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Original Research Article

Diagnostic and Prognostic Significance of High-Sensitivity Cardiac Troponin in Chronic Kidney Disease Patients Presenting with Chest Pain

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Abstract:

Background: Chronic kidney disease (CKD) is associated with a significantly increased risk of cardiovascular disease and acute coronary syndrome (ACS). Interpretation of high-sensitivity cardiac troponin (hs-cTn) levels in CKD patients is often challenging because baseline troponin concentrations may remain elevated even in the absence of acute myocardial injury. Despite this limitation, hs-cTn remains an important biomarker for cardiovascular risk assessment.

Aim: To evaluate the diagnostic and prognostic significance of high-sensitivity cardiac troponin in CKD patients presenting with chest pain.

Methodology: A prospective observational study was conducted over one year in the Department of General Medicine, Netaji Subhas Medical College and Hospital, Amhara, Bihta, Patna, Bihar, India. A total of 95 adult CKD patients who were present with chest pain were enrolled. Clinical evaluation, electrocardiography, echocardiography, renal function assessment, and serial hs-cTn measurements were performed.

Results: Among the 95 patients, 62.1% were males and 46.3% were aged above 60 years. ACS was diagnosed with 43.2% of patients. Elevated hs-cTn levels were significantly associated with advanced CKD stages and ACS diagnosis ($p<0.001$). Patients with higher troponin levels demonstrated increased rates of reduced left ventricular ejection fraction, heart failure (34.3%), arrhythmia (22.9%), ICU admission (42.9%), revascularization (37.1%), mortality (20.0%), and major adverse cardiac events (60.0%).

Conclusion: Elevated hs-cTn is a significant diagnostic and prognostic marker in CKD patients presenting with chest pain and is strongly associated with ACS and adverse cardiovascular outcomes.

Keywords: Chronic Kidney Disease, High-Sensitivity Cardiac Troponin, Acute Coronary Syndrome, Chest Pain, Major Adverse Cardiac Events, and Prognosis.

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Introduction

Chronic kidney disease (CKD) is one of the most prevalent public health issues affecting the world and leading to significant morbidity and mortality, especially from cardiovascular complications. Cardiovascular disease is still the most common cause of death among those with CKD and is a major contributor to hospital admissions and poor outcomes. This population is at a significantly higher cardiovascular risk due to accelerated atherosclerosis, dysfunction of the endothelial cells, vascular calcification, chronic inflammation, oxidative stress and metabolic abnormalities. Thus, acute coronary syndromes (ACS) and acute myocardial infarction (AMI) are much more common in patients with CKD than in healthy individuals [1]. Moreover, these patients have a significantly increased mortality after coronary thrombotic events due to peri-procedural complications, recurrent ischemic events, and complex and technically challenging coronary lesions [2].

Acute myocardial infarction (AMI) is among the leading causes of death and disability in the world. Prompt and early detection and initiation of evidence based therapeutic strategies are critical for achieving long-term cardiovascular outcomes and for improving survival [3]. The burden of CKD in patients who seek care for AMI is significant: about one-third of the patients with ST-segment elevation myocardial infarction (STEMI) and more than 40% of patients with non-ST-segment elevation myocardial infarction (NSTEMI) had underlying CKD [4]. Although the cardiovascular risk is higher in these patients, the diagnosis and management of ACS in

these patients is still very difficult because of atypical presentation of symptoms, pre-existing electrocardiographic abnormalities, and safety concerns of invasive diagnostic and therapeutic procedures [5].

The preferred biomarkers in the diagnosis of myocardial injury and AMI are cardiac troponins, such as cardiac troponin T (cTnT) and cardiac troponin I (cTnI). High sensitivity cardiac troponin assays (hs-cTnT and hs-cTnI) have transformed the diagnosis of acute coronary syndromes (ACS) by allowing reliable detection of very low levels of blood troponins with high analytical precision. Recent studies using high-sensitivity troponin assays have shown that the diagnostic accuracy of the early diagnosis of AMI is > 96% [6]. This has led to these biomarkers becoming the basis of current diagnostic protocols in patients with chest pain and suspected ACS [7].

Interpretation of troponin levels in patients with CKD is, however, often difficult. In patients with CKD, high levels of cardiac troponins are commonly present even without acute myocardial infarction (AMI) [8]. There are a few mechanisms suggested to account for this phenomenon. While the reduced renal clearance is a possible cause of troponin elevation, there is at present little evidence that persistent elevation of troponins is due to chronic subclinical myocardial injury caused by LVH, coronary microvascular disease, myocardial fibrosis, inflammation, volume overload, and repeated episodes of silent myocardial ischemia [9]. These factors can result in chronic low-level release and injury to cardiomyocytes, and thus chronically high baseline troponin levels.

Therefore, diagnosis of ACS in CKD patients with chest pain is more difficult. The clinical course can be atypical, and symptoms like dyspnea, fatigue, nausea, or generalized weakness can be more common than classical chest pain. In CKD patients, left ventricular hypertrophy, conduction abnormalities and electrolyte disturbances are more common, making interpretation of electrocardiograms more difficult [10]. Further, chronic elevation of troponin does not make it more specific as an acute myocardial injury marker, leading to higher rates of false-positive diagnosis and invasive testing. Thus, a persistent increase in troponin can be problematic for the clinician when trying to differentiate it from acute ischemic myocardial injury in patients with severe renal dysfunction.

In this population, other diagnostic tests might be restricted as well. The use of CTA has grown increasingly for the evaluation of CAD and might not be as helpful in patients with CKD due to extensive CAC and concerns about CIN [11]. In the same manner, there is still a lack of utilization of invasive coronary angiography (ICA) in patients with CKD, although evidence indicates that not receiving it leads to poorer outcomes [12].

While high-sensitive cardiac troponin assays have demonstrated a major advance in the diagnosis of myocardial infarction in the general population, there are relatively few studies that have specifically addressed their diagnostic accuracy in patients with moderate to severe CKD [13]. Most of the studies available have focused on patients with relatively good renal function, with a median estimated GFR (eGFR) of > 60 mL/min/1.73 m², limiting the applicability of the results to advanced CKD [14]. In addition, there is a lack of representation or exclusion of patients with CKD stages G4 and G5, including those undergoing maintenance dialysis, in major diagnostic studies. Therefore, optimal cutoffs and prognostic importance of high-sensitivity cardiac troponin are not fully known in this high-risk population.

Because of the rising incidence of CKD and the high burden of cardiovascular disease in this population, a greater understanding of the diagnostic and prognostic value of high-sensitivity cardiac troponin in the context of chest pain in these patients is needed. Correct interpretation of troponin level can help to expedite early diagnosis of ACS, risk stratification, timely therapeutic treatment and provide better clinical outcome. The present study, undertaken in General Medicine Department Amhara hospital, Bihta Patna, Bihar, therefore plans to evaluate the diagnostic and prognostic value of highly sensitive cardiac troponin levels in patients of chronic kidney disease with chest pain and its role to predict acute coronary events and short-term clinical outcome.

Methodology

Study Design: The present study will be a hospital-based prospective observational study conducted to evaluate the diagnostic and prognostic significance of high-sensitivity cardiac troponin (hs-cTn) in chronic kidney disease (CKD) patients presenting with chest pain. The study will follow the principles outlined in the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Study Area: The study will be conducted in the Department of General Medicine, Netaji Subhas Medical College and Hospital (NSMCH), Amhara, Bihta, Patna, Bihar, India.

Study Duration: The study will be carried out over a period of one year from January 2025 to December 2025.

Sample Size: A total of 95 patients fulfilling the eligibility criteria will be enrolled consecutively during the study period.

Study Population: The study population will comprise adult patients with established chronic kidney disease presenting to the Emergency Department or Department of General Medicine with chest pain suggestive of acute coronary syndrome.

Inclusion Criteria

- Age ≥18 years.
- Diagnosed cases of chronic kidney disease (CKD) according to Kidney Disease Improving Global Outcomes (KDIGO) criteria.
- Estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m², calculated using the Modification of Diet in Renal Disease (MDRD) equation.
- Presentation with chest pain or symptoms suggestive of acute coronary syndrome within the previous 24 hours.
- High-sensitivity cardiac troponin T or I measured at admission.
- Patients willing to provide written informed consent.

Exclusion Criteria

- Age below 18 years.
- Acute kidney injury without evidence of chronic kidney disease.
- Recent major surgery or trauma within the preceding four weeks.
- Acute myocarditis, pericarditis, pulmonary embolism, or sepsis causing secondary troponin elevation.
- Known active malignancy with life expectancy less than six months.
- Pregnancy.
- Patients are unwilling or unable to provide informed consent.
- Incomplete clinical, laboratory, or follow-up data.

Data Collection: After obtaining written informed consent, all eligible patients will be enrolled consecutively during the study period. Data will be collected using a predesigned and pretested case record form. Detailed demographic information including age, sex, body mass index, and relevant clinical history such as hypertension, diabetes mellitus, dyslipidemia, smoking status, previous cardiovascular disease, dialysis status, and duration of chronic kidney disease will be recorded. Clinical details regarding the onset, duration, and characteristics of chest pain, associated symptoms, and vital parameters at presentation will also be documented. Laboratory investigations including serum creatinine, blood urea, estimated glomerular filtration rate (eGFR), hemoglobin, blood glucose, lipid profile, and high-sensitivity cardiac troponin (hs-cTnT or hs-cTnI) levels will be obtained. Electrocardiographic findings, echocardiographic parameters including left ventricular ejection fraction, and details of coronary angiography whenever performed will also be recorded. Patients will be followed during hospitalization and up to 30 days after admission to assess clinical outcomes, including major adverse cardiac events (MACE), need for revascularization, length of hospital stay, and mortality.

Study Procedure: All patients with chronic kidney disease presenting with chest pain suggestive of acute coronary syndrome will undergo a comprehensive clinical evaluation upon admission to the Department of General Medicine or Emergency Department. Baseline assessment will include detailed history taking, physical examination, electrocardiography, and routine laboratory investigations. High-sensitivity cardiac troponin levels will be measured at admission (0 hours), followed by repeat measurements 3 hours and 6 hours after presentation. Additional serial measurements may be performed if clinically indicated. Venous blood samples will be collected under aseptic precautions and transported immediately to the central laboratory for analysis using standardized assay techniques. Renal functions will be assessed by calculating eGFR using the Modification of Diet in Renal Disease (MDRD) equation, and patients will be categorized according to CKD stage. Two-dimensional echocardiography will be performed to evaluate cardiac structure and function. Coronary angiography and subsequent revascularization procedures will be undertaken whenever clinically indicated by the treating cardiologist. All patients will receive standard treatment according to institutional protocols and current clinical guidelines. Clinical outcomes, including diagnosis of acute coronary syndrome, occurrence of major adverse cardiac events, and mortality, will be monitored throughout hospitalization and during the 30-day follow-up period.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation (SD) or median (IQR), while categorical variables were presented as frequencies and percentages. Normality was assessed using the Kolmogorov–Smirnov test. Group comparisons were performed using the independent t-test or Mann Whitney U test, while ANOVA or Kruskal Wallis test was used for multiple-group comparisons. Categorical variables were analyzed using the Chi-square test or Fisher's exact test. Correlation between hs-cTn levels and renal function parameters was assessed using Pearson's or Spearman's correlation coefficients. Binary logistic regression analysis was performed to identify independent predictors of acute coronary syndrome, major adverse cardiac events, and mortality. Receiver Operating Characteristic (ROC) curve analysis was used to determine the diagnostic accuracy of hs-cTn by calculating AUC, sensitivity, specificity, PPV, and NPV. Survival outcomes were evaluated using Kaplan–Meier curves and Cox proportional hazards regression. A p-value <0.05 was considered statistically significant".

Result

In Table 1, the baseline demographic and clinical characteristics of the study population is presented. The age group of patients (>60 years) accounted for the highest number of patients (46.3%) compared to the age group 50-60 years (34.7%) which showed chest pain to be more prevalent in the older age group. There was a male predominance, as males accounted for 62.1% of the study population. In terms of the severity of CKD, the more severe stages were most prevalent: Stage 4 (36.8%) and Stage 3 (32.6%) and Stage 5 (30.5%). The most common comorbidity was hypertension (80.0%) followed by diabetes mellitus (53.7%). Nearly one-third of the patients (28.4%) were dialysis dependent. 43.2% of patients with chest pain had acute coronary syndrome, while the other 56.8% had other causes of chest pain. The results indicate that the burden of cardiovascular risk factors in the new population of patients with CKD presenting with suspected cardiac symptoms is very high.

Table 1: Baseline Demographic and Clinical Characteristics of CKD Patients Presenting with Chest Pain (N=95)

Variable	Frequency (n)	Percentage (%)
Age <50 years	18	18.9
Age 50–60 years	33	34.7
Age >60 years	44	46.3
Male	59	62.1
Female	36	37.9
CKD Stage 3	31	32.6
CKD Stage 4	35	36.8
CKD Stage 5	29	30.5
Hypertension	76	80
Diabetes Mellitus	51	53.7
Dialysis Dependent	27	28.4
Smoking History	24	25.3
Acute Coronary Syndrome (ACS)	41	43.2
Non-ACS Chest Pain	54	56.8`

Table 2 demonstrates a significant association between CKD stage and high-sensitivity cardiac troponin levels ($p<0.001$). Patients with CKD Stage 5 predominately had hs-cTn levels above 100 ng/L (21 of 29 patients with Stage 5 CKD had hs-cTn levels >100 ng/L). As renal function decreased, the percentage of patients with a high troponin level rose. These results indicate that higher baseline troponin levels are more likely to occur in patients with advanced CKD as a result of chronic myocardial injury, LVH, microvascular ischemia, and decreased renal clearance of troponin fragments. The findings highlight the need to account for the stage of CKD when assessing high troponin levels in clinical practice.

Table 2: Association Between CKD Stage and High-Sensitivity Troponin Levels (N=95)

hs-cTn Level (ng/L)	Stage 3 (n=31)	Stage 4 (n=35)	Stage 5 (n=29)	Total
<50	19	8	2	29
50–100	9	16	6	31
>100	3	11	21	35
Total	31	35	29	95

Table 3 demonstrates that there was statistically significant correlation between the diagnosis of acute coronary syndrome (hs-cTn) level ($p<0.001$). The prevalence of ACS was much higher with higher levels of troponin, but only about 13.8% of patients with a troponin level below 50 ng/L were diagnosed with ACS. Patients with levels of hs-cTn above 100 ng/L were deemed to have ACS in 74.3% of them. In contrast, most patients without ACS were detected in the lower categories of troponin. These results suggest that, although CKD patients have chronically elevated troponin, those values that are markedly elevated are highly associated with acute coronary events. Hence, hscTn is a valuable diagnostic marker for the diagnosis of ACS in patients with CKD with chest pain.

Table 3: Association Between High-Sensitivity Troponin Levels and Diagnosis of Acute Coronary Syndrome (N=95)			
hs-cTn Level (ng/L)	**ACS Present**	**ACS Absent**	**Total**
<50	4	25	29
50–100	11	20	31
>100	26	9	35
Total	41	54	95

Table 4 shows that there was a significant relationship between hs-cTn levels and left ventricular systolic function (p<0.001). Most of the patients who had a low troponin (less than 50 ng/L) had an ejection fraction of more than 50%. Patients with hs-cTn levels exceeding 100 ng/L, however, had a significantly increased incidence of impaired left ventricular function. The 14 patients with severe LV dysfunction (LVEF <40%) were all in the highest troponin group. The results indicate that high levels of hs-cTn are correlated with not only myocardial ischemia but structural and functional damage to the heart. This association underlines the prognostic role of troponin in the identification of the patients with CKD who are at high risk for adverse cardiovascular events.

Table 4: Association Between hs-cTn Levels and Left Ventricular Ejection Fraction (LVEF) (N=95)				
LVEF (%)	**hs-cTn <50 (n=29)**	**hs-cTn 50–100 (n=31)**	**hs-cTn >100 (n=35)**	**Total**
>50%	21	18	9	48
40–50%	7	10	12	29
<40%	1	3	14	18
Total	29	31	35	95

Table 5 illustrates the prognostic value of the hs-cTn for short-term CV outcomes. Patients with levels of hs-cTn above 100 ng/L had a significantly higher frequency of heart failure (34.3%), arrhythmias (22.9%), admission to the ICU (42.9%), revascularization procedure (37.1%) and death (20.0%) than those with lower levels of troponin. In addition, patients with the highest category of troponin had the highest incidence of overall major adverse cardiac events, with 60.0% of this group having at least one adverse cardiac event within 30 days. By comparison, only 13.8% of those who had a troponin level less than 50 ng/L experienced MACE. The results of this study suggest that level of hs-cTn is a very helpful prognostic marker for risk stratification in patients with CKD who are present with chest pain and will prove to be a useful indicator of adverse clinical outcomes.

Table 5: Association Between hs-cTn Levels and Major Adverse Cardiac Events (MACE) at 30 Days (N=95)				
Outcome	**hs-cTn <50 (n=29)**	**hs-cTn 50–100 (n=31)**	**hs-cTn >100 (n=35)**	**p-value**
Heart Failure	2	5	12	0.002
Arrhythmia	1	3	8	0.018
ICU Admission	2	6	15	<0.001
Revascularization	1	4	13	<0.001
Mortality	0	2	7	0.011
Any MACE	4	10	21	<0.001

Discussion

Patients with chronic kidney disease (CKD) with chest pain are a special group of high-risk cardiovascular patients due to accelerated atherosclerosis, chronic inflammation, endothelial dysfunction, oxidative stress and vascular calcification. A large percentage of the patients in the present study had acute coronary syndrome (ACS) and there was a strong positive correlation between high-sensitivity cardiac troponin (hs-cTn) and the prevalence of ACS. Of patients who had a hs-cTn level greater than 100 ng/L, 74.3% had ACS, while 13.8% of those with levels less than 50 ng/L had ACS".

In addition, the higher the troponin level, the higher the stage of CKD; 72.4 % of CKD Stage 5 patients had a level of hs-cTn greater than 100 ng/L (p<0.001). The results in this study support the notion that CKD is an equivalent of coronary heart disease given the umbrella of traditional and non-traditional cardiovascular risk factors. This has been reported by Go et al. (2004), who showed that cardiovascular events and hospitalization rate increased progressively as renal function decreased [15]. Similarly, Weiner et al. (2004) reported that CKD patients with reduced glomerular filtration rate experienced a significant cardiovascular risk independently of other factors and this was associated

with more cardiovascular morbidity and mortality [16].

The present study additionally showed that high levels of hs-cTn continued to be highly diagnostic even when the issue of chronic baseline elevation of troponin in CKD patients was considered. As expected, there was a significant relationship between the levels of hs-cTn and ACS diagnosis ($p<0.001$), meaning that high levels of troponin are still a clinically important marker for acute myocardial injury. This was echoed by Stacy et al. (2014) who conducted a systematic review and found that although initial troponin levels are often raised in CKD, the magnitude of the raised over time and any changes over time are well correlated with acute coronary events and adverse cardiovascular outcomes. The same authors, Twerenbold et al. (2018), found a good diagnostic performance of serial high-sensitivity troponin measurements to confirm myocardial infarction in renal dysfunction patients, which encourages the continued use of troponin-based diagnostic algorithms in this population [17]. The present results thus confirm the need to consider the clinical context for the interpretation of troponin levels and not to overlook a raised value simply because it might be caused by renal insufficiency.

An additional important finding of the present study is that the levels of hs-cTn were significantly related to cardiac function. The majority of patients had reduced LVEF, with 77.8% of the patients in the highest troponin category having severe LVEF (<40%). This implies that high troponin levels can be correlated not only with acute ischemic injury but also with the continued structural myocardial injury. The same was shown by Apple et al in-end stage renal failure patients, who demonstrated that a persistent increase in troponin was correlated with underlying myocardial injury and adverse cardiac remodeling in these patients [18]. Khan et al. (2005) also reported a positive correlation between elevation of cardiac troponin T and future CV events and mortality in asymptomatic dialysis patients, highlighting the association of cardiac troponin T elevation with chronic myocardial damage in CKD.

In addition, the present study showed the prognostic value of hscTn. Heart failure incidence was 34.3% in patients with an hs-cTn greater than 100 ng/L, significantly higher than the other categories of patients with lower levels of troponin ($P<0.001$). Rates of hospitalization for arrhythmia, for ICU admission and for revascularization procedures were also higher (22.9%, 42.9%, 37.1%, respectively) than in patients with low levels of troponin ($P<0.001$, $P=0.005$, and $P=0.003$, respectively). Mortality was also higher (20.0%) in patients with an hs-cTn greater than 100 ng/L than in other patients with lower levels of troponin ($P=0.003$). The overall major adverse cardiac events (MACE) rate was 60.0% in the highest troponin group, and 13.8% in those with hs-cTn levels below 50 ng/L ($p<0.001$). These results suggest that, in patients with CKD, high levels of hs-cTn are a strong predictor of poor short-term cardiovascular outcomes in those with chest pain. Similarly, deFilippi et al. (2010) found that high levels of high-sensitivity troponin were associated with cardiovascular mortality and adverse cardiac events in renal dysfunction patients. As in this study, Gunsolus et al. (2018) also observed that both diagnostic and prognostic value of hs-cTnI was preserved in CKD patients, albeit not as specific as in non-CKD patients, and that higher levels of hs-cTnI were significantly linked to poor clinical outcomes [19].

The observed correlation of rising troponin with the progression of the degree of renal insufficiency/renal failure further confirms that there are strong links between chronic myocardial injury and renal insufficiency/renal failure. In this study, the serum hs-cTn level rose with the progression of renal failure and was clearly correlated with renal function. These have been reported by Yang et al., who showed that optimal troponin thresholds rose as a function of the progression of the stage of CKD and different thresholds according to renal stage were found to improve the diagnostic accuracy of the diagnosis of acute myocardial infarction [20]. In a similar study, Chenevier Gobeaux et al. (2013) found that patients with moderate renal dysfunction showed a greater diagnostic accuracy of hs-cTn assays than patients with advanced renal dysfunction, indicating that the level of renal dysfunction can have a significant effect on the way that troponin should be interpreted.

In general, present study data are in line with previously reported data that hcTn is a significant clinical and prognostic biomarker in CKD patients presenting with chest pain. Baseline troponin elevation is a frequent finding in advanced renal disease, but high levels are linked to ACS, poor left ventricular function and poor cardiovascular outcomes. The findings highlight the need for careful hscTn interpretation in addition to a clinical assessment, ECG findings and serial measurements. Moreover, the strong relationship between high levels of hs-cTn and subsequent MACE demonstrate the usefulness of hs-cTn for risk stratification and prognosis in this high-risk population.

Conclusion

The present study shows that the use of high-sensitivity cardiac troponin (hs-cTn) is useful for the diagnosis and prognosis in patients with chronic kidney disease (CKD) with chest pain. There was a significant correlation with elevated hs-cTn levels and advanced stages of CKD, as well as increased risk for acute coronary syndrome, decreased left ventricular ejection fraction and increased rate of major adverse cardiac events (heart failure, arrhythmia, re-

vascularization, intensive care unit admission and death). Baseline troponin levels are higher in patients with renal dysfunction, but particularly high levels of Hs-cTn were highly predictive of acute myocardial injury and short-term clinical outcomes. The results of these studies emphasize the need for combining hs-cTn with a clinical assessment, electrocardiographic features and serial measurements for better diagnosis and risk stratification. As such, hs-cTn can be a valuable instrument for identifying high-risk CKD patients at an early stage and thereby provide for early intervention which might optimize cardiovascular outcome of these vulnerable people.

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