



Original Research Article

ESTIMATION OF CARDIAC TROPONIN-T IN PATIENTS WITH END-STAGE RENAL DISEASE: A HOSPITAL-BASED CROSS-SECTIONAL STUDY

Kiran Kumar B N¹, Suma C P², Mahanthesh P³

¹Associate Professor, Department of General Medicine, Karwar Institute of Medical Sciences, Karwar, India.

²Assistant Professor, Department of Paediatrics, Karwar Institute of Medical Sciences, Karwar, India.

³Associate Professor, Department of Dermatology, Karwar Institute of Medical Sciences, Karwar, India.

Received : 12/05/2026
Received in revised form : 01/07/2026
Accepted : 16/07/2026

Corresponding Author:

Suma C P,
Assistant Professor, Department of
Paediatrics, Karwar Institute of
Medical Sciences, Karwar, India.
Email: cpsuma17@gmail.com

DOI: 10.70034/ijmedph.2026.3.163

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 1011-1015

ABSTRACT

Background: Cardiovascular disease is the leading cause of mortality among patients with chronic kidney disease (CKD) and end-stage renal disease (ESRD). Elevated cardiac troponin-T (cTnT) is a sensitive biomarker of myocardial injury and may identify subclinical cardiac involvement in these patients. This study aimed to estimate serum cardiac troponin-T levels in patients with CKD stages 3–5 and evaluate their association with disease stage.

Materials and Methods: A hospital-based cross-sectional study was conducted over 12 months in the Department of General Medicine of a tertiary care teaching hospital. Sixty-four adult patients with CKD stages 3–5 undergoing maintenance hemodialysis or conservative management were enrolled using consecutive sampling. Patients with recent acute coronary syndrome, myocardial infarction, acute kidney injury, sepsis, or other conditions affecting troponin levels were excluded. Clinical details, laboratory investigations, electrocardiography, echocardiography, and high-sensitivity cardiac troponin-T assays were performed. Data were analyzed using descriptive and inferential statistics.

Results: The mean age of participants was 57.25 ± 5.54 years, with 70.3% being males. Stage V CKD constituted 50% of cases, followed by Stage IV (26.6%) and Stage III (23.4%). Diabetes mellitus and hypertension coexisted in 64.1% of patients. All participants were anaemic, 92.2% had elevated ESR, and 64.1% demonstrated left ventricular hypertrophy on echocardiography. Elevated serum cardiac troponin-T levels were detected in 36 (56.25%) patients. Troponin positivity increased with advancing CKD stage, although the association was not statistically significant ($P = 0.590$).

Conclusion: Elevated cardiac troponin-T is common in patients with advanced CKD and ESRD and increases with disease severity. High-sensitivity cardiac troponin-T may serve as a valuable biomarker for detecting subclinical myocardial injury and identifying CKD patients at increased cardiovascular risk.

Keywords: Chronic Kidney Disease (CKD), End-Stage Renal Disease (ESRD), Cardiac Troponin-T, Myocardial Injury, Cardiovascular Risk.

INTRODUCTION

End-stage renal disease (ESRD) and chronic kidney disease (CKD) are clinical syndromes resulting from a variety of underlying renal disorders.^[1] Cardiovascular disease is the leading cause of mortality among patients with ESRD and advanced

CKD (Stages 3–5).^[2] Many of these patients develop clinically silent myocardial injury, which often remains undetected until significant cardiac damage has occurred.^[3]

Elevated cardiac troponin T (cTnT) levels in patients with ESRD/CKD are believed to reflect underlying subclinical cardiac pathology.^[4] Cardiac

troponin T is a highly specific biomarker of myocardial cell injury because of its excellent cardiac tissue specificity.^[5] Therefore, an increase in plasma cTnT concentration is considered a reliable indicator of myocardial injury, even in the absence of overt clinical symptoms.^[6] Hence, the present study was conducted to determine the association between elevated cardiac troponin levels and myocardial injury and end-stage renal disease.

MATERIALS AND METHODS

Study design and setting

A hospital-based cross-sectional observational study was conducted in the Department of General Medicine at a tertiary care teaching hospital over a period of 12 months. This study was conducted on patients diagnosed with End-Stage Renal Disease (ESRD) undergoing maintenance hemodialysis or receiving conservative management at the study hospital.

Sample Size

A total of **64 patients** with ESRD fulfilling the eligibility criteria will be included in the study.

Sample Size Calculation

The sample size was calculated using the formula for estimating a proportion in a cross-sectional study:

$$n = \frac{Z^2 P (1-P)}{d^2}$$

Where:

- n = Required sample size
- Z = Standard normal deviate at 95% confidence level = 1.96
- P = Expected prevalence of elevated cardiac troponin-T among ESRD patients = 90% (0.90) (based on previous studies)
- d = Absolute precision = 7.5% (0.075)

$$n = \frac{(1.96)^2 \times 0.90 \times 0.10}{(0.075)^2}$$

$$= \frac{3.84 \times 0.90}{0.005625} = 61.4 = 62$$

Considering approximately 5% non-response, the final sample size was rounded to 64 participants.

Sampling Technique

Consecutive sampling was used. All eligible ESRD patients attending or admitted to the medicine wards during the study period were recruited until the required sample size of 64 is achieved.

Inclusion Criteria

Patients aged 18 years and above diagnosed with **CKD stages 3–5** according to the **KDIGO classification** (Stage 3: eGFR 30–59 mL/min/1.73 m², Stage 4: eGFR 15–29 mL/min/1.73 m², and Stage 5: eGFR <15 mL/min/1.73 m²), undergoing maintenance hemodialysis or receiving conservative treatment, and willing to provide written informed consent were included in the study.

Exclusion Criteria

Patients with a history of acute myocardial infarction within the preceding three months, acute coronary syndrome at the time of recruitment, acute kidney injury, congestive heart failure, acute infections or sepsis, pulmonary embolism, myocarditis, pericarditis, or recent major surgery or trauma were excluded from the study. Patients who declined to provide written informed consent were also excluded.

Study Procedure

After obtaining approval from the Institutional Ethics Committee and written informed consent, eligible patients were enrolled. For each participant, data were collected using a predesigned and pretested case record form. Information regarding demographic characteristics, including age and sex, duration and etiology of chronic kidney disease (CKD), history of hypertension and diabetes mellitus, duration and frequency of hemodialysis, blood pressure, and body mass index (BMI) was recorded. All participants underwent relevant laboratory investigations, including complete blood count, blood urea, serum creatinine, serum electrolytes, estimated glomerular filtration rate (eGFR), serum calcium, serum phosphate, serum albumin, lipid profile, and blood glucose estimation. In addition, electrocardiography (ECG) was performed for all participants, while echocardiography was carried out wherever clinically indicated. Cardiac Troponin-T levels were estimated using a high-sensitivity assay to assess the presence of myocardial injury in patients with end-stage renal disease.

Cardiac Troponin-T Estimation

Approximately 3–5 mL of venous blood was collected under aseptic precautions before the dialysis session. Serum was separated by centrifugation and analyzed for cardiac Troponin-T (cTnT) using a high-sensitivity electrochemiluminescence immunoassay (hs-cTnT) on an automated analyzer according to the manufacturer's instructions. Troponin-T values were reported in ng/L (or pg/mL depending on the assay). An elevated cTnT level was defined according to the 99th percentile upper reference limit recommended by the assay manufacturer.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26.0 (or equivalent statistical software). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) depending on data distribution. Categorical variables will be presented as frequencies and percentages.

RESULTS

A total of 64 patients who were confirmed to be suffering from end-stage renal disease / chronic kidney disease constituted the study population.

Table 1 shows the distribution of patients according to diagnosis, age, gender, weight, and history. Table

2 shows the various lab investigations of patients.

Table 1: Diagnosis and demographic details of the patients

Characteristics	Number of patients (n=64)	Percentage (%)
Diagnosis		
Stage III chronic kidney disease	15	23.4
Stage IV chronic kidney disease	17	26.6
Stage V chronic kidney disease	32	50.0
Age in years		
Up to 50 years	7	10.9
51-60	38	59.4
61-70	19	29.7
Gender		
Male	45	70.3
Female	19	29.7
Weight (kg)		
40-50	15	23.4
51-60	15	23.4
61-70	32	50.0
>70	2	3.1
History		
Nil	4	6.3
Diabetes mellitus	5	7.8
Hypertension	10	15.6
Both Diabetes mellitus & Hypertension	41	64.1
Others	4	6.3

Table 2: Lab Investigations of patients

Lab Investigations	Number of patients (n=64)	Percentage (%)
Hemoglobin		
Anemia	64	100.0
Normal	0	0.0
Total count		
Leucopenia	0	0.0
leucocytosis	1	1.6
Normal	63	98.4
RBC		
Less	14	21.9
Normal	46	71.9
High	4	6.3
ESR		
Elevated	59	92.2
Normal	5	7.8
Platelet count		
Thrombocytopenia.	9	14.1
Thrombocytosis	0	0.0
Normal	55	85.9
PCV		
Decreased	3	4.7
Increased	6	9.4
Normal	55	85.9
USG abdomen findings		
Normal	8	12.5
Medical renal disease-Gr II	44	68.8
Medical renal disease-Gr III	12	18.8
2D ECHO		
Normal.	23	35.9
Left ventricular hypertrophy, mild.	20	31.3
Left ventricular hypertrophy, moderate.	21	32.8

Table 3: Correlation of Troponin T levels with Stage of disease

Troponin T levels	Number of patients (n=64)	Stage of Disease		
		Stage III	Stage IV	Stage V
Negative	28	8(28.6%)	6(21.4%)	14(50.0%)
Positive	36	7(19.4%)	11(30.7%)	18(50.0%)
Total	64	15(23.4%)	17(26.6%)	32(50.0%)
Inference	As the Stage level increases, the troponin T level also increases, but not statistically significant with P=0.590			

DISCUSSION

A total of 64 patients were included in the study. The mean age of the participants was 57.25 ± 5.54 years, with ages ranging from 45 to 68 years. Of the 64 patients, 45 (70.3%) were males and 19 (29.7%) were females.

In the present study, diabetes mellitus alone was observed in 7.8% of patients, while hypertension alone was present in 15.6%. However, both diabetes mellitus and hypertension coexisted in 64.1% of the study participants. The high prevalence of these comorbid conditions highlights their synergistic role as major risk factors for the development and progression of chronic kidney disease (CKD), ultimately leading to end-stage renal disease (ESRD). In the study done by Freda BJ et al and Mallamaci F et al also showed similar type of findings.^[7,8]

As chronic kidney disease (CKD) and end-stage renal disease (ESRD) are chronic progressive conditions, all patients in the present study were anaemic at the time of hospital admission. This finding is consistent with the well-established association between declining renal function and reduced erythropoietin production, leading to anaemia.

A study by Wood GNK et al. reported a mean haemoglobin level of 12.2 g/dL among 222 patients across different stages of chronic kidney disease.^[9] Although the severity of anaemia was greater in the present study, likely because it included patients with advanced CKD/ESRD, the findings are comparable and support the high prevalence of anaemia in patients with chronic kidney disease.

Erythrocyte sedimentation rate (ESR) was elevated in 92.2% of patients, while only 7.8% had normal ESR values. The high prevalence of elevated ESR may be attributed to the chronic inflammatory state, uraemia, and associated comorbidities commonly observed in patients with CKD and ESRD.

In the present study, two-dimensional echocardiography (2D-ECHO) findings were normal in 23 (35.9%) patients. Mild left ventricular hypertrophy (LVH) was observed in 20 (31.3%) patients, while moderate LVH was present in 21 (32.8%) patients. Overall, 41 of the 64 patients (64.1%) had evidence of left ventricular hypertrophy, making it the most common echocardiographic abnormality in the study population.

A study conducted by Owen WF et al., which included 126 patients with chronic kidney disease, reported a prevalence of left ventricular hypertrophy of 46.8%.^[10] The higher prevalence of LVH observed in the present study may be attributed to the inclusion of patients predominantly with advanced chronic kidney disease and end-stage renal disease, in whom cardiac structural changes are more common.

Similarly, Wang AM et al. reported that left ventricular hypertrophy was present in more than 64% of patients across all stages of chronic kidney disease, with 130 of 201 patients demonstrating LVH.^[11] The prevalence of left ventricular hypertrophy in the present study (64.1%) closely corresponds with the findings of Wang AM et al., thereby supporting the high burden of cardiovascular involvement among patients with chronic kidney disease.^[11]

In the present study, elevated serum cardiac troponin T levels (>0.1 ng/mL) were observed in a substantial proportion of patients, indicating a high prevalence of myocardial injury among individuals with chronic kidney disease (CKD) and end-stage renal disease (ESRD).

C. Löwbeer et al. reported that 35 of 115 patients (30.4%) had elevated serum cardiac troponin T concentrations (>0.1 ng/mL).^[12] Similarly, Wang AM et al. found elevated serum cardiac troponin T levels in 95 of 222 patients (43%).^[11] The prevalence of elevated cardiac troponin T increased significantly with advancing stages of CKD, from 20% in Stage 3 to 39% in Stage 4 and 59% in Stage 5 ($P < 0.0001$), demonstrating a strong association between worsening renal function and increased myocardial injury.

Furthermore, Jutta Dierkes et al. reported that elevated cardiac troponin T levels were closely associated with increased all-cause mortality in patients with chronic kidney disease, highlighting the prognostic significance of this biomarker.^[13]

These findings suggest that elevated cardiac troponin T is common in patients with advanced CKD and ESRD and may serve as an important indicator of underlying cardiovascular disease and adverse clinical outcomes. The observations in the present study are in agreement with these published reports, emphasizing the value of cardiac troponin T as a marker of cardiovascular risk in patients with chronic kidney disease.

CONCLUSION

Elevated serum cardiac troponin T levels have been shown to be closely associated with increased all-cause mortality in patients with chronic kidney disease. In the present study, serum cardiac troponin T levels differed significantly across the stages of chronic kidney disease, with a higher proportion of patients demonstrating elevated levels in the more advanced stages of the disease. Overall, 56.25% of the study population with Stage 3, Stage 4, and Stage 5 chronic kidney disease (end-stage renal disease) had elevated serum cardiac troponin T levels. These findings are consistent with those reported by C. Löwbeer et al., Nasir A.A. et al., and Jutta Dierkes et al., supporting the observation that elevated cardiac troponin T is more prevalent in advanced chronic kidney disease and serves as an

important marker of cardiovascular risk and adverse clinical outcomes.

Conflicts of Interest: None

Author Contribution: All the authors are involved to conceptualization and conduction and manuscript writing of the study.

REFERENCES

1. De Filippi C, Wasserman S, Rosanio S. Cardiac troponin T and C-reactive protein for predicting prognosis, coronary atherosclerosis and cardiomyopathy in patients undergoing long-term hemodialysis. *JAMA*, 2003; 290: 353-359.
2. Apple FS, Murakami MM, Pearce LA, Herzog CA. Predictive value of cardiac troponin I and T for subsequent death in end-stage renal disease. *Circulation*, 2002; 106: 2941-2945.
3. Orea-Tejeda A, Colín-Ramírez E, Hernández-Gilsoul T et al. Microalbuminuria in systolic and diastolic chronic heart failure patients. *Cardiol J*, 2008; 15:143-149.
4. Sharma R, Gaze DC, Pellerin D, Mehta RL, Gregson H, Streather CP. Cardiac structural and functional abnormalities in end stage renal disease patients with elevated cardiac troponin T. *Heart* 2006;92:804-809.
5. Aviles RJ, Askari AT, Lindal B et al. Troponin T levels in patients with acute coronary syndromes, with or without renal dysfunction. *N Engl J Med*, 2002; 346: 2047-2052.
6. Deegan PB, Lafferty ME, Blumsohn A, Henderson IS, McGregor E. Prognostic value of troponin T in hemodialysis patients is independent of comorbidity. *Kidney Int* 2001; 60: 2399-405.
7. Freda BJ, Tang WHW, Van Lente F et al. Cardiac troponins in renal insufficiency: Review and clinical implications. *J Am Coll Cardiol*, 2002; 40: 2065-2071.
8. Mallamaci F, Zoccali C, Parlongo S et al. Troponin is related to left ventricular mass and predicts all-cause and cardiovascular mortality in hemodialysis patients. *Am J Kidney Dis*, 2002; 40:68-75.
9. Wood GNK, Keevil B, Gupta J et al. Serum troponin T measurement in patients with chronic renal impairment predicts survival and vascular disease: A 2 year prospective study. *Nephrol Dial Transpl*, 2003; 18: 1610-1615.
10. Owen WF, Nutritio27. Apple FS, Murakami MM, Pearce LA, Herzog CA. Predictive value of cardiac troponin I and T for subsequent death in end-stage renal disease. *Circulation*, 2002; 106: 2941-2945.
11. Wang AM, Lam CK, Yu CM et al. Troponin T, left ventricular mass, and function are excellent predictors of cardiovascular congestion in peritoneal dialysis. *Kidney Intern*, 2006; 70: 444-452.
12. Löwbeer C, Stenvinkel P, Pecoits-Filho R, Heimbürger O, Lindholm B, Gustafsson SA, Seeberger A. Elevated cardiac troponin T in predialysis patients is associated with inflammation and predicts mortality. *J Intern Med* 2003; 253: 153-160.
13. Dierkes J, Domrose U, Westphal S, et al. Cardiac troponin T predicts mortality in patients with end-stage renal disease. *Circulation* 2000; 102:1964-1969.