e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com doi: 10.25258/ijpqa.16.12.132

International Journal of Pharmaceutical Quality Assurance 2025; 16(12); 828-834

Original Research Article

Association Between High-Sensitivity Troponin Levels and In-Hospital Outcomes in Hypertensive Emergencies

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Received: 12-10-2025 / Revised: 24-11-2025 / Accepted: 28-12-2025

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Conflict of interest: Nil

Abstract:

Background: "Hypertensive emergencies are acute, severe elevations in blood pressure accompanied by target organ damage, often leading to significant morbidity and mortality. High-sensitivity cardiac troponin (hs-cTn) has emerged as a biomarker for detecting myocardial injury and assessing prognosis in these patients.

Aim: To evaluate the prognostic significance of hs-cTn levels in patients presenting with hypertensive emergencies.

Methodology: This hospital-based observational study included 80 adult patients with hypertensive emergencies admitted to the Department of General Medicine, Patna Medical College and Hospital, India. Patients were stratified into normal and elevated hs-cTn groups. Clinical parameters, target organ damage, and in-hospital outcomes were recorded. ROC curve analysis assessed hs-cTn's predictive value for mortality.

Results: Patients with elevated hs-cTn were older, had higher prevalence of diabetes, longer hypertension duration, and more severe hemodynamic and renal impairment. They exhibited significantly greater target organ damage, including acute heart failure (45% vs. 15%), acute kidney injury (50% vs. 20%), hypertensive encephalopathy (35% vs. 12.5%), and advanced retinopathy (40% vs. 17.5%). Elevated hs-cTn was associated with longer hospital stay, increased ICU admission, mechanical ventilation, and higher in-hospital mortality (25% vs. 5%). ROC analysis showed AUC of 0.82, with 83.3% sensitivity and 75% specificity for predicting mortality.

Conclusion: Elevated hs-cTn is a strong prognostic marker in hypertensive emergencies, enabling early risk stratification and guiding intensive management.

Keywords: Hypertensive emergency, high-sensitivity troponin, myocardial injury, prognostic marker, in-hospital outcomes.

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Introduction

Hypertensive emergencies are a critical subgroup of acute hypertension that involves severe hypertension with the presence of acute damage of target organs [1]. These conditions, which can be heart, brain, kidney or vascular system, require early" identification and treatment to avert irreversible "morbidity and mortality. Although there has been an improvement in antihypertensive treatment and critical care management, the hypertensive emergencies remain a significant clinical challenge in that of their diverse manifestations and the unpredictability of outcomes. One of the most severe consequences is cardiovascular complications, especially acute myocardial injury and heart failure [2]. In this regard, cardiac biomarkers have been considered useful not only in the diagnosis but also as a risk stratifying tool. Of these, high-sensitivity cardiac troponin (hs-cTn) has received much interest due to its capacity to identify even low levels of myocardial damage and cast some significant doubts over its prognostic value in hypertensive crises.

The cardiac troponins comprise troponin I, and troponin T, are regulatory proteins that form part of the contractile apparatus of myocardial cells [3]. Conventionally, troponin tests were mostly applied in the diagnosis of acute myocardial infarction. Nevertheless, with the introduction of high-sensitivity assays, due to their capability to produce low-detection limits, the clinical practice has been transformed to allow the detection of extremely low levels of circulating troponin, which are usually lower than the conventional assays. In a broad array of clinical conditions beyond acute coronary syndromes, e.g., sepsis, pulmonary embolism, chronic kidney disease and hypertensive crises, high sensitivity troponin assays, including hs-cTnI and hs-cTnT may be used to detect myocardial injury [4]. These assays have increased analytical sensitivity enabling the earlier detection of myocardial damage and more precise risk stratification.

Hypertensive crisis is considered as sudden and prolonged increases in systemic vascular resistance, which results in augmented afterload and heart demand of oxygen [5]. Subclinical or evident myocardial injury may be triggered by the mismatch between the oxygen demand and the supply, microvascular dysfunction, and endothelial injury. This is even without the presence of obstructive coronary artery disease. In addition, structural cardiac alterations as left ventricular hypertrophy and diastolic dysfunction frequently occur in the presence of long-diagnosed hypertension and expose the myocardium to ischemic damages during acute hypertensive spikes [6]. High levels of" high-sensitivity troponin in this context could indicate the presence of continuing myocardial "stress, necrosis, or both.

Clinical meaning of hs-cTn increase during hypertensive emergencies is complicated [7]. Although a distinctly high troponin level can be a sign of type 1 myocardial infarction caused by the rupture of the plaque, less significant increases are often referred to as type 2 myocardial infarction or non-ischemic myocardial injury caused by the hemodynamic stress. These entities should be distinguished since the management strategies are very different. Still, despite the underlying mechanism, many studies have shown that even minor high-sensitivity troponin increase is linked to adverse outcomes, such as longer hospital stay, higher in-hospital mortality, and major adverse cardiovascular events rates [8].

Prognosis hs-cTn is a quantitative indicator of the myocardial injury burden. High levels of hs-cTn have been associated with an increased risk of acute heart failure, arrhythmias, and intensive care requirements in patients with hypertensive crises, who required intensive care support in the EDs of the involved hospital [9]. In addition, hs-cTn serial measurements can present more prognostic data because they reveal the dynamic changes that could be indicative of continuing injury. Constant or increasing troponin levels in the hospital have been associated with poor long and short-term outcomes. Therefore, hs-cTn not only assists in the clarification of the diagnosis but also provides an incremental prognostic in addition to the conventional clinical features, including the level of the blood pressure, renal functioning, and radiographic finding.

Notably, the prognostic value of hs-cTn in hypertensive crises is not limited to direct hospitalization. High levels have been linked to higher risks of readmission, chronic heart failure development and subsequent cardiovascular mortality. This is an indication that hs-cTn can help define a group of patients who might be having underlying subclinical coronary artery disease or advanced hypertensive heart disease and need additional intensive secondary prevention measures. The inclusion of hs-cTn into risk assessment models can thus help in improving clinical decision-making and supporting the customization of therapeutic interventions.

Even though it has a clinical application, there are a number of issues. Patients with comorbid conditions such as chronic kidney disease may have baseline levels of troponin that are chronic and difficult to interpret. Also, the best values of cutoff to be used in prognostication of hypertensive emergencies have not universally been standardized. More studies should be done to come up with clear diagnostic guidelines and to establish whether the troponin-guided management practices can serve as an effective approach to improve the outcomes.

In summary, high-sensitivity troponin has become an important biomarker used in the assessment of emergencies of" hypertension. Hs-cTn allows identifying minor myocardial damage, and this can be used as highly valuable prognostic data and utilized in "risk stratification, clinical management, and can possibly improve patient outcomes. The necessity to learn how it fits in the context of the overall pathophysiological process of hypertensive emergencies is crucial to maximize care and minimize the cardiovascular complication burden posed by this dangerous disease.

Methodology

Study Design: This study was conducted as a hospital-based observational analytical study to evaluate the prognostic significance of high-sensitivity troponin (hs-cTn) levels in patients presenting with hypertensive emergencies. The design aimed to assess the association between elevated hs-cTn levels at admission and short-term clinical outcomes, including in-hospital mortality, requirement of intensive care, duration of hospital stay, and occurrence of major adverse cardiovascular events.

Study Area: The study was carried out in the Department of General Medicine, Patna Medical College and Hospital, Patna, Bihar, India.

Study Duration: The study was conducted over a period of 8 months from February 2025 to September 2025

Study Participants: The study population consisted of adult patients presenting with hypertensive emergencies to the emergency department and medical wards of PMCH.

Inclusion Criteria

- Patients aged ≥18 years.
- Patients diagnosed with hypertensive emergency (systolic blood pressure ≥180 mmHg and/or diastolic blood pressure ≥120 mmHg) with evidence of acute target organ damage.
- Patients willing to provide" informed written consent.
- Patients in whom "high-sensitivity cardiac troponin (hs-cTn) testing was performed at admission.

Exclusion Criteria

- Patients diagnosed with acute coronary syndrome (STEMI/NSTEMI).
- Known cases of obstructive coronary artery disease.
- Patients with chronic kidney disease (Stage 4 or 5).
- Patients with pulmonary embolism, acute myocarditis, or pericarditis.
- Patients with severe valvular heart disease (e.g., severe aortic stenosis).
- Pregnant women.
- Patients with sepsis or systemic inflammatory conditions known to elevate troponin levels independently.

Sample Size: The total sample size for the study was 80 patients fulfilling the inclusion and exclusion criteria.

Procedure: All eligible patients presenting with hypertensive emergency were admitted through the emergency department. After obtaining informed written consent, detailed clinical history and physical examination were performed. Blood pressure was recorded using a standardized sphygmomanometer at presentation. Routine laboratory investigations, including complete blood count, renal function tests, blood glucose, serum electrolytes, and lipid profile, were conducted as part of standard care. High-sensitivity cardiac troponin (hs-cTn) levels were measured at admission using standardized immunoassay techniques available in the hospital laboratory.

Electrocardiography (ECG) was performed for all patients to assess cardiac rhythm and ischemic changes. Two-dimensional echocardiography (ECHO) was conducted to evaluate left ventricular function and detect structural or functional abnormalities. Left ventricular ejection fraction (LVEF) was assessed by the attending cardiologist. Evidence of target organ damage such as hypertensive encephalopathy, acute heart failure, acute kidney injury, or retinal changes was documented.

Based on hs-cTn levels, patients were stratified into two groups: normal hs-cTn group and elevated hs-cTn group. Patients were followed during their" hospital stay to record clinical outcomes including duration of hospitalization, development of complications, and in-hospital mortality. No additional "financial burden was imposed on the patients, as all investigations were part of routine management. Ethical approval was obtained from the Institutional Ethics Committee of PMCH prior to commencement of the study. Confidentiality and anonymity of all participants were strictly maintained throughout the study.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 27.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage. The comparison of continuous variables between the two groups (normal vs elevated hs-cTn) was performed using the student's independent t-test for normally distributed data. Categorical variables were analyzed using the Chi-square test or Fisher's exact test as appropriate. Receiver Operating Characteristic (ROC) curve analysis was performed to assess the predictive value of hs-cTn for in-hospital mortality. A p-value of <0.05 was considered statistically significant.

Result

Table 1 shows the baseline demographic characteristics of the study participants (n = 80) divided equally into normal and elevated hs-cTn groups. The mean age was significantly higher in the elevated hs-cTn group (62.8 ± 9.5 years) compared to the normal hs-cTn group (54.6 ± 10.2 years) (p = 0.001), indicating that older patients were more likely to have elevated hs-cTn levels. The proportion of males was higher in the elevated group (65%) than in the normal group (55%), but this difference was not statistically significant (p = 0.36). Diabetes mellitus was significantly more prevalent in the elevated hs-cTn group (52.5%) compared to the normal group (30%) (p = 0.04). Similarly, a longer duration of hypertension (>5 years) was significantly associated with elevated hs-cTn levels (60% vs. 37.5%, p = 0.04). Although smoking was more common in the elevated group (42.5%) than in the normal group (25%), the difference did not reach statistical significance (p = 0.09). Overall, older age, diabetes, and longer hypertension duration were significantly associated with elevated hs-cTn levels.

Table 1: Baseline Demographic Characteristics of Study Participants (n = 80)

Variable	Normal hs-cTn (n=40)	Elevated hs-cTn (n=40)	p-value
Age (years), Mean ± SD	54.6 ± 10.2	62.8 ± 9.5	0.001
Male, n (%)	22 (55%)	26 (65%)	0.36
Diabetes Mellitus, n (%)	12 (30%)	21 (52.5%)	0.04
Hypertension duration >5 yrs, n (%)	15 (37.5%)	24 (60%)	0.04
Smoking, n (%)	10 (25%)	17 (42.5%)	0.09

Table 2 shows the comparison of clinical and hemodynamic parameters at admission between patients with normal and elevated hs-cTn levels (n=40 each). Patients in the elevated hs-cTn group had significantly higher systolic blood pressure (194.8 ± 14.1 mmHg vs. 186.4 ± 12.3 mmHg; p=0.006) and diastolic blood pressure (118.3 ± 10.5 mmHg vs. 112.6 ± 8.9 mmHg; p=0.01) compared to the normal hs-cTn group. Similarly, heart rate was significantly higher in the elevated hs-cTn group (98.6 ± 13.2 bpm vs. 88.2 ± 11.4 bpm; p=0.001). Serum creatinine levels were also significantly increased among patients with elevated hs-cTn (1.8 ± 0.6 mg/dL vs. 1.3 ± 0.4 mg/dL; p=0.001), indicating greater renal dysfunction. In contrast, left ventricular ejection fraction (LVEF) was significantly lower in the elevated hs-cTn group (47.2 ± 8.5% vs. 55.4 ± 6.8%; p=0.001). Overall, elevated hs-cTn was associated with more severe hemodynamic compromise, renal impairment, and reduced cardiac function at admission.

Table 2: Clinical and Hemodynamic Parameters at Admission

Parameter	Normal hs-cTn (n=40)	Elevated hs-cTn (n=40)	p-value
Systolic BP (mmHg), Mean ± SD	186.4 ± 12.3	194.8 ± 14.1	0.006
Diastolic BP (mmHg), Mean ± SD	112.6 ± 8.9	118.3 ± 10.5	0.01
Heart Rate (bpm), Mean ± SD	88.2 ± 11.4	98.6 ± 13.2	0.001
Serum Creatinine (mg/dL), Mean ± SD	1.3 ± 0.4	1.8 ± 0.6	0.001
LVEF (%), Mean ± SD	55.4 ± 6.8	47.2 ± 8.5	0.001

Table 3 shows the distribution of target organ damage among study participants according to hs-cTn levels. Patients with elevated hs-cTn demonstrated a significantly higher frequency of all forms of organ damage compared to those with normal hs-cTn levels. Acute heart failure was observed in 45% of patients in the elevated group versus 15% in the normal group (p=0.003). Similarly, acute kidney injury occurred in 50% of patients with elevated hs-cTn compared to 20% in the normal group (p=0.004). Hypertensive encephalopathy was noted in 35% of the elevated group versus 12.5% of the normal group (p=0.02), while Grade III/IV retinopathy was present in 40% compared to 17.5% respectively (p=0.03). These findings indicate a significant association between elevated hs-cTn levels and increased incidence of target organ damage in hypertensive emergencies.

Table 3: Target Organ Damage Among Study Participants

Target Organ Damage	Normal hs-cTn (n=40)	Elevated hs-cTn (n=40)	p-value
Acute Heart Failure	6 (15%)	18 (45%)	0.003
Acute Kidney Injury	8 (20%)	20 (50%)	0.004
Hypertensive Encephalopathy	5 (12.5%)	14 (35%)	0.02
Retinopathy (Grade III/IV)	7 (17.5%)	16 (40%)	0.03

Table 4 shows the comparison of in-hospital outcomes between patients with normal and elevated hs-cTn levels (n = 40 each group). The mean duration of hospital stay was significantly longer in the elevated hs-cTn group (7.9 ± 2.4 days) compared to the normal hs-cTn group (4.6 ± 1.8 days), with a statistically significant difference (p = 0.001). ICU admission was markedly higher among patients with elevated hs-cTn (42.5%) than those with normal levels (12.5%), which was also statistically significant (p = 0.002). Similarly, the requirement for mechanical ventilation was greater in the elevated hs-cTn group (30%) compared to the normal group (7.5%) (p = 0.01). In-hospital mortality was significantly higher in patients with elevated hs-cTn (25%) than in those with normal hs-cTn levels (5%) (p = 0.01). Overall, elevated hs-cTn was strongly associated with poorer in-hospital outcomes.

Table 4: In-Hospital Outcomes

Outcome	Normal hs-cTn (n=40)	Elevated hs-cTn (n=40)	p-value
Duration of Hospital Stay (days), Mean ± SD	4.6 ± 1.8	7.9 ± 2.4	0.001
ICU Admission	5 (12.5%)	17 (42.5%)	0.002
Mechanical Ventilation	3 (7.5%)	12 (30%)	0.01
In-Hospital Mortality	2 (5%)	10 (25%)	0.01

Table 5 demonstrates the diagnostic performance of high-sensitivity cardiac troponin (hs-cTn) in predicting in-hospital mortality. The area under the ROC curve (AUC) was 0.82, indicating good discriminative ability of hs-cTn for mortality prediction. The 95% confidence interval (0.71–0.93) further supports the reliability and precision of this finding. An optimal cut-off value of 42.5 ng/L was identified, which yielded a sensitivity of 83.30% and specificity of 75.00%, suggesting that hs-cTn has high sensitivity and acceptable specificity in detecting patients at risk of in-hospital death. The association was statistically significant (p = 0.001), confirming that elevated hs-cTn levels are a strong and significant predictor of in-hospital mortality in the study population.

Table 5: ROC Curve Analysis of hs-cTn for Predicting In-Hospital Mortality

Parameter	Value
Area Under Curve (AUC)	0.82
95% Confidence Interval	0.71 – 0.93
Optimal Cut-off Value (ng/L)	42.5
Sensitivity (%)	83.30%
Specificity (%)	75.00%
p-value	0.001

Discussion

The study shows that high levels of high-sensitivity cardiac troponin (hs-cTn) which occur during hypertensive emergencies (HE) lead to increased risk of serious medical conditions and damage to vital body systems and reduced medical results during hospitalization. Our findings are consistent with previous observations by Afonso L et al. (2011) [10], who reported that approximately one-third of patients admitted with hypertensive emergencies had elevated cardiac troponin I (cTnI), whereas our cohort showed an even higher proportion of troponin elevation, suggesting that myocardial injury is common in severe hypertensive states. The higher prevalence in our study may reflect differences in patient profile, severity of blood pressure elevation, and the use of high-sensitivity assays capable of detecting minor myocardial injury.

Age emerged as a significant determinant of troponin elevation in our study, with older patients demonstrating a higher likelihood of elevated hs-cTn. these findings confirm the research results of Lee KK et al. (2019) [11] which showed that emergency department patients experience increased hs-cTn levels because of their growing age. However, in contrast, AlQassas I et al. (2019) [12] observed no significant association between age and troponin elevation in non-cardiac medical disorders. The difference happens because hypertensive emergencies create particular hemodynamic stress patterns which cause advanced age to increase myocardial vulnerability through arterial stiffness and subclinical structural heart disease.

The study found that diabetes mellitus and hypertension duration beyond five years led to higher hs-cTn levels which confirmed that chronic microvascular dysfunction together with myocardial remodeling created a vulnerability to damage during sudden blood pressure increases. The findings of our study differ from Mahajan N et al. (2006) [13] who showed that elevated troponin does not always indicate myocardial infarction because it fails to match every medical condition. Our research demonstrates that metabolic comorbidities during hypertensive crises create a supply-demand mismatch which occurs through mechanisms other than plaque rupture.

Our study found that hemodynamic parameters showed significant increases in the elevated hs-cTn group which included both systolic and diastolic blood pressure measurements and heart rate assessments. Alcalai R et al. (2007) [14] showed that patients with nonspecific troponin elevation experienced higher hemodynamic stress markers and more severe survival outcomes than patients without elevation. The average systolic blood pressure in troponin-positive patients from previous studies reached 220 mmHg which matched the severe blood pressure increases seen in our group. The evidence demonstrates that direct myocardial wall stress and acute afterload excess create conditions that result in subendocardial ischemia and troponin release.

Our analysis demonstrated that hs-cTn elevation correlated strongly with renal dysfunction because

patients exhibited elevated serum creatinine levels. The systematic review conducted by Ahmed AN et al. (2014) [15] found that non-cardiac hospitalized patients who showed troponin elevation experienced both kidney impairment and higher risk of mortality. Caujolle et al. (2018) [16] discovered that patients with non-cardiogenic shock who experienced decreased kidney function and elevated inflammatory markers showed higher hs-cTn levels. The parallels which exist between hypertensive emergency troponin elevation and systemic vascular damage show that patients experience multiple organ failure instead of only heart damage from their condition.

Our research demonstrated that patients with hs-cTn levels above the normal range showed higher rates of" left ventricular systolic dysfunction. Khan MH et al. (2019) [17] demonstrated a negative correlation between "troponin I levels and left ventricular ejection fraction ($r = -0.5394$, $p = 0.001$), supporting our observation that higher troponin levels are associated with reduced LVEF. The connection shows how myocardial damage and ventricular dysfunction interact with each other during hypertensive emergencies because sudden pressure increases can cause temporary or lasting systolic dysfunction.

The research found that patients with high hs-cTn levels experienced worse hospital outcomes because they needed longer hospital stays and had higher rates of ICU admissions and required more mechanical ventilation which resulted in five times greater mortality (25% vs. 5%) than normal patients. The results of this study match the findings of Kaura et al. (2019) [18] who showed that positive troponin levels independently increased short-term mortality risk in various acute care settings. The study by Ahmed et al. (2014) showed that elevated troponin levels in non-acute coronary conditions leads to higher mortality rates. The mortality difference between our study groups demonstrates how important hs-cTn testing results are for predicting outcomes in patients who experience hypertensive emergencies.

The ROC curve analysis in our study revealed an AUC of 0.82 for predicting in-hospital mortality which showed good ability to differentiate between outcomes. The performance of our study matches results from previous research which used high-sensitivity troponin assays to predict outcomes in patients with acute cardiovascular conditions because their AUC values fell between 0.75 and 0.85. The identified cut-off value of 42.5 ng/L provides 83.3% sensitivity and 75% specificity which indicates that hs-cTn serves as a reliable early risk assessment tool for hypertensive emergency situations.

Our research results support earlier studies which show that troponin levels rise during hypertensive crises and these elevations have vital predictive value. Elevated hs-cTn levels show combined cardiovascular stress and myocardial strain and organ system failure according to the accumulated research evidence. Thus, hs-cTn measurement should be considered an essential component of early evaluation in hypertensive emergencies for identifying high-risk patients and guiding intensive" management strategies.

Conclusion

The present study demonstrates that high-sensitivity cardiac troponin (hs-cTn) measurements above normal levels serve as accurate prognostic indicators for patients experiencing hypertensive emergencies. The study reveals that patients with elevated hs-cTn levels exist in two dimensions because they are both older and they have two medical conditions which include diabetes and prolonged hypertension and they display more extreme hemodynamic deterioration and kidney impairment and decreased left ventricular performance at the time of their initial assessment. The patients show a pronounced increase in target organ damage which includes acute heart failure and acute kidney injury and hypertensive encephalopathy and advanced retinopathy. The research demonstrated that elevated hs-cTn levels produced negative effects on hospital results which included extended hospital stays and more ICU admissions and increased need for mechanical ventilation and elevated death rates. The ROC analysis established that hs-cTn functions as a reliable predictor of in-hospital mortality which demonstrates its effectiveness as an initial risk assessment method for patients with hypertensive emergencies.

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