OPEN ACCESS
DOI: https://doi.org/10.70905/bmcj.07.01.0644

ORIGINAL ARTICLE

CHRONIC KIDNEY DISEASE AND DIAGNOSTIC ACCURACY OF HIGH-SENSITIVITY TROPONIN I IN ACUTE CORONARY SYNDROME

Naseer Ahmed[1], Mohsin Shabir[2], Mazhar Mahmood[3], Shah Khalid Mubarak[4], Salman Khan[5]

[1] Trainee Medical Officer, Department of Cardiology, Rehman Medical Institute, Peshawar, Pakistan
[2] Assistant Professor, Department of Cardiology, Rehman Medical Institute, Peshawar, Pakistan
[3] Assistant Professor, Department of Cardiology, Rehman Medical Institute, Peshawar, Pakistan
[4] Consultant cardiology, Department of Cardiology, Rehman Medical Institute, Peshawar, Pakistan
[5] Trainee Medical Officers, Department of Cardiology, Rehman Medical Institute, Peshawar, Pakistan

Correspondence:
Dr. Naseer Ahmad
Email: dr.naseer99@gmail.com

Abstract

Background: Acute coronary syndrome (ACS) is a major cause of heart-related illness and death. In patients with chronic kidney disease (CKD), it is hard to interpret high-sensitivity troponin I (hs-TnI) results because baseline levels may be consistently high.

Objective: To determine how the stage of CKD impacts the diagnostic accuracy of hs-TnI in patients being checked for ACS.

Material and Methods: This cross-sectional study took place in the Cardiology Department of Rehman Medical Institute, Peshawar, from January to April 2025. Out of 558 cardiac patients screened, 145 adults with CKD and symptoms of ACS were included. We recorded clinical symptoms, 12-lead electrocardiography, estimated glomerular filtration rate (eGFR), CKD stage, dialysis status, other health conditions, and hs-TnI levels. Data were analysed using SPSS version 27. Normality of age, eGFR, SBP, DBP, pulse pressure, and hs-TnI was assessed using the Shapiro-Wilk test and Q-Q plots. Means and standard deviations were calculated for normally distributed variables. Independent-samples t-test, one-way ANOVA, Pearson correlation, chi-square test, receiver operating characteristic analysis, and regression models were used as appropriate. A P value <0.05 was considered statistically significant.

Results: The average age of the patients was 59.48 ± 13.81 years. Among them, 91 (62.8%) were male and 54 (37.2%) were female. Patients with advanced CKD were older and had higher systolic blood pressure, diastolic blood pressure, pulse pressure, and ST-segment depression. hs-TnI levels varied across CKD stages according to one-way ANOVA ($F = 5.126$, $P < 0.001$), with the highest readings in stage 6. The area under the receiver operating characteristic curve was 0.594, showing weak ability to distinguish ACS in this CKD group..

Conclusion: hs-TnI levels increase with worsening CKD, so they should be interpreted carefully in this group. Diagnosing ACS in CKD patients should rely on the overall clinical situation, electrocardiographic results, and changes in biomarkers over time, rather than just one hs-TnI value.

Keywords: Chronic kidney disease; acute coronary syndrome; hs-TnI; eGFR; Diagnosis.
Cite this article: Ahmad N, Shabir M, Mahmood M,Mubarak K S,Khan S. Chronic Kidney Disease And Diagnostic Accuracy Of High-Sensitivity Troponin I In Acute Coronary Syndrome. BMC J Med Sci. 2026; 7(1):101-106 https://bmcjms.org/index.php/bmcj/issue/view/14

Introduction:

The acute coronary syndrome is a condition that entails ST-elevation myocardial infarction, non-ST elevation myocardial infarction, and unstable angina. The ACS usually results from an acute decrease in coronary perfusion due to plaque rupture, thrombosis, or imbalance between supply and demand of coronary arteries [1,2]. Important risk factors involved include smoking, hypertension, diabetes mellitus, hyperlipidemia, obesity, family history, and sedentary lifestyle [3,4].

Cardiovascular diseases present a huge problem within the community of Pakistan. A study conducted at a national level in Pakistan showed an occurrence of 918.18 cases/100,000 individuals and deaths of 357.88 individuals/100,000 due to CVDs [5]. Acute coronary syndrome occurred among 8.43% patients visiting the tertiary care ED of Karachi with cardiac symptoms [6].

Authorship Contribution: [1,5]Substantial contributions to the conception or design of the work; or the acquisition, Data analysis, Literature review, [2]Drafting the work or revising it critically for important intellectual content, [3,4,6]Final approval of the version to be published, Topic Selection & Supervision

Funding Source: none
Conflict of Interest: none

Received:April,23, 2026
Accepted: May,26, 2026
Published: June,30,6,2026

CKD occurs when there are persistent abnormalities in structure or function of kidneys for more than 3 months. CKD according to KDIGO can be determined by reduced estimated glomerular filtration rate (eGFR), albuminuria, abnormalities of urine sediment, and structural abnormalities on imaging studies [7]. Patients with CKD have increased chances of heart diseases due to uraemia, endothelial dysfunction, platelet activation, inflammation, and accelerated atherosclerosis [8,9].

The diagnosis of ACS among patients suffering from CKD is complicated by the fact that ECG reading may be influenced by left ventricular hypertrophy and electrolyte imbalance, while levels of cardiac troponins might be high due to decreased renal excretion and myocardium injury. High sensitivity Troponin I (hs-TnI) still can be used for diagnosing ACS; however, its specificity in cases of severe CKD and hemodialysis therapy is lowered.

Current literature concerning the value of hs-TnI test in diagnosing ACS in CKD patients is contradictory as some authors support its use, while others note the baseline level elevation and its independence from ACS development. The current research aimed at investigating the influence of CKD stages on the interpretation of hs-TnI results among patients suspected of having ACS.

Material and Method:

Study design and setting: This cross-sectional study was conducted in the Cardiology Department of Rehman Medical Institute (RMI), Peshawar, from January 2025 to April 2025. The study was approved by the Institutional Ethical Review Board of RMI (approval number: REC-RMI 113/12/24, dated 2 January 2025).

Study population: Adult patients were eligible because ACS diagnostic algorithms, consent procedures, and CKD staging in this protocol were designed for patients aged 18 years and older. During the study period, 558 patients presenting with cardiac symptoms were screened, and 145 patients with CKD and suspected ACS fulfilled the eligibility criteria.

Inclusion criteria:

- Age 18 years or older.
- Documented CKD for at least three months or eGFR-based CKD staging supported by the clinical record.
- Clinical presentation suggestive of ACS, including chest pain, dyspnoea, syncope, ST-segment depression, or T-wave inversion.

Exclusion criteria:

- Cardiac surgery within the past 30 days.
- Sepsis or acute infection.
- Strenuous physical exertion within the last 48 hours.
- Major psychological stress or emotional upset.
- ST-elevation myocardial infarction requiring primary PCI, since such patients were considered emergency cases and were not managed following standard diagnostic procedures.

Clinical evaluation and CKD categorization: Upon presentation, SBP, DBP, pulse pressure, symptoms, co-morbidity, dialysis, and demographic information were documented. The patient's ST depression and T-wave inversion were assessed by a 12-lead ECG. eGFR was estimated using the CKD-EPI formula. CKD staging was carried out according to the KDIGO eGFR classifications. Advanced CKD was categorized as those with eGFR<30 mL/min/1.73 m2, which corresponds to KDIGO Stages 4 and 5; in the present cohort, advanced CKD was analyzed separately from other CKD stages.

Definition of ACS: ACS was diagnosed according to European Society of Cardiology and ACCF/AHA recommendations [21,22]. Diagnosis required compatible ischemic symptoms, ischemic ECG changes, or a rise and/or fall in cardiac troponin with at least one value above the 99th percentile upper reference limit. Serial troponin measurements were obtained at presentation and repeated after 3-6 hours when clinically indicated to account for chronically elevated baseline values in CKD.

Troponin assay: Blood samples for hs-TnI were collected by venipuncture and analysed using an automated immunoassay analyser with the double-antibody sandwich immunoassay method (Abbott Architect STAT High Sensitive Troponin-I assay). The 99th percentile upper reference limits were 26 ng/L for males and 16 ng/L for females, with a coefficient of variation below 10% at these thresholds. Turnaround time ranged from 55 to 85 minutes.

Ethical considerations: Written informed consent was obtained from each patient. If a patient was unable to provide consent, consent was obtained from an accompanying caregiver. Patient identifiers were coded to maintain confidentiality.

Statistical analysis: Data were entered in Microsoft Excel and analysed using SPSS version 27. Normality of age, eGFR, SBP, DBP, pulse pressure, and hs-TnI was assessed using the Shapiro-Wilk test and Q-Q plots. Means and standard deviations were calculated for normally distributed variables. Independent-samples t-test, one-way ANOVA, Pearson correlation, chi-square test, receiver operating characteristic analysis, and regression models were used as appropriate. A P value <0.05 was considered statistically significant.

Results:

The sample size was calculated using the finite population correction formula, considering a total accessible population of 558 patients admitted with clinical features suggestive of acute coronary syndrome during the study period. A 95% confidence

level, 50% anticipated proportion, and 7.5% margin of error were used. The calculated sample size was approximately 131 patients. After adding 10% to account for incomplete records or non-response, the required sample size was increased to 144 patients. Therefore, the final enrolled sample of 145 patients was considered adequate for analysis.

During the study period, 558 patients were admitted to the cardiovascular department with clinical signs and symptoms related to ACS. Upon thorough investigation, ECG analysis, hs-Tn1, and GFR, 145 patients with CKD were reported to have ACS. Among the studied patients,62% (91/145) were male and 38% (54/145) were female participants. Among the patients, 85% had CKD, and 15% were reported to have advanced CKD. The mean age of the participants was 59.48±13.81; the mean age in the CKD patients was 57.28±14.38 and 66.94±8.15 in patients with advanced CKD. Patient socioeconomic characteristics and clinical parameters have been reported in Table 1.

Table 1. Demographic and clinical characteristics of patients with CKD and advanced CKD

Characteristics	Total (n=145)	CKD (n=112)	Advanced CKD (n=33)	P value
Age, years	59.48 ± 13.81	57.28 ± 14.38	66.94 ± 8.15	<0.001
Age groups, n	5	5	0	<0.001
18-30 years	15	15	0	
31-45 years	50	43	7	
46-60 years	75	49	26	
≥61 years				
Gender, n (%)	91 (62.8)	68	23	0.938
Male	54 (37.2)	44	10	
Female				
Body mass index, n	7	5	2	0.147
Underweight	57	49	8	
Normal weight	48	37	11	
Overweight	33	21	12	
Obesity				
Systolic blood pressure, mmHg	154.04 ± 18.38	146.90 ± 14.05	178.27 ± 7.21	<0.001
Diastolic blood pressure, mmHg	93.43 ± 9.02	90.41 ± 7.84	103.70 ± 3.45	<0.001
Pulse pressure, mmHg	60.60 ± 10.04	56.49 ± 7.05	74.57 ± 4.58	<0.001
ST-segment depression, n	109	79	30	<0.001
Present	36	33	3	
Absent				
T-wave inversion, n	130	103	27	0.083
Present	15	9	6	
Absent				
Chest pain, n	117	90	27	0.174
Present	28	22	6	
Absent				
Syncope, n	13	12	1	0.852
Present	132	100	32	
Absent				
Shortness of breath, n	42	35	7	0.264
Present	103	77	26	
Absent				
Glomerular filtration rate, mL/min/1.73 m²	55.92 ± 29.39	67.04 ± 23.76	18.21 ± 5.42	0.206
CKD stage by eGFR, n	21	21	0	<0.001
>90 mL/min	41	41	0	
60-89 mL/min	28	28	0	
45-59 mL/min	22	22	0	
30-44 mL/min	23	0	23	
15-29 mL/min	10	0	10	
<15 mL/min				
Dialysis, n	9	3	6	0.005
Yes	136	109	27	
No				
hs-TnI, ng/L	29.25 ± 9.53	28.47 ± 9.59	31.88 ± 9.00	0.397
Hypertension, n	81	61	20	0.067
Present	64	51	13	
Absent				
Diabetes mellitus, n	67	57	10	0.354
Present	78	55	23	
Absent				
Hyperlipidemia, n	51	37	14	0.032
Present	94	75	19	
Absent				
Smoking history, n	79	53	26	0.019
Present	66	59	7	
Absent				

Means were compared for hs-Tnlin six stages of CKD, which showed 77.2% of patients inCKD and 22.8% with advanced CKD. Stagewise increase was reported in hs-Tnllevels in patients with CKD, as reported in Figure 1.

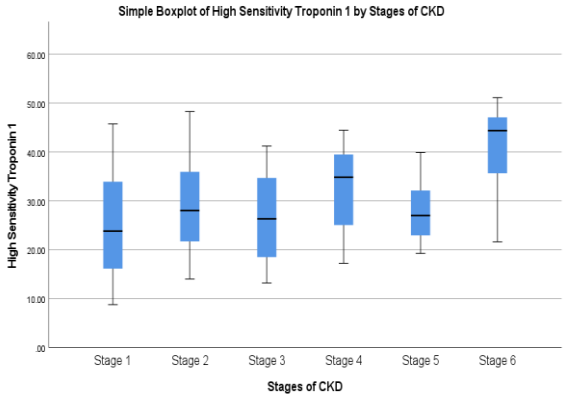

Figure I . Mean high-sensitivity troponin I (hs-TnI, ng/L) across chronic kidney disease (CKD) stages defined by estimated glomerular filtration rate (eGFR, mL/min/1.73 m²).

One-Way ANOVA was applied to look for the significance of hs-Tnllevel differences among six stages of CKD. Which showed a P<0.01 significant difference among stages, as reported in Table 2

Table 2. One-way ANOVA for hs-TnI levels across CKD stages

Source of variation	Sum of squares	df	Mean square	F	P value
Between groups	2039.557	5	407.911	5.126	<0.001
Within groups	11061.496	139	79.579		
Total	13101.053	144			

Post-Hoc analysis was carried out, which showed only Stage 6, eGFR<15mL/min, with a significant difference (P<0.01) when compared with other stages. The significantly higher Troponin level only in the advanced stage of CKD was further analysed with the ROC Curve, reporting Area Under the Curve (AUC) of 0.594, which lacks the credibility of differentiating the higher hs-Tnllevel in the advanced stage of CKD, to be influenced by ACS, as reported in Figure 2.

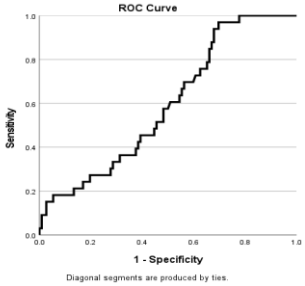

Figure 2. Receiver operating characteristic (ROC) curve showing the diagnostic performance of hs-TnI for ACS in patients with CKD. AUC, area under the curve; ACS, acute coronary syndrome.

The independent samples T-test showed no significant difference (P=0.397) for hs-Tnllevels between the CKD (stages 3-4) and the Advanced CKD (stages 5-6) groups, thus it affirmed the same findings from the ANOVA, which showed no significant difference among the groups from stage 1-5, although stage 6 was significantly different. Linear association between GFR and hs-TnI was evaluated with Pearson correlation, which was reported as P=0.052, a borderline significant value, affirming no linear association. Although the -0.162, the negative correlation coefficient, is suggestive that when the GFR reduces in the patients, the hs-TnI level increases.

Partial correlation was applied by controlling the covariates of Age, Systolic and diastolic blood pressures, pulse pressure and diabetes mellitus, although the model was unable to compute the correlation, as the GFR has been associated with the covariates.

The Chi-square was applied to look for the mean difference of presenting symptoms, chest pain, syncope and shortness of breath in patients with CKD and Advanced CKD, showing no significant difference as reported in Table 1.

Binary logistic Regression was applied to look for the prediction of hs-Tnlin patients with CKD. The model was applied with the entered method, and HTN was entered at the first block, age and gender in the second block, HTN, DM and hyperlipidemia in the third block.

Table 3. Logistic regression model for predictors associated with elevated hs-TnI in patients with CKD

Predictor	B	Wald	df	P value	Exp(B)	95% CI
hs-TnI	0.038	3.207	1	0.073	1.039	0.009-1.093
Age	0.066	10.706	1	0.001	1.068	1.026-1.110
Gender	-0.188	0.171	1	0.679	0.829	0.335-2.098
Hypertension	-0.022	0.003	1	0.960	0.978	0.411-2.329
Diabetes mellitus	-0.947	0.450	1	0.081	0.388	0.161-0.938
Hyperlipidaemia	0.111	0.062	1	0.803	1.117	0.467-2.670

The regression model showed the increase in Age (P=0.001) OR=1.608 (CI 1.026-1.110) as the only predictor of hs-Tnlwith an increase in CKD patients, as reported in Table 2. Linear regression was applied to look for the impact of GFR (P=0.206), HTN (P=0.662), and DM (P=0.636) on the hs-Tnllevels, which was not found to be significant for any predictor.

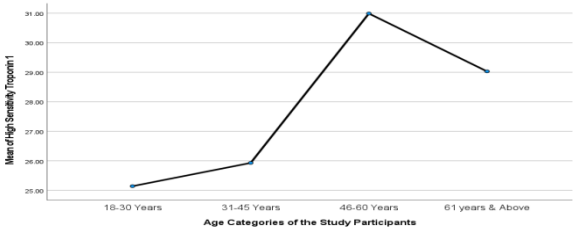

Figure III. Distribution of hs-TnI (ng/L) by dialysis status. hs-TnI, high-sensitivity troponin I.

One-Way ANOVA for mean difference in hs-TnIlevels across age groups of the study participants, with high variability, although there was no significant difference reported (P=0.218), as illustrated in Figure 4. In the same way, no significant difference (P=0.147) was reported for hs-TnIlevels across the four categories of BMI, despite hs-TnIlevels increasing respectively with each category of BMI, as illustrated in Figure 5.

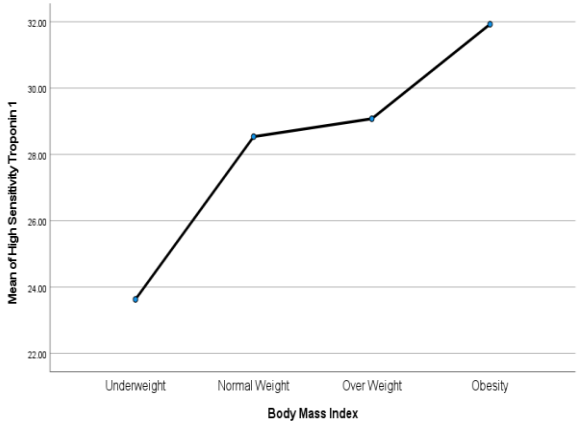

Figure IV. Mean hs-TnI (ng/L) across body mass index (BMI) categories.

Pearson correlation was highly significant (P<0.001) with a correlation coefficient of 0.318 for systolic BP and 0.343 for diastolic BP, respectively. Thus, for the pulse pressure, the correlation coefficient was reported as 0.723 with hs-TnIlevels for pulse pressure with a level of significance P<0.001, as reported in Table 1.

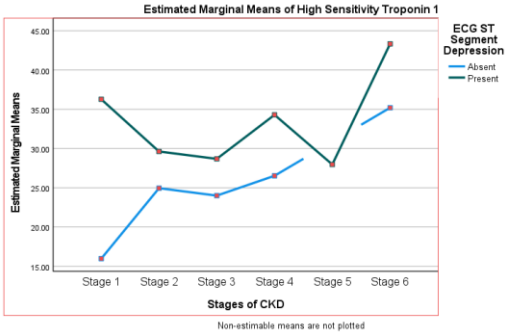

Figure V. Estimated Marginal Means of High-Sensitivity Troponin I Across CKD Stages Stratified by ST-Segment Depression

Univariate analysis was carried out with the General Linear Model to assess the level of hs-TnI in patients with and without ST-segment depression. The model reported a significance level of P<0.001 for the association of ST segment depression with CKD stages and P=0.009 with Troponin levels. For smoking (P=0.019), the association with Troponin level was higher in stages 5 and 6 of advanced CKD, but not significant in the lower stages, Troponin levels in advanced CKD were not only influenced by the CKD stage but the smoking status as well.

For T-wave inversion, no significant association (P=0.083) was reported no significant association with hs-TnIlevels, as there were only 10.3% (15/145) cases reported with T-wave inversion. In the same way, for Dialysis the model (P=0.154) was not significant, although mean hs-Tn1 levels in patients with dialysis were 43.08 ng/L and 28.28 ng/L in patients without dialysis, significantly higher by 14.8 ng/L. However, the General linear model for HTN (P=0.067), DM (P=0.354), and hyperlipidemia (P=0.958) was non-significant as well.

The main findings of the study showed that one-way ANOVA reported a significant difference in hs-TnI levels across CKD stages, with stage 6 showing significantly higher levels (P < 0.001). although when stage 5 and stage 6 were combined as dichotomous variable in the advanced CKD, the hs-TnIlevel drops due to lower levels of hs-Tn1 in stage 5, and thus the T-test showed no significant difference (P=0.071) between CKD and advanced CKD. Thus, another important finding was the ST depression and CKD stage interaction, which was influenced by the age of the study participants.

Discussion:

The primary observation made from the present study is that hs-TnI increased according to the progression of CKD, although its predictive power for ACS was poor in CKD patients. The ROC analysis revealed an AUC of 0.594, confirming that a one-time measurement of hs-TnI cannot be used to diagnose ACS in this patient group. Therefore, clinicians should evaluate hs-TnI test results with respect to clinical presentation, ECG findings, renal status, and biomarker fluctuations.

It has been found that advanced CKD patients were significantly older and presented higher blood pressures compared to patients with early stages of CKD. These observations are in agreement with data demonstrating the association between progressive kidney disease, vascular stiffness, and heart-related risk factors [7,8]. Stage 6 CKD was the only group with a significant elevation of hs-TnI levels, which could have been caused by lower clearance, myocardial workload, LVMH, and microinjury to the heart muscle [10,11,14].

ST-segment depression remained an important ECG finding in this cohort. In CKD, ECG interpretation can be complex, but ischemic ECG changes provide clinically relevant information when troponin values are difficult to interpret. The absence of significant associations with T-wave inversion, chest pain, syncope, and shortness of breath suggests that no single clinical feature should be used independently to diagnose ACS in patients with CKD.

Age was the only independent predictor in the regression model. This finding may be explained by the cumulative burden of renal impairment, hypertension, vascular disease, and subclinical myocardial injury in older patients. Although dialysis patients had higher mean hs-TnI levels than non-

dialysis patients, the small number of dialysis patients limits firm conclusions.

Previous studies have reported reduced specificity of troponin-based diagnosis in CKD because baseline troponin elevation is common [19,20]. The present findings are consistent with those reports and emphasise that serial changes, sex-specific thresholds, and integration with ECG findings may improve diagnostic interpretation in CKD patients suspected of ACS [21,22].

Limitations: This study had several limitations. First, it was a single-centre study with a relatively small sample size, which may limit the generalizability of the findings. Second, the cross-sectional design precludes the assessment of temporal relationships between CKD progression and troponin elevation. Third, the absence of a control group without CKD prevented calculation of diagnostic accuracy metrics such as sensitivity, specificity, and predictive values. Fourth, underlying conditions that may affect troponin levels, such as vitamin B7 (biotin) supplementation, were not assessed [23,24].

Conclusion:

High-sensitivity troponin I (hs-TnI) levels increase with advancing CKD stage and are significantly higher in patients on dialysis. The predictive value of hs-TnI for ACS in patients with CKD is poor, largely due to elevated baseline levels driven by reduced renal clearance, left ventricular hypertrophy, and other non-ischemic factors. ST-segment depression on ECG remains a strong predictor of ACS in this population. Therefore, hs-TnI should not be used as a standalone diagnostic tool for ACS in patients with CKD. Instead, clinicians should interpret hs-TnI in conjunction with clinical presentation, ECG findings, and serial troponin measurements, ideally using sex-specific thresholds and considering the degree of renal impairment to improve diagnostic accuracy

References:

1. Mihajlović D, Maksimović ŽM, Dojčinović B, Banjac N. Acute coronary syndrome (STEMI, NSTEMI and unstable angina pectoris) and risk factors, similarities and differences. Scripta Medica. 2020;51(4):252-60.
2. Karaca OF, Yalman H. Pathophysiology of Acute Coronary Syndrome.
3. Hamdan DY, Alarbid TH, Ali DH, Nabulsi A. Study of risk factors for acute coronary syndrome in young people.
4. Goyal RK, Haque A, Bar M, Das S, Chatterjee PK. A Study of Cardiovascular Risk Factors in Acute Coronary Syndrome in Different Age Groups. European Journal of Cardiovascular Medicine. 2025 Nov 16;15:242-6.
5. Samad Z, Hanif B. Cardiovascular diseases in Pakistan: imagining a postpandemic, postconflict future. Circulation. 2023 Apr 25;147(17):1261-3.
6. Ahmed S, Shah GA, Saghir T, Ahmed S, Mueed A, Roy N. The Crowd of Acute Coronary Syndrome in a Rural Emergency Room of Pakistan: Distribution of Demographic, Clinical, and Angiographic Characteristics. Pakistan Heart Journal. 2022 Dec 31;55(4):351-6.
7. Levin A, Stevens PE, Bilous RW, Coresh J, De Francisco AL, De Jong PE, Griffith KE, Hemmelgarn BR, Iseki K, Lamb EJ, Levey AS. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney international supplements. 2013 Jan 1;3(1):1-50.
8. Sourabha SN, Mahesh E, Gurudev KC, MS G, Yousuff M. WCN25-1361 ACUTE CORONARY SYNDROME IN PATIENTS WITH CHRONIC KIDNEY DISEASE (PRE ESRD & ESRD)–RISK FACTORS AND CLINICAL CHARACTERISTICS. Kidney International Reports. 2025 Feb 1;10(2):S699-700.
9. Abdeldayem T, Jeyalan V, Hayat A, Memon S, Farag M, Egred M. Antithrombotic therapy in acute coronary syndrome patients with end-stage renal disease: navigating efficacy and safety. Journal of Clinical Medicine. 2025 Jun 3;14(11):3956.
10. Haider MA, Shah MU, Kassianides X, Hazara A, Ali N, Feldman DN. Diagnosis and management of ACS in patients with ESRD on haemodialysis: A comprehensive review. Br. J. Cardiol. 2025;32:7-11.
11. Westwood M, Ramaekers B, Grimm S, Worthy G, Fayter D, Armstrong N, Buksnys T, Ross J, Joore M, Kleijnen J. High-sensitivity troponin assays for early rule-out of acute myocardial infarction in people with acute chest pain: a systematic review and economic evaluation. Health Technology Assessment (Winchester, England). 2021 May;25(33):1.
12. Ebrahimi S, Alivirdiloo V, Hajiabbasi M, Masoumi S, Mohammadi M, Hosseini S, Gargari SO, Mobed A. Cardiac troponin I biosensors: innovations in real-time diagnosis of cardiovascular diseases. Analytical Science Advances. 2025 Jun;6(1):e70009.
13. Mendonça L, Bigotte Vieira M, Neves JS, Castro Chaves P, Ferreira JP. A 4-variable model to predict cardio-kidney events and mortality in chronic kidney disease: the chronic renal insufficiency cohort (CRIC) study. American Journal of Nephrology. 2023 Nov 27;54(9-10):391-8..
14. Defilippi CR, Herzog CA. Interpreting cardiac biomarkers in the setting of chronic kidney disease. Clinical chemistry. 2017 Jan 1;63(1):59-65.
15. Twerenbold R, Boeddinghaus J, Nestelberger T, Wildi K, Rubini Gimenez M, Badertscher P, Mueller C. Clinical use of high-sensitivity cardiac troponin in patients with suspected myocardial infarction. Journal of the American College of Cardiology. 2017 Aug 22;70(8):996-1012.
16. Sandoval Y, Jaffe AS. Type 2 myocardial infarction: JACC review topic of the week. Journal of the American College of Cardiology. 2019 Apr 16;73(14):1846-60.
17. Zheng P, Wang X, Guo T, Gao W, Huang Q, Yang J, Gao H, Liu Q. Cardiac troponin as a prognosticator of mortality in patients with sepsis: a systematic review and meta-analysis. Immunity, Inflammation and Disease. 2023 Sep;11(9):e1014.
18. Chauin A. The main causes and mechanisms of increase in cardiac troponin concentrations other than acute myocardial infarction (part 1): physical exertion, inflammatory heart disease, pulmonary embolism, renal failure, sepsis. Vascular Health and Risk Management. 2021 Sep 21:601-17.
19. Güler EE, Payza U, Kayali A, Kirik S, Kanter E. Troponin efficacy in the diagnosis of acute coronary syndrome in patients with chronic renal failure. Medicine. 2024 Mar 1;103(9):e37280.
20. Noorayingarath H, Panjiyar BK, Gela I, Ramalingam L. Role of cardiac troponins in predicting adverse outcomes in acute coronary syndrome with renal dysfunction. Cureus. 2023 Oct 16;15(10).
21. Collet, J.P., Thiele, H., Barbato, E., Barthélémy, O., Bauersachs, J., Bhatt, D.L., Dendale, P., Dorobantu, M., Edvardsen, T., Folliguet, T. and Gale, C.P., 2021. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). *European heart journal*, *42*(14), pp.1289-1367.
22. Amsterdam EA, Wenger NK, Brindis RG, Casey Jr DE, Ganiats TG, Holmes Jr DR, Jaffe AS, Jneid H, Kelly RF, Kontos MC, Levine GN. 2014 AHA/ACC guideline for the management of patients with non–ST-elevation acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Journal of the American college of cardiology. 2014 Dec 23;64(24):e139-228.
23. Imtiaz S, Alam A. Epidemiology and demography of Chronic Kidney Disease in Pakistan-A review of Pakistani literature. Pakistan Journal of Kidney Diseases. 2023 Mar 26;7(1):2-7.
24. Michos ED, Wilson LM, Yeh HC, Berger Z, Suarez-Cuervo C, Stacy SR, Bass EB. 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. 2014 Oct 7;161(7):491-501.