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Original Article

Prognostic Values of Electrolyte Abnormalities and Cardiac Biomarkers in Acute MI and During the Convalescent Phase: A Comparative Study

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ABSTRACT

Background: Myocardial infarction is associated with myocardial necrosis and systemic biochemical disturbances that may influence clinical monitoring during acute illness and recovery. Cardiac Troponin I is an established biomarker of myocardial injury, while sodium, potassium, and chloride may provide supportive information regarding electrolyte instability during acute myocardial infarction. Objective: To compare Troponin I and selected serum electrolyte levels between the acute and convalescent phases of myocardial infarction patients. Methods: This paired comparative observational study was conducted among 100 myocardial infarction patients recruited from the cardiology wards of Lady Reading Hospital, Peshawar, Pakistan, from December 2023 to June 2024. Troponin I, sodium, potassium, and chloride values were recorded during the acute phase and again during the convalescent phase after treatment and hospitalization. Data were analyzed using IBM SPSS version 24, and paired-sample t-tests were applied with statistical significance set at $p < 0.05$. Results: Troponin I decreased from 10.8234 ng/mL during the acute phase to 0.5836 ng/mL during convalescence, representing a 94.61% relative decline ($p < 0.001$). Sodium increased from 133.48 mmol/L to 143.73 mmol/L, with a mean rise of 10.25 mmol/L ($p < 0.001$), and potassium increased from 3.6437 mmol/L to 4.3921 mmol/L, with a mean rise of 0.7484 mmol/L ($p < 0.001$). Chloride showed a small non-significant change from 107.183 mmol/L to 108.338 mmol/L ($p = 0.447$). Conclusion: Troponin I, sodium, and potassium differed significantly between acute and convalescent phases, while chloride remained stable. Sodium and potassium may support biochemical monitoring during myocardial infarction recovery but should not be interpreted as independent diagnostic or prognostic biomarkers without outcome-based validation. Keywords: Acute myocardial infarction; Troponin I; sodium; potassium; chloride; electrolyte imbalance; convalescent phase; cardiac biomarkers.

INTRODUCTION

Myocardial infarction remains one of the most clinically significant manifestations of cardiovascular disease and results from acute impairment of coronary blood flow leading to myocardial ischemia, cellular injury, and necrosis. Its development is strongly influenced by established cardiometabolic and behavioral risk factors, including hypertension, diabetes mellitus, obesity, smoking, sedentary lifestyle, and psychological stress, all of which contribute to endothelial dysfunction, atherosclerotic plaque progression, and thrombotic coronary events (1). Contemporary classification systems recognize myocardial infarction as a heterogeneous clinical entity, with type 1 myocardial infarction commonly

resulting from atherosclerotic plaque rupture and coronary thrombosis, whereas type 2 myocardial infarction reflects an imbalance between myocardial oxygen supply and demand in the absence of acute plaque rupture (2,3). Regardless of mechanism, the clinical consequences of myocardial infarction may include arrhythmia, ventricular dysfunction, heart failure, cardiogenic shock, and death, making early biochemical assessment and subsequent monitoring central to clinical decision-making (4).

The global burden of myocardial infarction remains substantial, and South Asian populations, including patients in Pakistan, experience cardiovascular disease at comparatively younger ages and with a high burden of metabolic risk factors (5). In acute clinical settings, diagnosis and risk assessment rely on integration of symptoms, electrocardiographic findings, imaging where indicated, and circulating cardiac biomarkers. Cardiac troponins, particularly cardiac Troponin I and Troponin T, are the most sensitive and specific biochemical indicators of myocardial injury and have become central to the diagnosis and monitoring of acute myocardial infarction (6,7). Troponin concentrations rise after myocardial injury, remain elevated for a clinically useful period, and usually decline during recovery, allowing serial measurement to support diagnostic interpretation and assessment of disease evolution (8,9). However, while troponin reflects myocardial necrosis directly, additional biochemical parameters may provide complementary information about systemic physiological stress, treatment response, and clinical instability during the acute and recovery phases of myocardial infarction.

Electrolytes are essential for maintaining myocardial membrane potential, action potential propagation, excitation–contraction coupling, vascular tone, and acid–base balance. Sodium and potassium are particularly important in cardiac electrophysiology, and disturbances in these ions may contribute to conduction abnormalities, arrhythmogenesis, impaired myocardial contractility, and adverse outcomes in patients with acute cardiac illness (10,11). Potassium imbalance has been associated with mortality risk and arrhythmic complications among patients with acute myocardial infarction, while sodium disturbances may reflect neurohormonal activation, hemodynamic stress, renal handling changes, fluid shifts, or severity of illness (12,13). Chloride, although less frequently emphasized in myocardial infarction research, contributes to extracellular fluid regulation and acid–base homeostasis and may provide additional biochemical context when evaluated alongside sodium and potassium (10). Previous studies have reported altered sodium and potassium levels among patients with acute myocardial infarction, but findings vary across populations, and fewer local studies have directly compared these electrolyte changes between the acute and convalescent phases within the same clinical context (14,15).

The available evidence therefore supports the need for focused evaluation of how cardiac Troponin I and selected electrolytes behave across the acute and recovery phases of myocardial infarction. Although Troponin I is an established biomarker of myocardial injury, the comparative pattern of sodium, potassium, and chloride during acute illness and convalescence may help clinicians understand biochemical instability and recovery trends in myocardial infarction patients. Importantly, electrolyte abnormalities should not be interpreted as stand-alone diagnostic biomarkers for myocardial infarction without diagnostic accuracy testing or comparison with appropriate control groups; rather, their value lies in supportive monitoring and clinical interpretation when assessed together with established cardiac biomarkers and the patient's clinical status.

Based on this rationale, the present study was designed around a PICO framework in which the population comprised patients diagnosed with myocardial infarction, the index phase was the acute myocardial infarction period, the comparator was the convalescent phase following treatment and hospitalization, and the outcomes were serum Troponin I, sodium, potassium, and chloride levels. The objective of the study was to compare Troponin I and selected electrolyte levels between the acute and convalescent phases of myocardial infarction among patients managed at a tertiary care hospital in Peshawar, Pakistan. The study hypothesized that Troponin I levels would be significantly higher during the acute phase than during convalescence, while sodium and potassium levels would be lower during

the acute phase and improve during recovery, with chloride showing comparatively smaller phase-related variation.

MATERIALS AND METHODS

This paired comparative observational study was conducted among patients with myocardial infarction who were managed at the cardiology wards of Lady Reading Hospital, Peshawar, Khyber Pakhtunkhwa, Pakistan, with academic supervision from the Department of Allied Health Sciences, City University of Science and Information Technology, Peshawar. Data collection was carried out from December 2023 to June 2024. The study was designed to compare biochemical measurements obtained during the acute phase of myocardial infarction with corresponding measurements obtained during the convalescent phase after treatment and hospitalization, thereby allowing within-patient comparison of cardiac biomarker and electrolyte changes over the course of clinical recovery.

The study population consisted of male and female patients diagnosed with myocardial infarction during the study period. A total of 100 patients were selected using a convenience sampling technique from eligible patients admitted to the cardiology wards. Patients diagnosed with acute myocardial infarction and subsequently assessed during the convalescent phase after treatment and hospitalization were included. Patients with chronic kidney disease and patients undergoing dialysis were excluded because renal dysfunction and dialysis may independently alter serum electrolyte concentrations and confound interpretation of sodium, potassium, and chloride levels. The same patient population was used for acute and convalescent phase comparisons, and each participant contributed paired biochemical observations for the variables analyzed.

Recruitment was performed after identification of eligible myocardial infarction patients from the hospital setting. Demographic information and relevant clinical data were collected through structured questionnaires and review of hospital medical records. Informed consent was obtained before data collection, and participant confidentiality was maintained throughout the study process. The data collection instrument captured demographic characteristics and laboratory parameters relevant to the study objective, including cardiac Troponin I and serum electrolytes. Laboratory values for Troponin I, sodium, potassium, and chloride were recorded for the acute phase of myocardial infarction and again for the convalescent phase following treatment and hospitalization. Troponin I was recorded in ng/mL, while sodium, potassium, and chloride were recorded in mmol/L.

The primary biochemical outcome was the change in Troponin I level between the acute and convalescent phases. Secondary biochemical outcomes were changes in serum sodium, potassium, and chloride levels between the same phases. The acute phase was operationally treated as the period of active myocardial infarction during hospitalization, while the convalescent phase referred to the post-treatment recovery period after hospitalization when repeat biochemical values were available. Sodium, potassium, and chloride were evaluated as continuous biochemical variables. Lower sodium and potassium values during the acute phase were interpreted as electrolyte disturbances only in relation to accepted clinical reference concepts and paired recovery patterns; diagnostic claims were avoided because the study did not include a non-myocardial infarction control group or diagnostic accuracy analysis.

Potential bias was addressed by using paired within-patient comparisons, which reduced between-person variability because each patient served as his or her own comparator across the acute and convalescent phases. Exclusion of patients with chronic kidney disease and dialysis reduced confounding from major renal causes of electrolyte disturbance. Data were obtained from structured questionnaires and hospital records to minimize incomplete capture of demographic and laboratory information. To improve data integrity, recorded values were checked for completeness and consistency before analysis, and biochemical units were standardized before statistical testing. The study did not treat sodium, potassium,

or chloride as independent diagnostic biomarkers; instead, they were analyzed as supportive biochemical indicators whose phase-related changes were interpreted alongside Troponin I.

The sample size consisted of 100 myocardial infarction patients enrolled during the defined study period. Statistical analysis was performed using IBM SPSS version 24. Continuous variables were summarized using means, and paired comparisons between acute and convalescent values were performed using paired-sample t-tests. The paired analytical approach was selected because acute and convalescent phase measurements were obtained from the same patient population. Statistical significance was assessed using a two-sided threshold of $p < 0.05$. For publication-ready reporting, each paired biochemical comparison should include the acute-phase mean, convalescent-phase mean, paired mean difference, standard deviation, 95% confidence interval, test statistic, and p-value, allowing both statistical and clinical interpretation of the magnitude of change. Missing or incomplete laboratory observations were checked during data cleaning, and only paired observations with corresponding acute and convalescent values were used for paired analysis.

Ethical approval was obtained from the Departmental Review Committee of City University of Science and Information Technology, Peshawar, and permission for data collection was obtained from Lady Reading Hospital, Peshawar. The study was conducted with respect for participant confidentiality and responsible handling of patient information. Data were anonymized for analysis, and access to study records was restricted to the research team. The reporting structure was aligned with standard expectations for observational biomedical research, including clear identification of study design, setting, participants, variables, data sources, statistical methods, ethical approval, and reproducibility measures.

RESULTS

A total of 100 patients with myocardial infarction were included in the paired comparison of biochemical parameters between the acute and convalescent phases. The analysis focused on cardiac Troponin I and three serum electrolytes: sodium, potassium, and chloride. Paired comparisons showed marked phase-related changes in Troponin I, sodium, and potassium, while chloride demonstrated only a small and statistically non-significant difference between phases. The complete paired comparison of biochemical parameters is presented in Table 1.

Table 1. Paired Comparison of Cardiac Troponin I and Electrolyte Levels Between Acute and Convalescent Phases of Myocardial Infarction Patients

Parameter	Unit	Acute Phase Mean	Convalescent Phase Mean	Mean Difference	Direction of Change During Convalescence	Relative Change	Number of Paired Observations	p-value
Troponin I	ng/mL	10.8234	0.5836	−10.2398	Decreased	−94.61%	100	<0.001
Sodium	mmol/L	133.48	143.73	+10.25	Increased	+7.68%	100	<0.001
Potassium	mmol/L	3.6437	4.3921	+0.7484	Increased	+20.54%	100	<0.001
Chloride	mmol/L	107.183	108.338	+1.155	Slightly increased	+1.08%	100	0.447

Values are reported from the available aggregated manuscript data. Mean difference was calculated as convalescent phase mean minus acute phase mean. Relative change was calculated in relation to the acute phase mean. Because standard deviations and paired individual-level data were not available in the manuscript text, confidence intervals, t-statistics, and effect-size estimates could not be calculated without reanalysis of the raw dataset.

Troponin I demonstrated the largest biochemical change between the acute and convalescent phases. The mean Troponin I level was 10.8234 ng/mL during the acute phase and decreased to 0.5836 ng/mL during convalescence, representing an absolute mean reduction of 10.2398 ng/mL and a relative decrease of approximately 94.61%. This difference was statistically significant ($p < 0.001$), supporting the expected pattern of marked Troponin I elevation during acute myocardial injury followed by substantial decline during recovery. This finding reinforces the role of Troponin I as the principal cardiac injury

biomarker in this cohort, while the paired decline also indicates biochemical improvement during the convalescent phase.

Serum sodium levels were lower during the acute phase and increased during convalescence. The mean sodium level rose from 133.48 mmol/L in the acute phase to 143.73 mmol/L in the convalescent phase, giving an absolute mean increase of 10.25 mmol/L and a relative increase of 7.68%. The difference was statistically significant ($p < 0.001$). The acute-phase mean was below the usual lower reference threshold for serum sodium, suggesting that reduced sodium levels were present during acute myocardial infarction at the group level, whereas the convalescent mean was within the usual reference range. However, because patient-level categorical data were not available, the exact number and percentage of patients with hyponatremia could not be determined from the aggregated results.

Serum potassium also showed a significant increase from the acute to the convalescent phase. The mean potassium level was 3.6437 mmol/L during the acute phase and increased to 4.3921 mmol/L during convalescence, producing an absolute mean increase of 0.7484 mmol/L and a relative increase of 20.54%. This paired difference was statistically significant ($p < 0.001$). The acute-phase mean was close to the lower end of the usual reference range, while the convalescent mean reflected normalization toward a more stable potassium range. These findings suggest that potassium levels improved during recovery; however, the proportion of patients with hypokalemia cannot be stated without individual patient values or categorical frequency data.

Chloride showed only a small numerical increase between phases. The mean chloride level was 107.183 mmol/L during the acute phase and 108.338 mmol/L during the convalescent phase, corresponding to an absolute mean increase of 1.155 mmol/L and a relative increase of 1.08%. This difference was not statistically significant ($p = 0.447$), indicating that chloride levels remained comparatively stable between acute myocardial infarction and convalescence. In contrast to Troponin I, sodium, and potassium, chloride did not show evidence of a meaningful phase-related biochemical shift in this cohort.

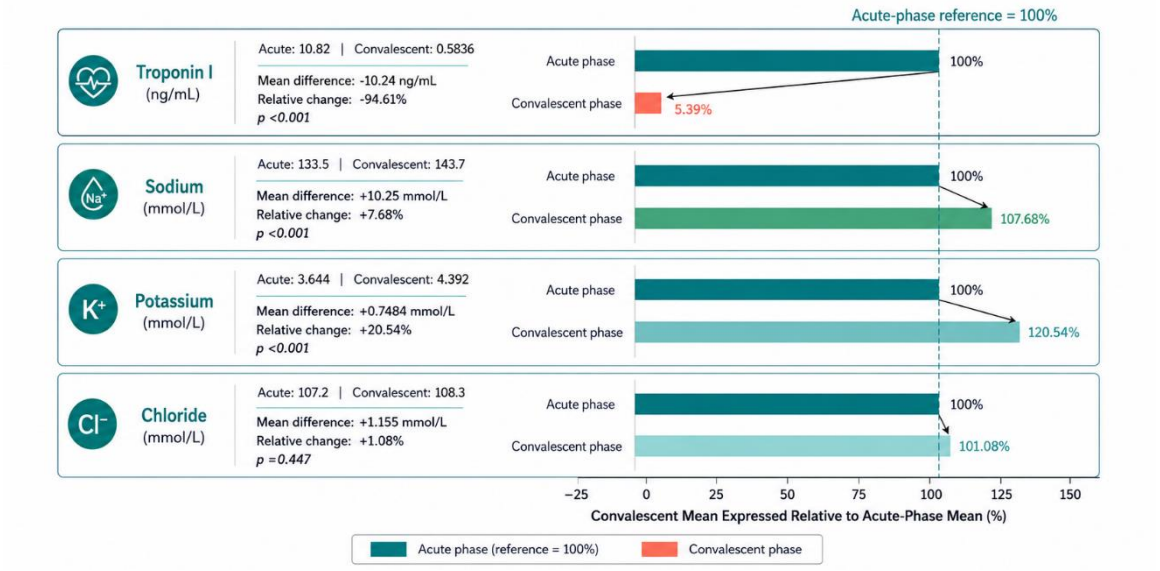

Figure 1 Phase-Related Biochemical Recovery Profile After Acute Myocardial Infarction

The panelled biochemical recovery profile shows a marked reduction in Troponin I from 10.8234 ng/mL during the acute phase to 0.5836 ng/mL during convalescence, corresponding to a mean decrease of 10.2398 ng/mL and a 94.61% relative decline ($p < 0.001$). Serum sodium increased from 133.48 mmol/L to 143.73 mmol/L, reflecting a mean rise of 10.25 mmol/L and a 7.68% relative increase ($p < 0.001$), while potassium increased from 3.6437 mmol/L to 4.3921 mmol/L, with a mean increase of 0.7484 mmol/L and a 20.54% relative improvement ($p < 0.001$). Chloride demonstrated only a minimal change from 107.183 mmol/L to 108.338 mmol/L, with a mean increase of 1.155 mmol/L and a 1.08% relative change

that was not statistically significant (p = 0.447). Overall, the figure highlights a strong recovery gradient for Troponin I, sodium, and potassium, whereas chloride remained comparatively stable across the acute and convalescent phases.

Overall, the paired biochemical analysis showed that Troponin I, sodium, and potassium differed significantly between the acute and convalescent phases of myocardial infarction, while chloride did not. Troponin I showed the greatest relative decline, decreasing by approximately 94.61%, consistent with recovery from acute myocardial injury. Sodium increased by 10.25 mmol/L and potassium increased by 0.7484 mmol/L during convalescence, suggesting improvement in acute-phase electrolyte disturbance. Chloride changed minimally and non-significantly, supporting the interpretation that chloride was not substantially altered across the two phases in this study population.

DISCUSSION

The present study compared cardiac Troponin I and selected serum electrolytes between the acute and convalescent phases of myocardial infarction in a paired cohort of 100 patients. The principal finding was a marked reduction in Troponin I from 10.8234 ng/mL during the acute phase to 0.5836 ng/mL during convalescence, corresponding to a mean decrease of 10.2398 ng/mL and an approximate 94.61% relative decline. This pattern is consistent with the established biological behavior of cardiac troponins, which rise following myocardial injury and subsequently decline as acute necrotic injury stabilizes and the patient enters recovery. Troponin I therefore remained the strongest biochemical indicator of myocardial injury in this study, and its significant phase-related decline supports its clinical value for monitoring the transition from acute infarction to convalescence (6,7,9).

The observed Troponin I pattern aligns with previous evidence showing that cardiac troponins are highly sensitive and specific markers of myocardial necrosis and are central to the biochemical diagnosis of acute myocardial infarction. Earlier studies have demonstrated that Troponin I and Troponin T rise within hours of myocardial injury and remain detectable for several days, making them clinically useful not only for confirming myocardial injury but also for interpreting the temporal evolution of infarction when serial values are available (8,9). In the present cohort, the acute-phase mean Troponin I level was substantially higher than the convalescent value, reinforcing that Troponin I reflects myocardial injury more directly than electrolyte variables. However, because the present study did not include a non-myocardial infarction control group or diagnostic accuracy analysis, the findings should be interpreted as evidence of expected paired biochemical change rather than as a new diagnostic validation study.

Serum sodium showed a significant increase from 133.48 mmol/L during the acute phase to 143.73 mmol/L during convalescence, representing an absolute mean rise of 10.25 mmol/L and a relative increase of 7.68%. This finding suggests that sodium levels were lower during acute myocardial infarction and improved during recovery. Acute-phase hyponatremia or reduced sodium levels in myocardial infarction may reflect neurohormonal activation, altered renal water handling, hemodynamic stress, fluid shifts, or severity of acute illness. Previous cardiovascular literature has indicated that sodium disturbances may accompany acute cardiac events and may be associated with worse clinical status, particularly when myocardial infarction is complicated by ventricular dysfunction or systemic stress responses (10,14). In this study, sodium improved significantly during convalescence, suggesting that sodium may serve as a useful supportive biochemical monitoring parameter during recovery; however, the study did not analyze mortality, arrhythmia, heart failure, or length of hospital stay, so sodium should not be described as an independently proven prognostic biomarker on the basis of these data alone.

Potassium also increased significantly between phases, rising from 3.6437 mmol/L during the acute phase to 4.3921 mmol/L during convalescence, with a mean increase of 0.7484 mmol/L and a relative improvement of 20.54%. This result is clinically relevant because potassium plays a central role in myocardial membrane potential, repolarization, conduction stability, and arrhythmia susceptibility. Prior

research has shown that abnormal potassium levels in acute myocardial infarction are associated with adverse cardiovascular outcomes, including arrhythmogenic risk and mortality, particularly when potassium values fall outside the physiologically optimal range (12,13). The acute-phase potassium mean in this study was close to the lower end of the usual reference range and increased toward a more stable convalescent value, indicating recovery of electrolyte balance after the acute event. Nevertheless, because individual patient-level values were not available in the manuscript, the exact frequency of hypokalemia could not be determined, and the result should be reported as a significant improvement in mean potassium level rather than as proof that all or most patients had hypokalemia.

Chloride showed only a minimal and statistically non-significant increase from 107.183 mmol/L during the acute phase to 108.338 mmol/L during convalescence, with a mean difference of 1.155 mmol/L, a relative change of 1.08%, and a p-value of 0.447. This finding indicates that chloride remained comparatively stable across the two phases and did not demonstrate the same recovery gradient observed for Troponin I, sodium, and potassium. Since chloride contributes to extracellular fluid balance and acid–base regulation, it may still provide contextual biochemical information, but the present results do not support a major phase-related chloride shift in this cohort. The non-significant chloride result is important because it suggests that electrolyte changes during acute myocardial infarction may not affect all measured ions uniformly, and that sodium and potassium may be more responsive to the acute-to-recovery transition than chloride in this dataset.

The findings of this study are broadly consistent with previous reports describing reduced sodium and potassium levels among patients with acute myocardial infarction and improvement after treatment or clinical stabilization (14,15). The results also support the biological distinction between direct cardiac injury biomarkers and supportive systemic biochemical parameters. Troponin I directly reflects myocardial necrosis, whereas sodium and potassium may reflect broader physiological stress, renal and neurohormonal responses, medication effects, fluid therapy, or recovery processes. For this reason, sodium and potassium should be interpreted as clinically relevant monitoring parameters rather than stand-alone diagnostic biomarkers for myocardial infarction. Their value may be greatest when combined with clinical assessment, electrocardiographic findings, cardiac biomarkers, renal function, treatment history, and patient outcomes.

The clinical implication of the present findings is that routine assessment of sodium and potassium alongside Troponin I may help clinicians identify biochemical instability during acute myocardial infarction and monitor normalization during recovery. The marked Troponin I decline provides expected evidence of recovery from acute myocardial injury, while the rise in sodium and potassium suggests correction of acute-phase electrolyte disturbance. In practical terms, monitoring these parameters may be particularly relevant in patients at risk of arrhythmias, hemodynamic instability, renal dysfunction, or treatment-related electrolyte shifts. However, electrolyte interpretation must be cautious because sodium and potassium can be influenced by multiple factors, including renal function, diuretic therapy, intravenous fluids, vomiting, heart failure, and neurohormonal activation.

This study has several limitations that should be considered when interpreting the findings. The convenience sampling method and single-center recruitment limit generalizability. The manuscript does not provide detailed information on the exact timing of acute and convalescent blood sampling, myocardial infarction subtype, treatment received, medication exposure, renal function values, heart failure status, or electrolyte replacement therapy, all of which may influence biomarker and electrolyte levels. The available results report means and p-values but do not include standard deviations, paired confidence intervals, t-statistics, or effect sizes, limiting assessment of statistical precision and variability. The study also lacks clinical outcome endpoints such as mortality, arrhythmias, recurrent myocardial infarction, heart failure, intensive care admission, or length of hospital stay; therefore, the term “prognostic” should be used cautiously. Future studies should include clearly defined sampling windows, standardized laboratory procedures, adjustment for confounders, patient-level electrolyte abnormality

categories, and outcome-based prognostic analyses to determine whether acute-phase sodium and potassium independently predict clinically meaningful outcomes.

CONCLUSION

In this paired comparative observational study of 100 myocardial infarction patients, Troponin I, serum sodium, and serum potassium showed significant differences between the acute and convalescent phases, whereas chloride remained comparatively stable. Troponin I decreased markedly from 10.8234 ng/mL to 0.5836 ng/mL, confirming its central role as a cardiac injury biomarker and demonstrating substantial biochemical recovery during convalescence. Sodium increased from 133.48 mmol/L to 143.73 mmol/L and potassium increased from 3.6437 mmol/L to 4.3921 mmol/L, suggesting improvement in acute-phase electrolyte disturbance during recovery. Chloride changed only minimally from 107.183 mmol/L to 108.338 mmol/L and was not statistically significant. These findings support the clinical value of monitoring sodium and potassium alongside Troponin I in myocardial infarction patients, but they should be interpreted as supportive biochemical indicators rather than independent diagnostic or prognostic biomarkers unless validated against clinical outcomes in larger prospective studies.

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