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Evaluation of Troponin I Values in Acute Coronary Syndrome Patients with and without Impaired Renal Function

Rukhsana Tumrani,[1] Afsheen Nigar,[2] Muhammad Usman,[3] Madiha Naqvi,[4] Fahad Liaqat,[5] Zainab Awais[6]

ABSTRACT

Background & Objective: Elevated cardiac troponin levels are common in chronic kidney disease (CKD) which make it challenging to diagnose acute coronary syndrome in these patients. It is difficult to interpret the cardiac troponin levels in CKD patients due to persistent increase of troponin levels in these patients which limit their diagnostic value. Our study aims to evaluate troponin-I levels in patients with acute coronary syndrome with and without impaired renal function

Methodology: Retrospective Cross-sectional study conducted in Pathology department, Shalamar Hospital Laboratory, Lahore from February 2025 to August 2025. A total of 202 Patients aged between 25 to 65 years both genders diagnosed as acute coronary syndrome with evaluated renal function were included. Data was divided in two groups i.e., Group A (Preserved renal function, eGFR≥ 60ml/min/1.73m2) Group B (Impaired renal function, eGFR<60ml/min/1.73m2). Troponin I levels were compared between patients with and without impaired renal function and p value <0.05 taken as statistically significant.

Results: Median age was 42 years in patients with preserved renal function and 53 years in patients with impaired renal function. Troponin I levels were significantly higher in patients with impaired renal function (6.80ng/ml) than in patients with preserved renal function (3.80ng/ml). Weak negative, statistically insignificant correlation found between eGFR and troponin I (r, p value: -0.18, 0.09) in patients with preserved renal function (Group A). Moderate to strong, significant negative correlation between eGFR and troponin I (r, p value: -0.56, <0.001) in patients with impaired renal function (Group B). Statistically significant difference in troponin I levels between patients with preserved and impaired renal function with higher levels in impaired group with P value 0.012.

Conclusion: It has been concluded that serum troponin I levels are significantly higher in patients with acute coronary syndrome with impaired renal function. These findings highlight the importance of interpreting troponin I values with caution in acute coronary syndrome patients with renal impairment to avoid overdiagnosis of myocardial infarction. Further studies are recommended to establish adjusted diagnostic cutoff for troponin I in patients with impaired renal function to improve the clinical decision making.

KEY WORDS: Troponin I, Acute coronary syndrome, Myocardial infarction, renal impairment, cardiac biomarker, chronic kidney disease

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INTRODUCTION

Elevated cardiac troponin levels are common in chronic kidney disease which make it challenging to diagnose acute coronary syndrome (ACS) in these patients. The preferred biomarker for diagnosing acute myocardial infarction (AMI) in the absence of ST-segment elevation (NSTE) is cardiac troponin.[1,2] Troponin levels are persistently increased in chronic kidney disease (CKD) patients, which lessens their diagnostic value when NSTE-AMI is suspected.[3,4] Cardiac biomarkers play a major role in the diagnosis when pathognomonic alterations in the ECG cannot be used as a basis. The preferred biomarkers for acute myocardial infarction are cardiac troponins I (cTnI) and T (cTnT), although CKD patients frequently have nonspecific increases in these biomarkers.[5,6]

The term "high-sensitivity" (hs) refers to the degree of improvement in the sensitivity and reliability of cardiac troponin I or T tests, particularly point-of-care cardiac troponin I or T testing. These tests enable the determination of cardiac troponin I or T values in at least 50% of blood samples from healthy individuals, in blood samples from patients who have recently experienced an acute myocardial infarction and in blood samples that exhibit an increase in cardiac troponin I or T levels. [7,8]

As cardiac troponin tests continue to advance, the quality of diagnosis is improved and the time between diagnosis and hospital discharge is shortened, which results in fewer additional clinical evaluations and lower medical expenses.[9,10] Higher baseline troponin I levels are often found in patients with renal impairment, even in the absence of acute coronary ischemia. In true ACS cases, both groups show elevated

Correspondence:
Dr Rukhsana Tumrani
Assistant Professor Chemical Pathology, Department of Pathology
Shalamar Medical and Dental College, Lahore
Email: r.tumrani333@gmail.com

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troponin, but patients with CKD may have disproportionately higher values, making interpretation difficult.[11,12]

Study aims to evaluate troponin-I values in patients with acute coronary syndrome with and without impaired renal function. The diagnosis of acute coronary syndrome by using cardiac troponin levels in patients with renal impairment is challenging. It is difficult to interpret the cardiac troponin levels in CKD patients due to persistent increase of troponin levels in these patients which limit their diagnostic value. There is need to evaluate the significant difference in cardiac troponin levels in acute coronary syndrome patients with and without renal impairment to prevent mis-interpretation and misdiagnosis. Limited research related to our study available in our country and no such study has been conducted previously at our institution.

METHODOLOGY

After taking ethical clearance from institutional review committee, REF # SMDC-IRB/AL/2025-078 Dated 8-9-2025, retrospective cross-sectional study was conducted at Pathology department, Shalamar hospital laboratory, Lahore from February 2025 to August 2025. Convenient sampling technique was used. Sample size 202 calculated by using the formula ($n = (Z\alpha/2 + Z\beta)^2 * 2\sigma^2 / d^2$) where Confidence interval taken as 95%, Power 80%, Population variance 100, Difference of troponin I values taken as 2.8ng/dl (Manufacturer trop I value: 4.7ng/dl and trop I value in Patients with CKD was 7.5ng/dl)[13]. Patients were classified in two groups as follows;

Group A: Preserved renal function, eGFR≥60ml/ min/ 1.73m^2 (n=101)

Group B: Impaired renal function, eGFR<60ml/min/ 1.73m^2 (n=101). eGFR calculated by using CKD-EPI equation. Chronic kidney disease labelled according to KDIGO classification of CKD defined as eGFR<60ml/min/1.73m^2 for 3months or marker of kidney damage such as albuminuria on the basis of albumin to creatinine ratio >30mg/g.[14] Patients aged between 25 to 65 years both genders diagnosed as Acute coronary syndrome with evaluated renal function tests willing to be included in study were included. Patients not evaluated renal function tests and not willing to be included in study were excluded. Informed consent was taken from patients or their attendants. Data was collected on pre designed proforma for demographic characteristics such as age, gender and for Laboratory results: Troponin I(ng/ml), Creatinine (mg/dl), eGFR(ml/min/1.73m^2). Data was recruited from laboratory online software i2. Data was entered and analyzed using SPSS version 25. Shapiro Wilk test was used to check the normality of data in both groups A & B. Normally distributed variables were presented in terms of mean and SD. Non normally distributed variables were presented in terms of median and IQR. Categorical variables such as gender were presented in terms of frequency and percentages.

Troponin I levels were compared between patients with and without impaired renal function by using Mann-Whitney U test. Normally distributed continuous variables were compared using independent sample t-test. While non-normally distributed variables were compared using Mann-Whitney U test. Categorical variables were compared using Chi square test. Spearman correlation was used to assess the relationship between eGFR and troponin I levels. P value <0.05 was taken as statistically significant.

RESULTS

Of the total 202 study subjects, median age in group A with preserved renal function(n=101) was 42(37-47) years while in group B with impaired renal function(n=101), median age was 53(48-58) years (Table 1). Median eGFR in group A was 88(77-99) ml/min/1.73m^2 while in group B was 25(16-34) ml/min/1.73m^2 (Table 1). Median troponin I(ng/ml) in group A was 3.45(1.20-8.70) while in group B was 6.80(2.40-15.60) as shown in figure 1. Weak negative,

Table I: Distribution of different variables in both groups A (with preserved renal function, n=101) and group B (with impaired renal function, n=101)

Parameter	*Preserved renal function (n=101)*	*Impaired renal function (n=101)*
Median age (IQR) years	42 (37-47)	53(48-58)
%Male (n)	63.36% (64)	55.44% (56)
Median eGFR (IQR)	88(77-99)	25(16-34)
Median troponin I (ng/ml) (99TH Percentile cutoff : 0.04)	3.45(1.20-8.70)	6.80 (2.40-15.60)
Correlation between eGFR And troponin (r, p value)	-0.18(0.09)	-0.56(<0.001)

Table II: Troponin I levels in both groups A & B sub-grouped on the basis of eGFR (n=202)

	Preserved Renal function (Group A)		**Impaired renal function (Group B)**		**P value (Mann-Whitney U test, non-parametric comparison**
Parameter	eGFR 60-90	eGFR>90	eGFR<30	eGFR (30-60)	
Troponin I (Median IQR)	4.80 (1.60-9.10)	2.10 (0.90-5.30)	10.80 (4.60-22.40)	5.40 (1.90-12.20)	0.012
P (Mann-Whitney U)	0.023		0.02		

statistically insignificant correlation found between eGFR and troponin I (r, p value: -0.18, 0.09) in patients with preserved renal function (Group A) as shown in table I. Moderate to strong, significant negative correlation was found between eGFR and troponin I (r, p value: -0.56, <0.001) in patients with impaired renal function (Group B) as shown in table I. Troponin I levels in both groups A & B sub-grouped on the basis of eGFR shown in figure 2. Statistically significant difference in troponin I levels between patients with preserved and impaired renal function was found with P value 0.012 (table 2).

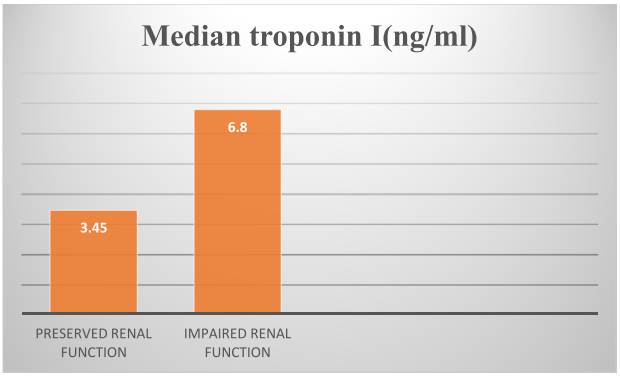

Figure 1: Median Troponin I (ng/ml) in patients with preserved renal function (Group A) and Impaired renal function (Group B)

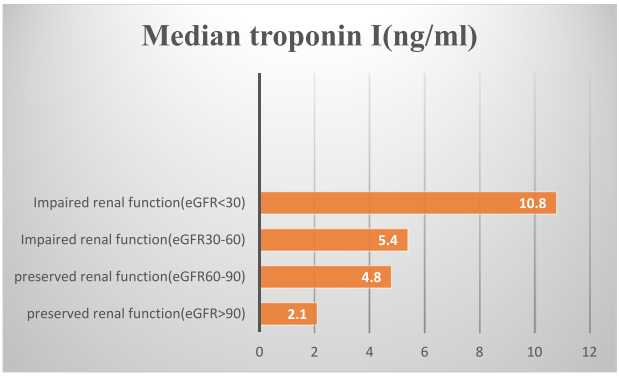

Figure 2: Troponin I levels in both groups A & B sub-grouped on the basis of eGFR (n=202)

DISCUSSION

The gold standard for detecting acute coronary syndrome (ACS) is cardiac troponin I (cTnI), a highly sensitive and specific biomarker for myocardial damage. However, because of altered troponin kinetics, elevated baseline levels, and overlapping clinical characteristics, interpreting troponin levels becomes difficult in individuals with reduced renal function. Even in the absence of an acute myocardial infarction (AMI), patients with chronic kidney disease (CKD) or acute kidney injury (AKI) may have increased baseline troponin I levels. [15]

A retrospective study was conducted by Ren D et al and in this study, patients presenting with chest pain (n ≈ 3,300; ~23% with reduced eGFR were included and it was concluded that optimal cut off values of hs cTnI for diagnosing acute myocardial infarction increase as glomerular filtration rate declines; in patients with CKD, the diagnostic threshold was substantially higher than in those with preserved renal function. Importantly, even in CKD patients without AMI, hs cTnI correlated weakly with the degree of renal impairment.[16]

Kampmann J. et al conducted a systematic review and meta analysis in 2022. They included studies of both troponin I and T in patients with impaired renal function and it was found that the optimal cut off for troponin I in non dialysis CKD for diagnosing AMI lies considerably above standard reference limits, helping to improve specificity without compromising the sensitivity excessively.[17] Another study by Alushi B et al demonstrated that diagnostic accuracy in patients with severe CKD (eGFR very low) for hs troponin T has been shown to improve when using higher assay specific cutoff levels combined with early absolute changes (delta troponin over 3h). However, it was established for troponin T and it was suggested to establish similar strategies for troponin I as well.[18]

Recent studies over the past five years have increasingly focused on quantifying how renal impairment modifies both diagnostic performance and prognostic significance of troponin assays. In a large stepped wedge cluster randomized trial, implementation of gender specific 99th percentile thresholds for hs cTnI in patients with and without kidney impairment (eGFR<60 mL/min/1.73m²) showed that patients with kidney impairment were much more likely to have hs cTnI above the 99th percentile. In such patients, the risk of adverse outcomes (type 1 MI or cardiovascular death) was approximately double than in those without renal impairment when troponin exceeded that threshold.[19]

Studies of patients with renal impairment vs those without renal impairment demonstrated "delta" changes (rise/fall) in high sensitivity troponin, and whether the presence of CKD significantly alters the performance of these changes in predicting ACS requiring PCI or revascularization. Some have found that delta changes maintain utility even in CKD, though specificity is reduced.[20]

A study conducted by Miller-Hodges E et al demonstrated the hs-Troponin and the risk stratification of renal impaired patients presenting with suspected ACS and it was shown that hs-cTnI levels in patients with and without renal impairment (eGFR < 60 mL/min/1.73 m²) in a sizable prospective cohort (~4,739 people) were over the 99th centile twice as common in patients with renal insufficiency. Regardless of renal function, the incidence of MI or cardiac mortality after 30 days was low in those with troponin < 5 ng/L at presentation; however, only around 1 in 5 people with renal impairment had such low troponin. Additionally, patients with renal impairment were twice as likely to experience a severe cardiac incident as those without renal impairment for equal elevations in troponin. Even in cases of renal impairment, low absolute troponin can rule out risk; however, specificity for Type-1 MI is reduced due to increased baseline levels. [21]

Another study conducted by Hemmert KC et al showed

the clinical implementation of hs-troponin assay in patients with renal Impairment. While raised hs-cTnI levels rise as kidney function deteriorates, a significant portion of elevated outcomes in renal impairment are not Type 1 myocardial infarctions (i.e., plaque-rupture coronary occlusion), according to a randomized study by Mills/Gallacher et al. about 66% of patients with eGFR < 30 mL/min/1.73 m² had high hs-cTnI in the randomized trial (n = 46,927) of hs-cTnI implementation, however only about 35% of those were classified as Type 1 MI. This highlights the trade-off in renal impairment diagnosis; increased sensitivity but significantly poorer specificity.[22]

Another study conducted by Brunner FJ et al on Hs-Troponin T and I and the severity of stable coronary artery disease in CKD Patients and it was demonstrated that despite being "stable coronary artery disease," this provides insight and both hsTnT and hsTnI were assessed and linked with CAD severity (SYNTAX/Gensini scores) in a cohort of about 2,209 patients undergoing angiography, of whom approximately 595 had eGFR < 60 mL/min/1.73m². Patients with reduced renal function had higher troponin levels, which were associated with more severe CAD. [23]

Many factors contribute to raised troponin levels in patients with renal impairment, such as decreased renal clearance of troponin fragment, increased heart strain as a result of volume overload and left ventricular hypertrophy etc. According to recent literature search, diagnostic accuracy of hs-cTnI assays can be increased by modifying the cutoffs keeping in view the renal function. Nevertheless, research is currently ongoing to determine generally recognized reference ranges for various stages of CKD. In true ACS cases, both groups show elevated troponin, but patients with CKD may have disproportionately higher values, making interpretation difficult.[24]

Renal insufficiency reduces the specificity of increased troponin I in the diagnosis of myocardial infarction. Regardless of renal function, a rising/falling troponin I pattern is still a good sign of acute MI. Even in patients with renal impairment, troponin I is still a valuable biomarker for the diagnosis of ACS. Clinicians must, however, take into account baseline increase due to chronic kidney disease (CKD) and concentrate on dynamic variations in troponin levels rather than absolute values. Accurate diagnosis and risk stratification require a multimodal approach that includes serial biomarkers, clinical presentation, imaging, and ECG.[25]

It is recommended to use high-sensitivity troponin assays with established reference ranges for CKD populations. Serial measurements and trend analysis are crucial in patients with renal dysfunction. Alternative biomarkers (e.g., copeptin, CK-MB) can be considered in ambiguous cases. Further studies are needed to refine troponin cutoffs specific to renal function levels.

CONCLUSION

It has been concluded that serum troponin I levels are significantly higher in acute coronary syndrome patients with impaired renal function. These findings highlight the importance of interpreting troponin I values with caution in ACS patients with renal impairment to avoid misdiagnosis of myocardial infarction. Further studies are recommended to establish adjusted diagnostic cutoff for troponin I in patients with impaired renal function to improve the clinical decision making.

Ethical approval:

Ethical approval was taken from institutional review board of Shalamar Medical and Dental College Lahore, vide REF # SMDC-IRB/AL/2025-078 Dated 8-9-2025.

Conflict of Interest:

Authors declare no conflict of interest.

Financial Disclosure:

None

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Authors' Contributions:

RT & AN: Conceptualization & study design.

AN, MU, MN: Data Collection and manuscript drafting.

FL, ZA: Data Analysis and critical review.

AN, RT, MU, MN: Supervision &Manuscript drafting & proof reading.

All authors have read and approved the final version of the manuscript and are responsible and accountable for the accuracy and integrity of the work.

1. Rukhsana Tumrani
   Assistant Professor Pathology, Department of Pathology,
   Shalamar Medical and Dental College, Lahore
2. Afsheen Nigar
   Assistant Professor Pathology,
   University College of Medicine and Dentistry,
   University of Lahore
3. Muhammad Usman
   Senior Registrar Cardiology,
   Shalamar Medical and Dental College, Lahore
4. Madiha Naqvi
   Assistant Professor Pathology, Department of Pathology,
   Shalamar Medical and Dental College, Lahore
5. Fahad Liaqat
   Senior Registrar Cardiology,
   University College of Medicine and Dentistry,
   University of Lahore, Lahore
6. Zainab Awais
   Senior Demonstrator Pathology,
   University College of Medicine and Dentistry,
   University of Lahore, Lahore