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Comparison of Cardiac Biomarker Trends in Acute Kidney Injury versus Chronic Kidney Disease Patients with Acute Coronary Syndrome

Dr. Santosh Kumar Maurya[1], Dr. Nikesh Mishra[2], Dr. Abhishek Sachdeva[3*]

[1]Assistant Professor, Department of Nephrology, MLN Medical College, Prayagraj, India
[2]Assistant Professor, Department of Cardiology, MLN Medical College, Prayagraj, India
[3]Associate Professor, Department of Cardiology, MLN Medical College, Prayagraj, India

***Corresponding author:**
Dr. Abhishek Sachdeva
Associate Professor, Department of Cardiology, MLN Medical College, Prayagraj, India
Email ID: drabhisheksachdeva@gmail.com

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ABSTRACT

Background: Cardiac biomarkers such as Troponin I, CK-MB, and NT-proBNP play a pivotal role in diagnosing and prognosticating acute coronary syndrome (ACS). However, their interpretation can be significantly influenced by underlying renal dysfunction, particularly in patients with acute kidney injury (AKI) or chronic kidney disease (CKD). The varying kinetics and clearance of these biomarkers in renal impairment pose a diagnostic challenge.

Aim: To compare the trends of cardiac biomarkers—Troponin I, CK-MB, and NT-proBNP—in patients with ACS having either AKI or CKD, and to evaluate the predictive factors influencing NT-proBNP levels.

Material and Methods: This prospective, comparative observational study was conducted in the Departments of Nephrology and Cardiology at a tertiary care teaching hospital. Sixty adult patients with ACS were enrolled and categorized into two groups: Group A (AKI, n=30) and Group B (CKD, n=30), based on KDIGO 2012 criteria and MDRD-based eGFR. Demographics, comorbidities, renal parameters, and serial cardiac biomarkers (at 0, 6, 12, and 24 hours) were recorded. Left ventricular function was assessed using echocardiography. Statistical analysis included t-tests, chi-square tests, repeated measures ANOVA, and multiple linear regression.

Results: Baseline demographics and comorbidities were comparable between the AKI and CKD groups. Serum creatinine and eGFR showed significant intergroup differences ($p = 0.048$ and $p = 0.026$, respectively). Troponin I and CK-MB levels followed a similar trend in both groups, peaking at 12 hours and declining by 24 hours, with no significant differences at any point. However, NT-proBNP levels were significantly higher in the CKD group at all time points ($p < 0.001$). Multivariate regression revealed CKD status, age, serum creatinine, Killip class ≥ II, and lower LVEF as significant predictors of NT-proBNP levels, with an adjusted R^2 of 0.65.

Conclusion: Troponin I and CK-MB exhibit comparable patterns in both AKI and CKD patients with ACS. In contrast, NT-proBNP levels are significantly elevated in CKD due to chronic volume overload and impaired clearance. CKD status was the strongest predictor of NT-proBNP elevation. Hence, cardiac biomarker interpretation in ACS must consider renal function for accurate risk stratification and management.

Keywords: *Acute Coronary Syndrome, Acute Kidney Injury, Chronic Kidney Disease, Cardiac Biomarkers, NT-proBNP*

1. INTRODUCTION

Acute coronary syndrome (ACS) is a significant cause of morbidity and mortality worldwide, frequently intersecting with both acute kidney injury (AKI) and chronic kidney disease (CKD). The interplay between the heart and kidneys, known as the cardiorenal axis, has garnered increasing attention due to the bidirectional and often deleterious consequences each organ system has on the other during acute pathophysiologic events. AKI and CKD both represent distinct yet interconnected

conditions that can profoundly alter cardiovascular outcomes in patients presenting with ACS. However, the pathophysiological distinctions between these renal disorders imply potentially different implications for cardiac biomarker kinetics, diagnostic accuracy, and risk stratification in the context of ACS.AKI is characterized by a sudden decline in renal function, which can significantly modulate the clearance and serum concentration of cardiac biomarkers, particularly troponins. Troponin levels, which serve as the cornerstone for ACS diagnosis, may be falsely elevated in AKI due to decreased renal excretion rather than myocardial necrosis. This can complicate the diagnostic clarity and may lead to either overestimation of cardiac damage or delayed intervention in ambiguous cases. Moreover, emerging evidence suggests that AKI induces systemic inflammation, endothelial dysfunction, and oxidative stress, all of which can independently stimulate cardiac injury markers even in the absence of ischemia[1]. These dynamics underscore the complexity of interpreting biomarker trends in this patient population.In contrast, CKD presents a chronic milieu of sustained uremia, progressive nephron loss, and systemic atherosclerotic burden. The persistent nature of CKD-induced biochemical changes, such as the chronic elevation of baseline troponin levels, reflects not only cumulative cardiovascular strain but also subclinical myocardial injury. The clinical challenge in CKD lies not only in distinguishing baseline troponin elevations from acute-on-chronic myocardial infarction but also in interpreting fluctuations that may or may not correlate with ACS events. The longstanding cardiovascular risk burden in CKD patients often leads to structural and functional cardiac abnormalities, including left ventricular hypertrophy and diastolic dysfunction, further complicating the biomarker profile[2].Both AKI and CKD fall under the broader umbrella of cardiorenal syndromes (CRS), particularly CRS types 1 and 4, respectively. In CRS type 1, acute decompensation of cardiac function leads to a rapid deterioration in renal function, as seen in AKI superimposed on ACS. Conversely, CRS type 4 involves chronic kidney disease precipitating or worsening cardiovascular disease[3]. These syndromes illustrate how closely interwoven the cardiac and renal systems are in the pathogenesis of acute and chronic illnesses. Importantly, CRS is also associated with poorer outcomes, longer hospital stays, and higher rates of readmission, making the timely and accurate differentiation of renal status in ACS crucial for prognostication and management[4].The diagnostic implications of altered biomarker kinetics in AKI and CKD are particularly relevant for clinicians managing ACS patients. Serum creatinine, while still widely used for renal assessment, lacks sensitivity in early injury detection and may not reflect real-time kidney function changes[5]. Consequently, newer renal biomarkers—such as neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, and kidney injury molecule-1 (KIM-1)—have been explored for their ability to provide early and specific insights into renal injury, which in turn could assist in refining the interpretation of cardiac biomarkers[6]. However, their integration into clinical workflows remains limited, and their predictive value in dynamic ACS settings needs further exploration.Recent studies have emphasized the overlapping and sometimes contradictory biomarker profiles in patients with ACS and renal impairment. For example, elevation of troponin in AKI without underlying ischemia can mimic myocardial infarction, while low-level increases in CKD patients may desensitize clinicians to genuine ACS events[7]. This diagnostic gray zone necessitates a nuanced approach to evaluating troponin kinetics—ideally integrating clinical context, electrocardiographic findings, imaging studies, and sequential measurements—rather than relying on absolute values alone.Additionally, the evolving landscape of renal-specific biomarkers has prompted calls for consensus on how best to integrate them into the management of ACS patients with renal impairment. Biomarkers such as NGAL and KIM-1 have shown promise in identifying subclinical kidney injury even before changes in serum creatinine occur, offering a potential window for early intervention[8]. Nonetheless, concerns remain regarding their specificity, cost-effectiveness, and standardization in routine clinical practice[9].Furthermore, the burden of cardiorenal pathology extends beyond acute diagnosis to long-term management. CKD patients with a history of ACS are more likely to develop recurrent ischemic events, experience heart failure hospitalizations, and suffer from arrhythmias due to chronic inflammation, vascular calcification, and neurohormonal activation. Similarly, AKI episodes in the context of ACS are associated with higher in-hospital mortality and can predispose to long-term renal dysfunction, thereby potentially transitioning into CKD—a phenomenon referred to as acute kidney disease (AKD)[10].Given this intricate interdependence, a comparative assessment of cardiac biomarker trends in AKI versus CKD patients with ACS is both timely and clinically relevant. Understanding the nuanced differences in biomarker elevation patterns, peak values, time to normalization, and their correlation with clinical outcomes can enhance diagnostic precision and therapeutic decision-making. It also offers insights into patient stratification, resource allocation, and development of targeted interventions aimed at mitigating adverse outcomes in this high-risk cohort.

2. MATERIAL AND METHODS

This prospective, comparative observational study was conducted in the Department of Nephrology and Cardiology at a tertiary care teaching hospital following approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment.A total of 60 adult patients (age ≥ 18 years) admitted with a confirmed diagnosis of acute coronary syndrome (ACS)—including ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina—were included in the study. Patients were further stratified into two equal groups based on renal status:

- **Group A (n = 30):** Patients diagnosed with Acute Kidney Injury (AKI) as per the KDIGO 2012 criteria (an increase in serum creatinine by ≥ 0.3 mg/dL within 48 hours or a 1.5-fold increase from baseline within 7 days).
- **Group B (n = 30):** Patients with pre-existing Chronic Kidney Disease (CKD) stage 3 or above (eGFR < 60 mL/min/1.73

m^2 for more than 3 months, based on the MDRD equation).

Inclusion Criteria

- Adult patients (≥18 years) admitted with ACS.
- Confirmed diagnosis of either AKI or CKD based on laboratory parameters and clinical history.
- Availability of serial cardiac biomarker data (troponins, CK-MB, and NT-proBNP) at admission and 6, 12, and 24 hours post-admission.

Exclusion Criteria

- End-stage renal disease patients on maintenance dialysis.
- Patients with underlying valvular heart disease, myocarditis, or recent cardiac surgery.
- Those with incomplete data or who were unwilling to provide informed consent.

Methodology

Demographic data (age, sex), clinical parameters (blood pressure, heart rate, Killip class), and comorbidities (hypertension, diabetes, dyslipidemia) were recorded using a structured case proforma. Renal function tests, complete blood count, electrolytes, and serial cardiac biomarkers—Troponin I/T, Creatine Kinase-MB (CK-MB), and NT-proBNP—were measured at four intervals: at admission (0 hour), 6 hours, 12 hours, and 24 hours post-presentation using standardized laboratory methods.

Renal function (serum creatinine and eGFR) was assessed at baseline and monitored over 48–72 hours for AKI identification or CKD confirmation. Cardiac imaging (echocardiography) was performed within 24 hours to assess left ventricular function and structural abnormalities.

Statistical Analysis

Data were analyzed using IBM SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Intergroup comparisons were performed using Student's t-test or Mann–Whitney U test for continuous variables and Chi-square test for categorical variables. Trends in biomarker levels over time were analyzed using repeated measures ANOVA. A p-value < 0.05 was considered statistically significant.

3. RESULTS

Table 1 shows the baseline demographic and clinical characteristics of the study population. The mean age of patients in the AKI group was 59.4 ± 10.2 years, while in the CKD group it was slightly higher at 62.1 ± 9.6 years, though this difference was not statistically significant ($p = 0.216$). The gender distribution showed a male predominance in both groups (Male:Female = 21:9 in AKI vs. 19:11 in CKD; $p = 0.584$). The prevalence of comorbidities including hypertension (66.67% in AKI vs. 76.67% in CKD; $p = 0.393$), diabetes mellitus (56.67% vs. 63.33%; $p = 0.598$), dyslipidemia (43.33% vs. 50.00%; $p = 0.605$), and smoking (40.00% vs. 33.33%; $p = 0.592$) was comparable across groups with no statistically significant differences. Similarly, the percentage of patients presenting in Killip class ≥ II was higher in the CKD group (46.67%) compared to the AKI group (36.67%), but the difference did not reach statistical significance ($p = 0.424$).

Table 2 outlines the renal function and baseline laboratory parameters of both groups. Serum creatinine levels were significantly higher in CKD patients (3.1 ± 1.2 mg/dL) compared to the AKI group (2.6 ± 0.9 mg/dL), with a p-value of 0.048. Correspondingly, the mean estimated glomerular filtration rate (eGFR) was significantly lower in the CKD group (28.5 ± 9.7 mL/min/1.73 m^2) than in the AKI group (34.8 ± 10.4 mL/min/1.73 m^2; $p = 0.026$). Other laboratory parameters such as hemoglobin, serum potassium, and serum sodium showed no statistically significant intergroup differences. Hemoglobin levels were slightly lower in the CKD group (11.4 ± 1.7 g/dL) compared to the AKI group (12.1 ± 1.8 g/dL; $p = 0.144$), consistent with the known anemia of chronic kidney disease, although not statistically significant.

Table 3 demonstrates the trends in Troponin I levels over 24 hours. At baseline (admission), mean Troponin I levels were slightly higher in the AKI group (4.2 ± 2.3 ng/mL) compared to the CKD group (3.9 ± 2.1 ng/mL; $p = 0.572$). Both groups showed a rise in troponin levels at 6 and 12 hours, peaking at 12 hours in both cohorts (6.1 ± 3.0 ng/mL in AKI vs. 5.8 ± 2.6 ng/mL in CKD). Thereafter, levels slightly declined at 24 hours. However, at no point during the 24-hour assessment did the differences in Troponin I levels between the two groups reach statistical significance, indicating similar myocardial injury kinetics despite differing renal statuses.

Table 4 presents the serial values of CK-MB, another cardiac biomarker. CK-MB levels rose from baseline in both groups, peaking at 12 hours (48.3 ± 11.1 IU/L in AKI vs. 45.7 ± 10.2 IU/L in CKD), then gradually declined by 24 hours. Although mean values were slightly higher in the AKI group across all time points, the differences between the two groups were not statistically significant ($p > 0.05$ at all time points). These findings suggest that CK-MB trends mirrored Troponin I,

reinforcing similar biomarker patterns of myocardial injury in both renal dysfunction subtypes.

Table 5 reveals a striking and statistically significant difference in NT-proBNP levels between the groups. At admission, NT-proBNP was markedly elevated in CKD patients (7800.00 ± 1700.00 pg/mL) compared to AKI patients (5200.00 ± 1400.00 pg/mL; $p < 0.001$). This trend persisted consistently at 6, 12, and 24 hours, with NT-proBNP values remaining significantly higher in the CKD group throughout ($p < 0.001$ for all time points). These elevated levels likely reflect the chronic volume overload and impaired clearance in CKD patients, in addition to cardiac stress. The persistently higher NT-proBNP in CKD, despite similar troponin and CK-MB profiles, underscores its limitation in distinguishing cardiac severity in chronic renal impairment.

Table 6 details the results of a multiple linear regression analysis conducted to identify independent predictors of NT-proBNP levels at 24 hours. The regression model revealed that CKD status was the strongest independent predictor ($B = 982.40$, $\beta = 0.582$, $p < 0.001$). Additionally, age ($B = 12.35$, $p = 0.015$), serum creatinine ($B = 210.75$, $p = 0.030$), and Killip class ≥ II ($B = 765.88$, $p = 0.003$) were all significant positive predictors. Conversely, left ventricular ejection fraction (LVEF) had a significant negative association ($B = –45.62$, $\beta = –0.319$, $p < 0.001$), indicating that lower LVEF was associated with higher NT-proBNP levels. The model had an adjusted R^2 of 0.65, indicating it explained 65% of the variance in NT-proBNP levels at 24 hours, suggesting strong predictive power.

Table 1: Baseline Demographic and Clinical Characteristics of Study Population (n = 60)

Parameter	Group A (AKI, n = 30)	Group B (CKD, n = 30)	p-value
Age (years, mean ± SD)	59.4 ± 10.2	62.1 ± 9.6	0.216
Male : Female ratio	21 : 9	19 : 11	0.584
Hypertension (%)	20 (66.67%)	23 (76.67%)	0.393
Diabetes Mellitus (%)	17 (56.67%)	19 (63.33%)	0.598
Dyslipidemia (%)	13 (43.33%)	15 (50.00%)	0.605
Smoking (%)	12 (40.00%)	10 (33.33%)	0.592
Killip Class ≥ II (%)	11 (36.67%)	14 (46.67%)	0.424

Table 2: Renal Function and Baseline Laboratory Parameters

Parameter	Group A (AKI)	Group B (CKD)	p-value
Serum Creatinine (mg/dL)	2.6 ± 0.9	3.1 ± 1.2	0.048*
eGFR (mL/min/1.73 m²)	34.8 ± 10.4	28.5 ± 9.7	0.026*
Hemoglobin (g/dL)	12.1 ± 1.8	11.4 ± 1.7	0.144
Serum Potassium (mmol/L)	4.8 ± 0.6	4.9 ± 0.5	0.501
Serum Sodium (mmol/L)	137.2 ± 3.4	136.5 ± 3.8	0.472

*Statistically significant at $p < 0.05$

Table 3: Trends in Troponin I Levels (ng/mL) Over 24 Hours

Time Point	Group A (AKI) (Mean ± SD)	Group B (CKD) (Mean ± SD)	p-value
At admission	4.2 ± 2.3	3.9 ± 2.1	0.572
6 hours	5.6 ± 2.7	5.2 ± 2.3	0.524
12 hours	6.1 ± 3.0	5.8 ± 2.6	0.664
24 hours	5.4 ± 2.6	5.0 ± 2.5	0.581

Table 4: Trends in CK-MB Levels (IU/L) Over 24 Hours

Time Point	Group A (AKI) (Mean ± SD)	Group B (CKD) (Mean ± SD)	p-value
At admission	35.6 ± 9.2	33.9 ± 8.7	0.452
6 hours	45.1 ± 10.3	42.8 ± 9.6	0.341
12 hours	48.3 ± 11.1	45.7 ± 10.2	0.298
24 hours	40.5 ± 9.8	39.1 ± 9.3	0.591

Table 5: Trends in NT-proBNP Levels (pg/mL) Over 24 Hours

Time Point	Group A (AKI) (Mean ± SD)	Group B (CKD) (Mean ± SD)	p-value
At admission	5200.00 ± 1400.00	7800.00 ± 1700.00	<0.001*
6 hours	5400.00 ± 1500.00	7900.00 ± 1600.00	<0.001*
12 hours	5500.00 ± 1300.00	8000.00 ± 1800.00	<0.001*
24 hours	5400.00 ± 1250.00	8100.00 ± 1900.00	<0.001*

Table 6: Multiple Linear Regression Analysis for Predictors of NT-proBNP Levels at 24 Hours

Independent Variable	Unstandardized Coefficient (B)	Standard Error (SE)	Standardized Coefficient (β)	t-value	p-value
Age (years)	12.35	4.92	0.262	2.51	0.015*
Group (AKI = 0, CKD = 1)	982.40	221.50	0.582	4.43	<0.001*
Serum Creatinine (mg/dL)	210.75	94.62	0.216	2.23	0.030*
LVEF (%)	–45.62	11.24	–0.319	–4.06	<0.001*
Killip Class ≥ II (Yes = 1)	765.88	242.10	0.341	3.16	0.003*

4. DISCUSSION

The demographic and clinical profiles between the AKI and CKD groups were broadly comparable. The mean age was 59.4 ± 10.2 years in the AKI group and 62.1 ± 9.6 years in the CKD group, with male predominance in both. The prevalence of hypertension (66.67% in AKI vs. 76.67% in CKD), diabetes mellitus (56.67% vs. 63.33%), and dyslipidemia (43.33% vs. 50.00%) was not significantly different. These findings are consistent with previous studies such as that by **Moisi et al.** (2020)[11], who reported that patients with CKD and ACS tend to be older and have a higher burden of comorbidities including hypertension and diabetes. In our study, although not statistically significant, Killip class ≥ II was observed more frequently in CKD patients (46.67%) than in those with AKI (36.67%), supporting the findings by **Chen et al.** (2020)[12] who demonstrated more frequent cardiac decompensation among CKD patients admitted with ACS. This reflects the chronic cardiovascular stress in CKD, which predisposes patients to worse functional class on presentation.

Serum creatinine was significantly higher in the CKD group (3.1 ± 1.2 mg/dL) compared to the AKI group (2.6 ± 0.9 mg/dL), and eGFR was significantly lower in CKD (28.5 ± 9.7 vs. 34.8 ± 10.4 mL/min/1.73 m²). These differences are expected given the chronicity of kidney injury in CKD. Similar trends were observed in the cohort studied by **Chang et al.** (2015)[13], where patients with CKD had persistently reduced eGFR and higher serum creatinine compared to those with AKI. Interestingly, although our study showed no statistically significant difference in hemoglobin levels (12.1 ± 1.8 g/dL in AKI vs. 11.4 ± 1.7 g/dL in CKD), this numerical reduction supports the concept of renal anemia in CKD as emphasized by **Ronco et al.** (2008)[14], where anemia serves as both a marker and a contributor to the cardiorenal burden.

Troponin I kinetics showed peak levels at 12 hours in both groups (6.1 ± 3.0 ng/mL in AKI vs. 5.8 ± 2.6 ng/mL in CKD), followed by a decline at 24 hours. While baseline levels were higher in AKI (4.2 ± 2.3 ng/mL) than in CKD (3.9 ± 2.1 ng/mL), differences were not statistically significant. This mirrors the findings of **Banerjee et al.** (2019)[15], who concluded that troponin elevations in renal impairment are common but not always proportional to the extent of myocardial damage

due to altered clearance and baseline cardiac stress. In our study, the similar troponin kinetics suggest that both AKI and CKD patients with ACS experience comparable myocardial injury patterns. A comparable trajectory was documented by **Chen et al.** (2020)[12], who showed that troponin rises and falls were similar in both renal groups, indicating that renal dysfunction may affect biomarker clearance more than injury magnitude.

CK-MB levels peaked at 12 hours (48.3 ± 11.1 IU/L in AKI vs. 45.7 ± 10.2 IU/L in CKD) with subsequent decline. At all time points, levels were slightly higher in AKI, though not statistically significant. These findings are in line with observations from **Thygesen et al.** (2012)[16], who noted that while CK-MB is less affected by renal clearance than troponin, its clinical interpretation still requires contextual assessment. Our findings reinforce the conclusion that CK-MB trends parallel troponin kinetics, and neither biomarker alone can adequately differentiate between AKI and CKD in terms of ACS-related myocardial injury severity.

NT-proBNP levels were significantly higher in CKD patients at all time points (admission: 7800.00 ± 1700.00 pg/mL in CKD vs. 5200.00 ± 1400.00 pg/mL in AKI, $p < 0.001$). These persistently elevated levels in CKD are likely due to both decreased renal clearance and chronic cardiac strain. **Ho et al.** (1993)[17] reported that elevated NT-proBNP levels are strongly associated with worse prognosis in heart failure, though this marker may be confounded in renal patients. **Ronco et al.** (2008)[14] emphasized that in cardiorenal syndrome, elevated NT-proBNP reflects a combination of left ventricular dysfunction and impaired natriuretic peptide clearance. Our results confirm this, suggesting that while NT-proBNP is a sensitive marker for volume overload and cardiac stress, it lacks specificity in the context of CKD. Compared to **Chen et al.** (2012)[18], who found moderate elevations in NT-proBNP in AKI patients, our study shows that chronicity plays a larger role in BNP elevation than the acute renal insult itself.

Multivariate regression findings demonstrated CKD status as the strongest independent predictor of NT-proBNP levels at 24 hours ($B = 982.40$, $\beta = 0.582$, $p < 0.001$). Other significant predictors included age ($B = 12.35$, $p = 0.015$), serum creatinine ($B = 210.75$, $p = 0.030$), and Killip class ≥ II ($B = 765.88$, $p = 0.003$), while LVEF was inversely related ($B = –45.62$, $\beta = –0.319$, $p < 0.001$). These findings are in line with **Chang et al.** (2015)[13], who demonstrated that NT-proBNP levels are modulated by both renal function and cardiac performance. Similarly, **Parikh et al.** (2006)[19] found that renal dysfunction independently contributed to biomarker elevations, especially NT-proBNP and urinary biomarkers. The high explanatory power of the model (adjusted $R^2 = 0.65$) supports its robustness and clinical utility in predicting NT-proBNP levels based on combined renal and cardiac variables. These findings stress the importance of integrated cardiorenal assessment when using NT-proBNP to prognosticate or guide therapy in ACS patients[20-30].

5. CONCLUSION

In conclusion, this study demonstrates that while cardiac biomarkers such as Troponin I and CK-MB exhibit similar trends in both AKI and CKD patients with acute coronary syndrome, NT-proBNP levels are significantly higher in CKD due to chronic volume overload and impaired clearance. CKD status emerged as the strongest independent predictor of NT-proBNP levels. These findings highlight the importance of interpreting cardiac biomarkers in the context of underlying renal function. Integrated cardiorenal assessment is essential for accurate diagnosis and prognostication in ACS patients with renal impairment.

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