RESEARCH PAPER

Troponin I and CPK-MB in Chronic Kidney Disease Stage IV and V

Dr. Praveen Kumar K Reddy[1], Dr. Rama Mishra R[2], Dr. Anand A V[3*], Dr. Sandhya R[4], Dr. Mekkina Pranathi[5]

[1]Senior Resident, Vydehi Institute of Medical Sciences and RC
[2]Professor, Vydehi Institute of Medical Sciences and RC
[3*]Consultant Physician, KLE Hospital - Bengaluru, (KLE Academy of Higher Education and Research)
Email: alurvishnuanand@gmail.com
[4]Assistant Professor, Vydehi Institute of Medical Sciences and RC
[5]Post Graduate, Vydehi Institute of Medical Sciences and RC

Received: 15th Jul, 2026 | Revised: 25th Jul, 2026 | Accepted: 5th Aug, 2026 | Available Online: 12th Aug, 2026

Abstract

Background: Chronic kidney disease (CKD) is associated with a markedly increased risk of cardiovascular disease, particularly coronary artery disease (CAD). Interpretation of cardiac biomarkers in CKD is challenging because reduced renal clearance may result in elevated baseline levels. This study evaluated the utility of Troponin I (Trop I) and Creatine Kinase-MB (CK-MB) in detecting CAD among patients with CKD Stage IV and V.

Methods: A cross-sectional study was conducted in the Departments of General Medicine, Cardiology, and Nephrology at Vydehi Institute of Medical Sciences and Research Centre from March 2023 to August 2024. Ninety patients aged >18 years with CKD Stage IV or V were enrolled. Demographic details, clinical history, laboratory investigations, serum creatinine, estimated glomerular filtration rate (eGFR), Troponin I, CK-MB, electrocardiography, and two-dimensional echocardiography findings were recorded. Comparisons were made between patients with and without CAD. Receiver operating characteristic (ROC) analysis was performed to assess the diagnostic performance of Troponin I.

Results: The mean age of participants was 58.93 ± 12.00 years, and 71.1% were males. CAD was present in 28 (31.1%) patients. Serum creatinine and eGFR were comparable between CAD and non-CAD groups ($p>0.05$). Mean Troponin I levels were significantly higher in patients with CAD than those without CAD (1.10 ± 1.18 vs. 0.04 ± 0.05 ng/mL; $p<0.001$). In contrast, CK-MB levels did not differ significantly between groups (4.71 ± 6.47 vs. 3.73 ± 3.44 ng/mL; $p=0.352$). ROC analysis demonstrated excellent diagnostic performance of Troponin I for predicting CAD, with an AUC of 0.946, sensitivity of 89.29%, specificity of 98.39%, and an optimal cut-off value of >0.15 ng/mL.

Conclusion: Troponin I is a reliable and clinically useful biomarker for detecting CAD in patients with CKD Stage IV and V, whereas CK-MB has limited diagnostic value. Troponin I may aid cardiovascular risk stratification when interpreted alongside clinical and imaging findings.

Keywords: Chronic kidney disease, Troponin I, CK-MB, Coronary artery disease, Cardiac biomarkers, eGFR.

How to cite this article: Praveen Kumar K Reddy1 Dr. Rama Mishra R2 Dr. Anand A V3* Dr. Sandhya R4 Dr. Mekkina Pranathi5. Troponin I and CPK-MB in Chronic Kidney Disease Stage IV and V. Int J Drug Deliv Technol. 2026;16(77s):1386-1392. DOI: 10.25258/ijddt.16.77s.161

Introduction

Chronic kidney disease is a pathophysiological process with numerous etiologies, resulting in inexorable attrition of nephron number and function leading to end stage renal disease. [1] The intricate relationship between CKD and cardiovascular disease extends beyond conventional risk factors. Patients with CKD often show signs of accelerated atherosclerosis, vascular calcification, and endothelial dysfunction, all of which increase their risk of ischemic events[4]. Additionally, CKD is linked to a pro-inflammatory state, oxidative stress, and changes in lipid metabolism, which further amplify the cardiovascular risk. [2]

Chronic kidney disease (CKD) significantly affects troponin levels, with research demonstrating elevated serum troponin concentrations in patients with CKD, even without acute coronary syndrome (ACS) [3,4] Cardiac-specific isoforms of troponin I (cTnI) and

troponin T (cTnT) have become preferred biomarkers for diagnosing myocardial infarction because of their high sensitivity and specificity for myocardial injury. [5,6]

The severity of kidney function impairment correlates with troponin levels, as patients with an estimated glomerular filtration rate (eGFR) below 30 ml/min/1.73 m2 show a threefold increase in expected high-sensitivity troponin T compared to those with an eGFR > 60. [4] In CKD patients, troponin T levels are more frequently elevated than troponin I levels. [3] This persistent elevation poses challenges in diagnosing acute myocardial infarction, particularly in cases without ST-segment elevation. [7,8]

The increase in troponin levels is attributed to multiple factors including reduced renal clearance, subclinical myocardial injury, left ventricular hypertrophy, and inflammation. [4,8] To improve the diagnostic accuracy of ACS in patients with CKD, researchers recommend using optimized cutoffs and serial measurements. [9] These strategies aim to differentiate between chronic troponin elevation due to CKD and acute elevation indicative of ACS.

Despite these diagnostic challenges, troponin levels remain valuable prognostic indicators in patients with CKD, correlating with an increased risk of cardiovascular events and mortality. [3,10] The relationship between CKD and troponin levels extends beyond the diagnostic considerations. Studies have shown that elevated troponin levels in patients with CKD are associated with a higher risk of adverse cardiovascular outcomes including heart failure, arrhythmias, and sudden cardiac death. [3,10] This association underscores the importance of regular cardiac monitoring in patients with CKD even in the absence of acute symptoms.

This study aims to find cardiac abnormalities using ECG, 2D-ECHO and also to determine what proportion of chronic kidney disease with connective tissue disease patients will have conduction abnormalities. This study aims to highlight the needs for physicians to have a high degree of suspicion and diligently search for cardiac involvement in patients of CKD with connective tissue disease. Early intervention may greatly reduce the morbidity and mortality outcome in such patients.

Materials and Methods

The cross-sectional study was conducted in the Department of General Medicine, Department of Cardiology and Department of Nephrology in Vydehi Institute of Medical Sciences and Research Centre, over a period of 1.5 years (March 2023- August 2024). The study was started after receiving Ethical clearance to the Institutional Ethics Committee.84 subjects fulfilling the inclusion criteria were selected for the study.

Inclusion & Exclusion Criteria

Patients age more than 18 years, diagnosed with Chronic Kidney Disease stage IV, V were included.

Patients with acute kidney injury, non-sinus rhythm, history of Ischemic Heart Disease, Cardiomyopathy, Severe valvular heart disease, Acute infectious disease in past 4 weeks were excluded.

A detailed clinical evaluation was performed for each participant. Demographic data, including age and sex, were recorded along with relevant medical history such as duration of chronic kidney disease (CKD), treatment history, and drug history.

A comprehensive physical examination was conducted, including measurement of height, weight, body mass index (BMI), pulse rate, and blood pressure. Systemic examination findings were also documented.

The diagnosis of chronic kidney disease was established based on the presence of structural or anatomical abnormalities of the kidneys, persistent proteinuria, or an estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m^2 persisting for more than three months, in accordance with established diagnostic criteria.

All enrolled CKD patients underwent measurement of cardiac biomarkers, including Troponin I (Trop I) and Creatine Kinase-MB (CK-MB). In addition, laboratory investigations included complete blood count, serum urea, and serum creatinine levels. Renal function was assessed by calculating eGFR using the Modified Diet in Renal Disease (MDRD) equation.

Cardiovascular assessment comprised a 12-lead electrocardiogram (ECG) and two-dimensional echocardiography (2D ECHO) to evaluate cardiac structure and function.

The collected clinical, biochemical, and cardiovascular data were analyzed using the predefined assessment tools, and the findings were interpreted to assess the relationship between cardiac biomarkers and cardiovascular abnormalities among patients with chronic kidney disease.

Data was entered into Microsoft excel and analyzed using SPSS 22 version software. Categorical data will be represented in the form of Frequencies and proportions. **Chi-square test** will be used as test of significance for qualitative data. Continuous data will be represented as mean and standard deviation.

Independent t test will be used as test of significance to identify the mean difference between two quantitative variables. **P value** (Probability that the result is true) of <0.05 will be considered as statistically significant.

Result

Table 1. Baseline Demographic and Clinical Characteristics of the Study Participants (n = 90)

Variable	Category/Measure	Value
Age Group (years)	30–39	6 (6.7%)
	40–49	13 (14.4%)
	50–59	25 (27.8%)
	60–69	30 (33.3%)
	>70	16 (17.8%)
Gender	Male	64 (71.1%)
	Female	26 (28.9%)
Age (years)	Mean ± SD	58.93 ± 12.00
	Range	30–82
Serum Creatinine (mg/dL)	Mean ± SD	4.44 ± 2.14
	Range	2.10–12.80
eGFR (mL/min/1.73 m²)	Mean ± SD	16.20 ± 6.43
	Range	5.0–29.2
Troponin I (ng/mL)	Mean ± SD	0.369 ± 0.819
	Range	0.002–4.629
CK-MB (ng/mL)	Mean ± SD	4.04 ± 4.59
	Range	0.22–32.90

The study included 90 patients with CKD Stage IV and V, with a mean age of 58.93 ± 12.00 years. Most participants were aged 60–69 years (33.3%), indicating a higher burden of advanced CKD among older adults. Males predominated in the study population (71.1%).

The mean serum creatinine level was 4.44 ± 2.14 mg/dL, and the mean eGFR was 16.20 ± 6.43 mL/min/1.73 m², confirming advanced renal dysfunction. The mean Troponin I and CK-MB levels were 0.369 ± 0.819 ng/mL and 4.04 ± 4.59 ng/mL, respectively. These findings suggest that the study cohort comprised predominantly older male patients with severe CKD and varying levels of cardiac biomarker elevation, providing a suitable population for assessing the association between Troponin I, CK-MB, and coronary artery disease.

Table 2: - Distribution of subjects according to age group and prevalence of CAD

	CAD		NO CAD	
	N	**%**	**N**	**%**
30-39yrs	1	16.7%	5	83.3%
40-49yrs	4	30.8%	9	69.2%
50-59yrs	7	28.0%	18	72.0%
60-69ys	10	33.3%	20	66.7%
≥70yrs	6	37.5%	10	62.5%
TOTAL	**28**		**62**	

Among the 90 CKD Stage IV and V patients, 28 (31.1%) had coronary artery disease (CAD). The prevalence of CAD increased with age, from 16.7% in the 30–39 year's age group to 37.5% among patients aged over 70 years. The highest number of CAD cases was observed in the 60–69 year's age group (n=10). These findings suggest a positive association between advancing age and the occurrence of CAD in patients with advanced chronic kidney disease.

Table 3: - Distribution of subjects according to Gender and CAD

Among the study participants, CAD was present in 34.6% of females and 29.7% of males. Although females demonstrated a slightly higher prevalence of CAD compared to males, the difference was minimal. Statistical analysis revealed no significant association between gender and the presence of CAD (p = 0.820). These findings indicate that the occurrence of CAD was comparable between male and female patients with CKD Stage IV and V, suggesting that gender did not significantly influence the prevalence of CAD in the study population.

Table 4. Comparison of Renal Function Parameters Between Patients with and Without Coronary Artery Disease (CAD)

Parameter	CAD (n = 28) Mean ± SD	No CAD (n = 62) Mean ± SD	p-value
Serum Creatinine (mg/dL)	4.27 ± 1.75	4.50 ± 2.30	0.645
eGFR (mL/min/1.73 m²)	16.05 ± 6.23	16.26 ± 6.56	0.884

The mean serum creatinine level was 4.28 ± 1.75 mg/dL in patients with CAD and 4.51 ± 2.30 mg/dL in those without CAD. Similarly, the mean eGFR was 16.05 ± 6.23 mL/min/1.73 m² among patients with CAD and 16.27 ± 6.56 mL/min/1.73 m² among those without CAD. The differences in serum creatinine (p = 0.645) and eGFR (p = 0.884) between the two groups were not statistically significant. These findings indicate that the degree of renal dysfunction was comparable in patients with and without CAD, suggesting that differences in cardiac biomarker levels were unlikely to be attributable to variations in kidney function.

	CAD		NO CAD	
	N	%	N	%
Male	19	29.7%	45	70.3%
Female	9	34.6%	17	65.4%

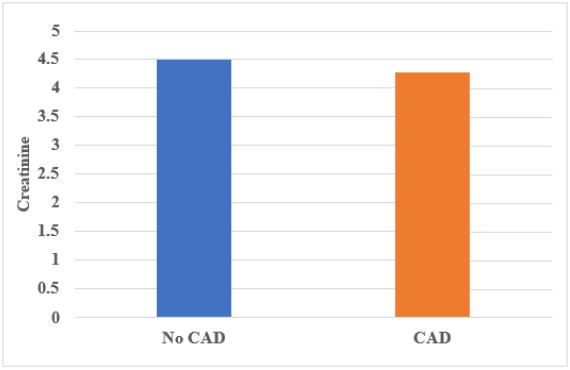

Figure 1:- Graph showing Comparison of mean creatinine among subjects with CAD and without CAD.

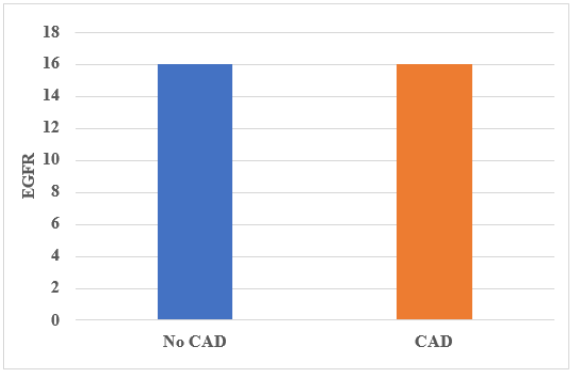

Figure 2: - Graph showing Comparison of mean EGFR among subjects with CAD and without CAD.

Table 5. Comparison of Cardiac Biomarkers Between Patients with and Without CAD

Biomarker	CAD (Mean ± SD)	No CAD (Mean ± SD)	p value
Troponin I (ng/mL)	1.10 ± 1.18	0.04 ± 0.05	<0.001
CK-MB (ng/mL)	4.71 ± 6.47	3.73 ± 3.44	0.352

Patients with CAD had significantly higher mean Troponin I levels (1.10 ± 1.18 ng/mL) compared to

those without CAD (0.04 ± 0.05 ng/mL), and this difference was highly statistically significant ($p < 0.001$). This finding suggests a strong association between elevated Troponin I levels and the presence of CAD in patients with CKD Stage IV and V.

In contrast, the mean CK-MB level was slightly higher in patients with CAD (4.71 ± 6.47 ng/mL) than in those without CAD (3.73 ± 3.44 ng/mL); however, the difference was not statistically significant ($p = 0.352$). These results indicate that Troponin I was a more sensitive and reliable biomarker than CK-MB for identifying CAD in patients with advanced chronic kidney disease.

Table 6. Diagnostic Performance of Troponin I for Predicting CAD in CKD Stage IV–V Patients

Parameter	Value
AUC	0.946
95% CI	0.878–0.983
p value	<0.0001
Optimal Cut-off	>0.15 ng/mL
Sensitivity (%)	89.29
Specificity (%)	98.39
PPV (%)	96.2
NPV (%)	95.3

An AUC of 0.946 indicates that TROP I has excellent discriminatory ability to distinguish subjects with CAD from those without. This value is close to 1.0 (perfect prediction) and well above 0.5 (random chance). This high AUC suggests TROP I is strongly associated with CAD, likely reflecting its role as a specific marker of myocardial injury.

Figure 3: - ROC for TROP I in predicting CAD

Discussion

Chronic Kidney Disease (CKD) is a progressive condition characterized by a gradual loss of kidney function over time. In its advanced stages—Stage IV and Stage V—patients are at a significantly higher risk of cardiovascular complications, including Ischemic Heart Disease (IHD). Among the biomarkers used to assess myocardial injury, cardiac troponin I (TROP I) and creatine kinase-MB (CK-MB) are commonly employed. However, interpreting these biomarkers in CKD patients poses a diagnostic challenge, as reduced renal clearance can lead to elevated baseline levels even in the absence of acute cardiac events. This study aims to estimate TROP I and CK-MB levels in patients with CKD Stage IV and V and to compare these levels in those with and without IHD, in order to enhance diagnostic accuracy and guide appropriate clinical management in this high-risk population.

The study population had a mean age of 58.93 ± 12.00 years, with the majority of patients belonging to the 60–69 year's age group (33.3%). Males constituted 71.1% of the study cohort. These findings indicate that advanced CKD predominantly affects older adults and males. Similar observations have been reported by Maringhini and Zoccali [1] and Nakano et al. [11], who demonstrated that the prevalence and severity of CKD increase with advancing age and are more common among males due to a combination of biological susceptibility, higher prevalence of cardiovascular risk factors, and lifestyle-related determinants.

In the present study, CAD was identified in 28 of 90 patients, corresponding to a prevalence of 31.1%. The prevalence of CAD increased progressively with age, from 16.7% in patients aged 30–39 years to 37.5% among those older than 70 years. Although this trend suggests increasing cardiovascular risk with advancing age, the differences across age groups were not statistically significant. These findings are consistent with the established understanding that both CKD and CAD share common risk factors, including hypertension, diabetes mellitus, endothelial dysfunction, oxidative stress, and chronic inflammation.

The observed prevalence of CAD is comparable to previous reports demonstrating a high cardiovascular burden among patients with advanced CKD. Herzog et al. [12] highlighted that cardiovascular disease remains the leading cause of morbidity and mortality in CKD patients, while Law et al. [2] emphasized the complex interplay between declining renal function and accelerated atherosclerosis.

The prevalence of CAD was 34.6% among females and 29.7% among males. Despite a slightly higher proportion of CAD among female participants, the difference was not statistically significant ($p = 0.820$). This finding suggests that gender did not significantly influence CAD prevalence in the present cohort of CKD Stage IV and V patients.

Previous studies have shown variable associations between gender and cardiovascular outcomes in CKD. While males generally exhibit a higher incidence of cardiovascular disease in the general population,

advanced CKD may attenuate these differences because the cardiovascular risk associated with severe renal dysfunction becomes the predominant determinant of disease occurrence. Similar observations have been reported by Herzog et al. [12], who noted that CKD-related cardiovascular risk often outweighs traditional demographic predictors.

The mean serum creatinine levels were comparable between patients with CAD (4.28 ± 1.75 mg/dL) and those without CAD (4.51 ± 2.30 mg/dL). Likewise, mean eGFR values did not differ significantly between the two groups (16.05 ± 6.23 vs. 16.27 ± 6.56 mL/min/1.73 m²). These findings indicate that the severity of renal dysfunction was similar among patients irrespective of CAD status.

The absence of significant differences in serum creatinine and eGFR supports the concept that conventional renal function markers alone are inadequate predictors of cardiovascular disease in advanced CKD. Similar conclusions were reported by Savoj et al. [13], who demonstrated that renal impairment and cardiovascular disease frequently coexist but are not directly predictive of each other. Michos et al. [9] also emphasized that cardiovascular risk assessment in CKD requires incorporation of cardiac-specific biomarkers rather than reliance solely on measures of renal function.

A major finding of the present study was the significantly higher Troponin I levels observed among patients with CAD compared to those without CAD (1.10 ± 1.18 ng/mL vs. 0.04 ± 0.05 ng/mL; $p < 0.001$). These results indicate a strong association between elevated Troponin I concentrations and the presence of CAD in patients with advanced CKD.

Troponin I is a highly sensitive and specific biomarker of myocardial injury. However, interpretation of elevated troponin levels in CKD patients remains challenging because reduced renal clearance and chronic myocardial injury may result in persistently elevated baseline values. Despite this limitation, several studies have demonstrated the diagnostic and prognostic value of Troponin I in CKD populations.

Mueller [14] reported that elevated Troponin I levels are frequently observed in CKD patients even in the absence of acute coronary events, reflecting chronic myocardial remodeling and subclinical cardiac injury. Gregg et al. [15] similarly observed that high-sensitivity Troponin I concentrations are significantly higher among CKD patients with ischemic heart disease compared with those without evidence of myocardial ischemia.

Further support comes from Kraus et al. [8] and Dubin et al. [4], who demonstrated that Troponin I retains excellent diagnostic utility when CKD-specific cut-off values are employed. These investigators suggested that diagnostic accuracy improves substantially when interpretation accounts for baseline elevations commonly observed in advanced renal dysfunction. Banerjee et al. [7] additionally highlighted the prognostic significance of elevated Troponin I in CKD, showing its association with future cardiovascular events and mortality.

Receiver operating characteristic (ROC) analysis demonstrated excellent diagnostic performance of Troponin I for predicting CAD, with an area under the curve (AUC) of 0.946 (95% CI: 0.878–0.983; $p < 0.0001$). The optimal cut-off value was >0.15 ng/mL, yielding a sensitivity of 89.29% and specificity of 98.39%. Positive and negative predictive values were 96.2% and 95.3%, respectively.

These findings indicate that Troponin I possesses excellent discriminative ability for identifying CAD in patients with CKD Stage IV and V. The very high AUC observed in the present study is comparable to findings reported by Kraus et al. [8] and Banerjee et al. [7], who demonstrated that Troponin I remains a reliable diagnostic biomarker even in the setting of advanced renal dysfunction. The high specificity observed in the present study further supports its clinical utility in distinguishing true ischemic cardiac injury from biomarker elevation attributable solely to renal impairment.

In contrast to Troponin I, CK-MB levels did not differ significantly between patients with and without CAD (4.71 ± 6.47 ng/mL vs. 3.73 ± 3.44 ng/mL; $p = 0.352$). Furthermore, CK-MB demonstrated poor diagnostic performance, with an AUC of only 0.535, indicating minimal discriminative ability. Although sensitivity remained relatively high, specificity was low, resulting in poor overall diagnostic accuracy.

These findings suggest that CK-MB is of limited value in identifying CAD among patients with advanced CKD. The lack of specificity may be attributable to non-cardiac influences on CK-MB levels, including skeletal muscle injury, metabolic abnormalities, and reduced renal clearance. Previous studies have similarly demonstrated the superiority of cardiac troponins over CK-MB for diagnosing myocardial injury in patients with renal dysfunction.

Consequently, contemporary clinical guidelines increasingly favor cardiac troponins as the preferred biomarkers for evaluation of suspected ischemic heart disease in CKD patients.

Limitations of the study include small sample size (n=90) limits statistical power of the study . Other potential confounding factors such as diabetes, anemia, and medications are not analyzed in our study. As it is a Cross section study long-term prognostic data are not included, which could have strengthened the predictive relevance of biomarkers.

Conclusion

The present study demonstrated that Troponin I is a reliable biomarker for detecting coronary artery disease in patients with CKD Stage IV and V, showing significantly higher levels in patients with CAD and excellent diagnostic accuracy. In contrast, CK-MB exhibited limited diagnostic utility and poor discriminatory performance. Although cardiac biomarkers may be elevated in CKD due to reduced renal clearance, Troponin I remains a valuable tool for identifying myocardial ischemia when interpreted alongside clinical findings, ECG, and echocardiographic evidence. Further multi-centric studies are required to establish CKD-specific diagnostic thresholds and optimize the use of cardiac biomarkers in this high-risk population.

References

1) Maringhini, S., & Zoccali, C. (2024). Chronic Kidney Disease Progression-A Challenge. Biomedicines, 12(10).
2) Law, J. P., Ferro, C. J., Pickup, L., Pavlovic, D., & Townend, J. N. (2022). Hypertension and cardiomyopathy associated with chronic kidney disease: epidemiology, pathogenesis and treatment considerations. Journal of Human Hypertension, 37(1), 1–19.
3) Abbas, N. A., John, R. I., Webb, M. C., Kempson, M. E., Potter, A. N., Price, C. P., Vickery, S., & Lamb, E. J. (2005). Cardiac Troponins and Renal Function in Nondialysis Patients with Chronic Kidney Disease. Clinical Chemistry, 51(11), 2059–2066.
4) Dubin, R. F., Investigators, T., Yang, W., Li, Y., Mishra, R., Keane, M., Lash, J. P., Go, A. S., Makos, G., Rosas, S. E., Shlipak, M. G., Wolf, M., Jaar, B. G., He, J., Kallem, R., Townsend, R. R., Defilippi, C., & Soliman, E. Z. (2013). Predictors of high sensitivity cardiac troponin T in chronic kidney disease patients: a cross-sectional study in the chronic renal insufficiency cohort (CRIC). BMC Nephrology, 14(1).
5) De Lemos, J. A. (2013). Increasingly Sensitive Assays for Cardiac Troponins. JAMA, 309(21), 2262.
6) Mair, J., Plebani, M., Hammarsten, O., Giannitsis, E., Müller, C., Möckel, M., Thygesen, K., Huber, K., Jaffe, A. S., & Lindahl, B. (2017). How is cardiac troponin released from injured myocardium? European Heart Journal: Acute Cardiovascular Care, 7(6), 553–560.
7) Banerjee, D., Perrett, C., & Banerjee, A. (2019). Troponins, Acute Coronary Syndrome and Renal Disease: From Acute Kidney Injury Through End-stage Kidney Disease. European Cardiology, 14(3), 187–190.
8) Kraus, D., Palapies, L., Von Jeinsen, B., Zeiher, A. M., Tzikas, S., Wanner, C., Münzel, T., Blankenberg, S., Drechsler, C., Bickel, C., Keller, T., Fette, G., Zeller, T., Neumann, J. T., Lackner, K. J., & Baldus, S. (2018). Cardiac Troponins for the Diagnosis of Acute Myocardial Infarction in Chronic Kidney Disease. Journal of the American Heart Association, 7(19).
9) Michos, E. D., Bass, E. B., Stacy, S. R., Yeh, H.-C., Wilson, L. M., Berger, Z., & Suarez-Cuervo, C. (2014). Prognostic value of cardiac troponin in patients with chronic kidney disease without suspected acute coronary syndrome: a systematic review and meta-analysis. Annals of Internal Medicine, 161(7), 491–501.
10) Soares, A. A., Silveiro, S. P., Eyff, T. F., Camargo, J. L., Ritter, L., & Campani,R. B. (2009). Glomerular filtration rate measurement and prediction equations. Clinical Chemistry and Laboratory Medicine, 47(9).
11) Nakano, T. (2025). Atherosclerotic Diseases in Chronic Kidney Disease. Journal of Atherosclerosis and Thrombosis, 32(2), 111–119.
12) Herzog, C. A. (2003). How to manage the renal patient with coronary heart disease: the agony and the ecstasy of opinion-based medicine. Journal of the American Society of Nephrology, 14(10), 2556–2572.
13) Savoj, J., Lombardo, D., Gallieni, M., Fusaro, M., Becerra, B., Kim, J. K., & Lau, W. L. (2019). Utility of Cardiac Biomarkers in the Setting of Kidney Disease. Nephron, 141(4), 227–235.
14) Mueller, C. (2013). Biomarkers and acute coronary syndromes: an update European Heart Journal,35(9), 552–556.https://doi.org/10.1093/eurheartj/eht530
15) Gregg, L. M., Jialal, I., & Wasserman, A. M. (2017). Troponins in patients with chronic kidney disease: Interpretation and prognostic significance. Journal of Clinical Pathology, 70(10), 807–812.