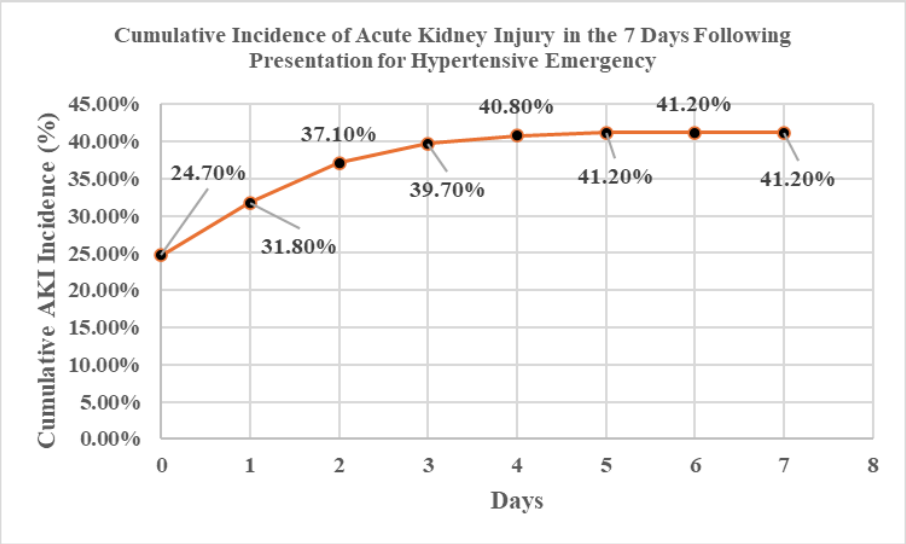

Figure 1. Timing of Acute Kidney Injury Onset Following Hypertensive Emergency Presentation

Interpretation- This Kaplan-Meier curve illustrates the timing of acute kidney injury (AKI) among 267 patients presenting with hypertensive emergency. The cumulative incidence of AKI reached 41.2% (n=110) by day 7. Notably, most AKI cases (60.0%, n=66) were identified on the day of admission (Day 0), indicating that renal injury is frequently established at the time of presentation rather than developing as a complication of hospital management. The remaining 40.0% of cases developed over the subsequent days, with a median onset of 2 days (IQR 1–3) after admission. This highlights a critical window for early risk stratification and intervention upon first medical contact.

Clinical Outcomes- Clinical outcomes according to AKI status are presented in Table 2. Patients with AKI had significantly longer hospital and intensive care unit stays, higher in-hospital mortality, and lower rates of renal recovery at discharge compared to those without AKI. In-hospital mortality occurred in 14 patients (5.2% of the total cohort), with significantly higher mortality in patients with AKI (12.7% vs. 0%, p<0.001). Among AKI patients, mortality increased with AKI severity: 5.3% in stage 1, 14.7% in stage 2, and 26.3% in stage 3 (p for trend <0.001).

Renal recovery at discharge among survivors with AKI was achieved in 72 patients (65.5%), with higher recovery rates in stage 1 (83.3%) than in stage 2 (58.6%) or stage 3 (35.7%) (p<0.001). The composite of major adverse kidney events occurred in 30 days in 31 patients (11.6% of the total cohort), including 14 deaths, 8 patients requiring renal replacement therapy, and 20 patients with persistent kidney dysfunction (some patients experienced multiple events).

Table 2. Clinical Outcomes According to AKI Status

Outcome	Total (N=267)	AKI (N=110)	No AKI (N=157)	P-Value
Hospital LOS, days, median (IQR)	6 (4–10)	9 (6–15)	4 (3–6)	<0.001
ICU admission, n (%)	130 (48.7)	78 (70.9)	52 (33.1)	<0.001
ICU LOS, days, median (IQR)	3 (2–6)	5 (3–9)	2 (1–3)	<0.001
Renal replacement therapy, n (%)	8 (3.0)	8 (7.3)	0 (0)	<0.001
In-hospital mortality, n (%)	14 (5.2)	14 (12.7)	0 (0)	<0.001
Renal recovery at discharge, n (%) *	72 (65.5)	72 (65.5)	N/A	-
MAKE30, n (%)	31 (11.6)	31 (28.2)	0 (0)	<0.001

LOS, length of stay; ICU, intensive care unit; MAKE30, major adverse kidney events at 30 days (death, renal replacement therapy, or persistent kidney dysfunction); IQR, interquartile range.
Among surviving AKI patients (n=96).

Predictors of Acute Kidney Injury- Results of univariable and multivariable logistic regression analyses for predictors of AKI are presented in Table 3. In univariable analysis, multiple demographic, clinical, and laboratory variables were associated

with increased risk of AKI. After multivariable adjustment, six variables remained independently associated with AKI development.

Table 3. Univariable and Multivariable Logistic Regression Analysis for Predictors of AKI

Variable	Univariable OR (95% CI)	P-Value	Multivariable aOR (95% Ci)	P-Value
Age (per 10-year increase)	1.26 (1.07–1.48)	0.005	1.18 (0.97–1.43)	0.10
Black race	1.82 (1.17–2.83)	0.008	2.04 (1.24–3.35)	0.005
Diabetes mellitus	1.61 (1.02–2.54)	0.04	1.42 (0.85–2.38)	0.18
Pre-existing CKD	3.78 (2.21–6.47)	<0.001	1.88 (0.96–3.68)	0.07
Admission SBP (per 10 mmHg)	1.16 (1.02–1.32)	0.02	1.09 (0.94–1.26)	0.24
SBP variability >25%	2.58 (1.64–4.06)	<0.001	2.24 (1.37–3.66)	0.001
Heart rate (per 10 bpm)	1.16 (1.02–1.31)	0.02	1.09 (0.95–1.25)	0.22
Admission creatinine >1.1 mg/dL	5.84 (3.63–9.40)	<0.001	3.91 (2.38–6.42)	<0.001
Cystatin C >1.2 mg/L	4.42 (2.78–7.02)	<0.001	1.58 (0.87–2.87)	0.13
hs-Troponin I >26 ng/L	3.58 (2.28–5.62)	<0.001	2.72 (1.68–4.41)	<0.001
TyG index ≥9.2	3.32 (2.12–5.20)	<0.001	2.38 (1.46–3.88)	<0.001
Proteinuria	3.78 (2.36–6.05)	<0.001	1.68 (0.98–2.88)	0.06

AKI, acute kidney injury; CKD, chronic kidney disease; SBP, systolic blood pressure; hs-Troponin I, high-sensitivity cardiac troponin I; TyG, triglyceride-glucose; OR, odds ratio; aOR, adjusted odds ratio; CI, confidence interval.

Admission creatinine >1.1 mg/dL demonstrated the strongest independent association with AKI (adjusted odds ratio 3.91, 95% CI 2.38–6.42, p<0.001). Elevated high-sensitivity troponin I (>26 ng/L) was independently associated with more than 2.5-fold increased odds of AKI (aOR 2.72, 95% CI 1.68–4.41, p<0.001). Triglyceride-glucose index ≥9.2, representing the upper tertile in this cohort, conferred an independent 2.4-fold increased risk (aOR 2.38, 95% CI 1.46–3.88, p<0.001). Excessive blood pressure variability during initial management, defined as coefficient of variation >25% for systolic blood pressure measurements during the first six hours, was associated with significantly increased AKI risk (aOR 2.24, 95% CI 1.37–3.66, p=0.001). The Black race remained independently associated with AKI after adjustment for other risk factors (aOR 2.04, 95% CI 1.24–3.35, p=0.005). Pre- existing chronic kidney disease, cystatin C elevation, and proteinuria demonstrated trends toward independent association but did not reach statistical significance in the fully adjusted model, likely reflecting collinearity with admission creatinine and other biomarkers.

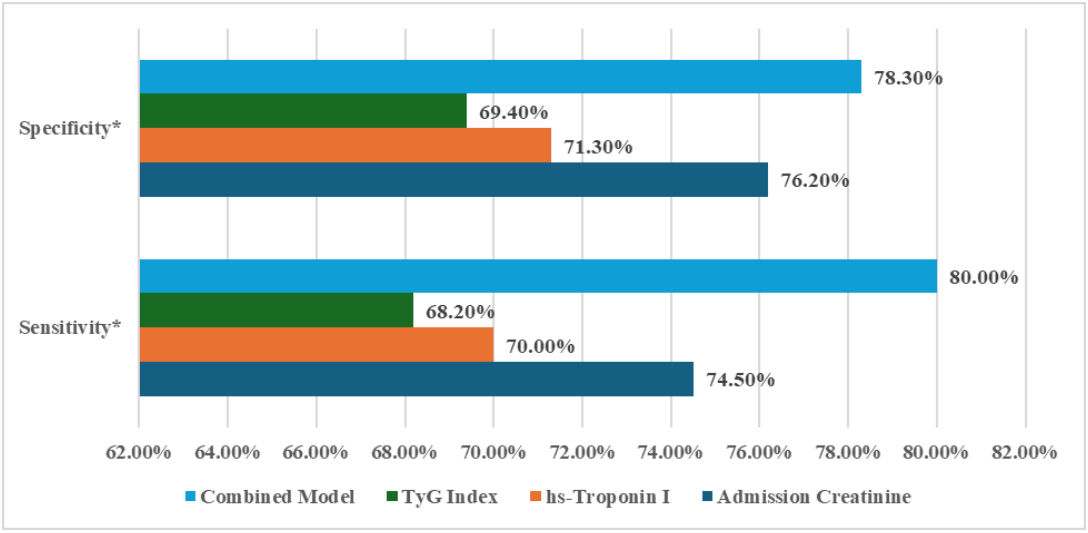

Figure 2: Comparative Performance of Individual Biomarkers and the Combined Model for Predicting Acute Kidney Injury

Note: It is common to use a dual-panel chart to compare Sensitivity and Specificity. The title above effectively covers the content of both panels.

Interpretation: This bar chart compares the sensitivity and specificity of three individual biomarkers, the triglyceride-glucose (TyG) index, high-sensitivity troponin I, and admission creatinine, against the combined multivariable prediction model derived in this study. The combined model demonstrates the highest performance, with a sensitivity of 80.0% and a specificity of 78.3%, underscoring its utility as a balanced and accurate tool for early risk stratification. Among the individual biomarkers, admission creatinine shows the highest specificity (76.2%), confirming its strong association with renal dysfunction at presentation. In contrast, high-sensitivity troponin I exhibits a more balanced profile (sensitivity 70.0%, specificity 71.3%), reflecting its role as a marker of systemic microvascular injury. The TyG index, while a significant independent predictor in the regression analysis, shows the lowest individual performance (sensitivity 68.2%, specificity 69.4%). This suggests that while insulin resistance contributes to AKI risk, its predictive power is enhanced when integrated with other clinical variables in the combined model. These findings support a multi-marker approach for the earliest and most accurate identification of patients at high risk for AKI following a hypertensive emergency.

Table 4. Simplified Risk Score for AKI Prediction in Hypertensive Emergency

Risk Factor		**Points**
Admission creatinine >1.1 mg/dL		3
hs-Troponin I >26 ng/L		2
TyG index ≥9.2		2
SBP variability >25%		2
Black race		1
Risk Category	**Total Points**	**AKI Incidence (%)**
Low	0–2	11.8
Intermediate	3–5	42.3
High	6–10	79.4

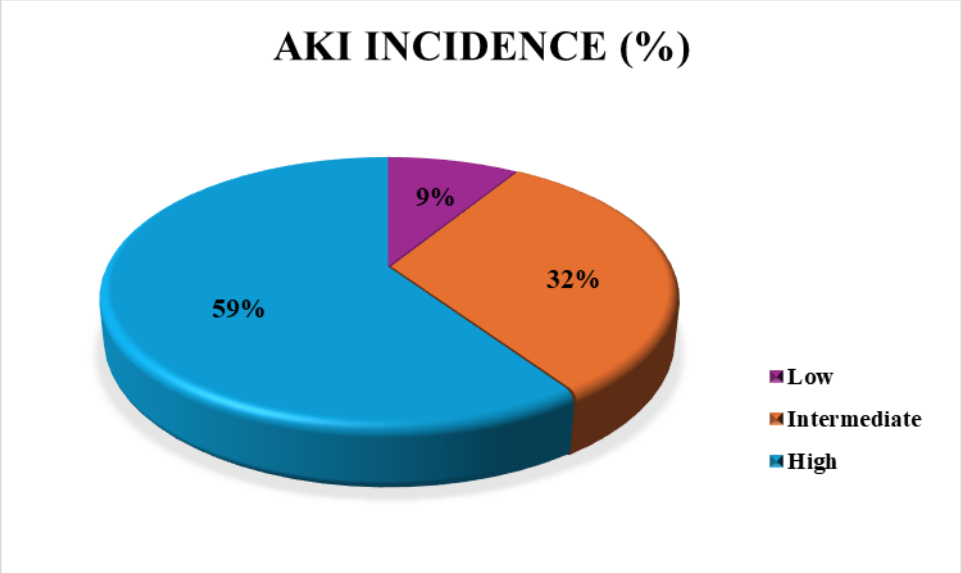

Figure 3: Distribution of Patients by AKI Risk Category

Interpretation- This pie chart illustrates the distribution of the 267 patients across the three risk categories defined by the simplified AKI prediction score. Most patients presenting with hypertensive emergency, 59% (n=158), were classified into the high-risk category (score 6–10). One-third of patients (32%, n=85) fell into the low-risk category (score 0–2), while the smallest proportion, 9% (n=24), were in the intermediate-risk group (score 3–5). This distribution highlights that a significant majority of the cohort possesses multiple risk factors and is therefore at elevated risk for AKI, justifying the need for intensive monitoring and early intervention in most patients. The small size of the intermediate group suggests that the score effectively polarises patients into distinct low- and

high-risk cohorts, thereby streamlining clinical decision-making for triage and resource allocation.

Subgroup and Sensitivity Analyses- The model's predictive performance remained consistent across prespecified subgroups. In patients without pre-existing chronic kidney disease (n=200), the model maintained good discrimination (AUC 0.83, 95% CI 0.78–0.88). Similarly, in patients with diabetes mellitus (n=99) and those without (n=168), AUC values were 0.84 and 0.85, respectively. Sensitivity analyses excluding patients with missing data (n=8) and using multiple imputations yielded virtually identical results.

DISCUSSION

In this prospective observational study of 267 patients presenting with hypertensive emergency, we found that acute kidney injury complicates more than 40% of cases, with one-fifth of affected patients developing moderate-to-severe (KDIGO stage 2–3) AKI. The presence of AKI was associated with substantially longer hospitalisations, higher intensive care unit admission rates, and significantly higher in-hospital mortality compared to patients without renal involvement. We identified five readily available clinical and laboratory parameters: admission creatinine, elevated high-sensitivity troponin I, triglyceride-glucose index, excessive blood pressure variability during initial management, and Black race that independently predicted AKI development and enabled accurate risk stratification. The 41.2% incidence of AKI observed in our cohort aligns with the upper range of previously reported estimates but exceeds the 20–30% incidence reported in some older series [1,5]. Several factors may explain this higher detection rate. First, we employed the more sensitive KDIGO 2024 criteria, which capture smaller creatinine increments and incorporate urine output criteria more systematically than earlier definitions [16f]. Second, our prospective design with systematic daily creatinine measurement minimised ascertainment bias that may affect retrospective analyses. Third, our tertiary care setting, with its high acuity, may have selected patients with more severe presentations. Importantly, most AKI cases (60.0%) were present on admission, suggesting that renal injury often precedes or coincides with presentation rather than resulting from hospital management. This observation underscores the importance of early recognition and intervention.

The strong independent association between elevated high-sensitivity troponin I and AKI extend previous observations linking cardiac and renal injury in hypertensive emergencies. Troponin elevation in this context likely reflects multiple mechanisms: increased myocardial wall stress from acute afterload elevation, subendocardial ischemia in the setting of left ventricular hypertrophy and elevated diastolic pressures, and systemic microvascular injury that simultaneously affects coronary and renal circulations. Our findings suggest that troponin elevation identifies patients with more severe systemic vascular injury who warrant particularly close renal monitoring and careful blood pressure titration. Whether troponin-guided management strategies could improve renal outcomes warrants prospective investigation. The triglyceride-glucose index emerged as a novel independent predictor of AKI in our cohort [14,20,21]. This simple biomarker, derived from routine fasting lipid and glucose measurements, reflects underlying insulin resistance and has been associated with adverse renal outcomes in various critical care settings. In a hypertensive emergency, insulin resistance may contribute to endothelial dysfunction, impaired microvascular reactivity, and heightened susceptibility to hemodynamic injury. The TyG index may also identify patients with underlying metabolic syndrome who have a greater burden of subclinical microvascular disease and reduced renal functional reserve. Its independent predictive value beyond traditional risk factors supports its inclusion in routine risk assessment, particularly given its low cost and widespread availability.

Excessive blood pressure variability during initial management independently predicted AKI even after adjustment for admission severity and other risk factors [5,6]. This finding aligns with emerging evidence that the pattern of blood pressure reduction may be as important as its magnitude. The renal microcirculation in hypertensive emergency exists in a state of impaired autoregulation due to vascular injury and elevated afterload. Rapid or fluctuating pressure reduction may precipitate episodes of renal hypoperfusion that exacerbate ischemic injury, particularly in kidneys with pre-existing microvascular disease. Our results support recent guideline recommendations favouring controlled, gradual blood pressure reduction in hypertensive emergencies and suggest that minimising blood pressure variability should be a specific therapeutic target [16,23]. The independent association between Black race and AKI risk, persisting after adjustment for other risk factors, merits careful consideration [1,22]. This observation is consistent with large database studies demonstrating higher rates of hypertensive emergency and associated end-organ damage among Black patients [1]. Potential explanations include higher prevalence of treatment-resistant hypertension, greater burden of subclinical microvascular disease, genetic factors influencing renal susceptibility to hypertensive injury, and social determinants of health, including healthcare access and medication adherence. While our study cannot fully disentangle these complex factors, the finding

underscores the need for targeted interventions and enhanced monitoring in this high-risk population.

Admission creatinine demonstrated the strongest individual association with AKI, reflecting both pre-existing chronic kidney disease and early acute injury present at the time of presentation [10,11,17]. The modest independent effect of pre-existing CKD after adjustment for admission creatinine suggests that the absolute creatinine value captures much of the relevant risk information. However, the trend toward independent association between proteinuria and cystatin C suggests that these markers may provide complementary information about underlying renal structural damage and functional reserve, warranting further investigation [17,18,19]. The simplified risk score developed from our data enables rapid bedside stratification of AKI risk using routinely available parameters. Patients in the low-risk category (0–2 points) had an AKI incidence of only 11.8%, suggesting that intensive care unit admission and invasive monitoring may not be routinely necessary in this group if other indications are absent. Conversely, high-risk patients (6–10 points) had nearly 80% AKI incidence, justifying intensive monitoring, careful fluid management, and possibly early nephrology consultation. Intermediate-risk patients (3–5 points) with a 42% AKI incidence represent a group in which heightened vigilance and individualised decision-making are appropriate. Prospective validation of this score in independent cohorts is needed before widespread clinical implementation [15].

Strengths of the Study- This study possesses several methodological strengths that enhance the validity and reliability of its findings. The prospective design with systematic data collection minimised ascertainment bias and enabled accurate capture of AKI timing and severity, which is often missed in retrospective analyses. Use of contemporary KDIGO 2024 criteria ensures relevance to current clinical practice and aligns with the most up-to-date diagnostic standards. Comprehensive assessment of multiple biomarkers allowed direct comparison of their predictive utility within the same cohort, providing insights into their relative performance. The racially diverse cohort, with 50% Black representation, enhances generalizability to populations at highest risk for hypertensive emergency and its complications. Rigorous statistical methods, including thorough adjustment for potential confounders and assessment of model calibration and discrimination, support the robustness of our predictive model. The simplified risk score derived from regression coefficients provides a clinically practical tool that can be readily implemented at the bedside without computational assistance.

Limitations- Several limitations warrant acknowledgement when interpreting our findings. First, the single-centre design may limit generalizability, although our centre serves a diverse urban population that likely reflects broader patient mixes encountered in tertiary care settings. Second, we could not definitively establish baseline renal function for all patients, and some patients classified as having AKI on admission may have unrecognised chronic kidney disease. However, sensitivity analyses excluding patients without documented baseline values yielded similar results, and we used multiple imputations to address missing data. Third, the urine output criteria for AKI definition could not be systematically applied in all patients, particularly those in non- intensive care settings, potentially leading to underestimation of AKI incidence. Fourth, we assessed blood pressure variability only during the first six hours; longer-term variability patterns beyond this initial period may provide additional predictive information not captured in our analysis. Fifth, we did not systematically measure novel urinary biomarkers such as neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, or interleukin-18, which might enhance early detection and risk stratification. Sixth, the observational design cannot establish causality between identified predictors and AKI, and residual confounding may exist despite multivariable adjustment for multiple potential confounders. Seventh, the sample size, while adequate for the primary analysis, limited our ability to perform extensive subgroup analyses or to assess interactions between predictors. Eighth, the risk score, while internally valid, requires external validation in independent cohorts before widespread clinical implementation.

Clinical Implications- Our findings have several important implications for clinical practice. First, the high incidence of AKI in hypertensive emergencies mandates systematic renal function assessment in all patients, including those without overt renal symptoms at presentation. Daily creatinine monitoring should be considered standard of care for at least the first 72 hours of hospitalisation. Second, the independent predictive value of troponin and the TyG index supports their routine measurement as part of initial risk stratification in the emergency department, providing additional prognostic information beyond traditional risk factors. Third, the association between excessive blood pressure variability and AKI suggests that hemodynamic optimisation should prioritise smooth, controlled pressure reduction over rapid achievement of specific numeric targets. This may influence the choice of antihypertensive agents and titration protocols. Fourth, the heightened risk in Black patients calls for enhanced monitoring and consideration of social and structural factors that may influence outcomes, including assessment of medication adherence and

healthcare access barriers. Fifth, the simplified risk score may facilitate triage decisions and resource allocation, helping to identify patients who warrant intensive care unit admission versus those who can be safely managed in intermediate care or telemetry settings. Sixth, the high rate of renal non-recovery among patients with stage 2–3 AKI supports early nephrology consultation and close post-discharge follow-up for this subgroup.

Future Research Recommendations- These findings generate several questions warranting further investigation. Prospective multicenter studies should validate our risk prediction model across diverse settings and populations, including community hospitals and international cohorts with varying demographic compositions. Randomised controlled trials could evaluate whether risk-stratified management strategies improve outcomes compared to uniform approaches, potentially randomising intermediate-risk patients to different levels of monitoring intensity. The optimal blood pressure reduction strategy to minimise renal injury while achieving cardiovascular protection requires further study, with careful assessment of both mean pressure reduction and variability. Comparative effectiveness research should examine whether specific antihypertensive agents (e.g., nicardipine, labetalol, or clevidipine) differentially affect renal outcomes through their hemodynamic effects. The role of interventions targeting insulin resistance or endothelial function in modifying AKI risk among patients with elevated TyG index remains speculative but intriguing and warrants exploration in pilot studies. Integration of novel urinary biomarkers with clinical risk factors may enable even earlier identification of patients at imminent risk for progressive renal injury, potentially allowing preventive interventions before creatinine rises. Long-term follow-up studies should examine whether AKI during hypertensive emergency accelerates the progression of chronic kidney disease or increases the risk of cardiovascular events beyond the index hospitalisation. Finally, implementation science research should evaluate strategies to promote adoption of evidence-based AKI prevention practices in emergency department and intensive care unit settings.

CONCLUSION

Acute kidney injury complicates more than 40% of hypertensive emergency presentations and is associated with substantially increased morbidity, mortality, and healthcare resource utilisation. Routine clinical and laboratory parameters available at the time of presentation, including admission creatinine, high-sensitivity cardiac troponin I, triglyceride-glucose index, blood pressure variability during initial management, and Black race, enable accurate early identification of patients at the highest risk. These findings support systematic risk stratification in hypertensive emergencies to guide monitoring intensity, therapeutic decision-making, and resource allocation. Prospective validation of integrated risk prediction models in multicenter cohorts and investigation of targeted preventive strategies represent important next steps toward improving renal and cardiovascular outcomes in this high-risk population.

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