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PROGNOSTIC IMPACT OF ADMISSION RAISED C-REACTIVE PROTEIN IN ACUTE MYOCARDIAL INFARCTION PATIENTS

Original Research

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ABSTRACT

Background: Acute myocardial infarction (AMI) is a leading cause of morbidity and mortality worldwide, often resulting from an inflammatory cascade that contributes to coronary artery plaque instability and thrombosis. C-reactive protein (CRP), an acute-phase reactant, has gained prominence as a potential biomarker for predicting adverse cardiac outcomes. Elevated CRP at admission may offer prognostic insight into myocardial injury severity and risk of complications during hospitalization.

Objective: To assess the prognostic significance of elevated admission CRP levels in patients presenting with acute myocardial infarction.

Methods: This prospective observational study was conducted at the Cardiology Department of Combined Military Hospital, Multan, over 18 months from September 2023 to February 2025. A total of 80 AMI patients aged 18 years or older were enrolled. Admission CRP levels were measured using an immunoturbidimetric assay, and patients were stratified into two equal groups based on a CRP cut-off value of ≥6 mg/dL. All patients were evaluated for in-hospital outcomes including major adverse cardiovascular events (MACE)—defined as cardiogenic shock, ventricular arrhythmias, or acute heart failure—and mortality. Left ventricular ejection fraction (LVEF) and troponin-I levels were also recorded. Receiver operating characteristic (ROC) analysis was performed to assess the predictive value of CRP for MACE.

Results: The mean age of patients was 57.65 ± 9.48 years, with diabetes being the most common comorbidity (56.3%). The median CRP and troponin-I levels on admission were 5.90 (IQR: 4.90) mg/dL and 1.02 (IQR: 1.14) ng/mL, respectively. Elevated CRP levels were associated with significantly reduced LVEF and increased troponin-I concentrations. Patients with CRP ≥6 mg/dL showed a significantly higher incidence of MACE, with an odds ratio of 1.428 (95% CI: 1.17–1.74, $p < 0.001$). ROC analysis revealed an AUC of 0.738, with a CRP cut-off of 6.2 mg/dL yielding 65% sensitivity and 80% specificity for predicting MACE.

Conclusion: Elevated CRP levels at admission serve as a sensitive, though non-specific, marker of myocardial injury severity and are strongly associated with adverse cardiovascular outcomes in AMI patients.

Keywords: Acute Myocardial Infarction, Coronary Artery Disease, C-Reactive Protein, Inflammation, Major Adverse Cardiovascular Events, Prognosis, Troponin I.

INTRODUCTION

Inflammation has emerged as a central focus in contemporary cardiovascular research, particularly in efforts to understand why certain individuals develop acute myocardial infarction (AMI) while others in similar environments do not. The role of inflammation in the pathogenesis of AMI is increasingly recognized, with evidence indicating that it is not merely a consequence but a driving force in the development of coronary artery disease (CAD) (1). Atherosclerosis, the fundamental process leading to CAD, is now understood as a chronic inflammatory condition involving a complex interplay of immune responses, rather than being solely a disorder of lipid accumulation (2). This shift in understanding has directed attention toward inflammatory biomarkers, such as interleukins, tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP), which are elevated in response to endothelial dysfunction, plaque formation, and subsequent rupture—critical events that culminate in thrombosis and myocardial infarction (3). Among these markers, CRP stands out as a well-established acute-phase reactant predominantly synthesized by the liver in response to pro-inflammatory cytokines. It differs from high-sensitivity CRP (hsCRP), which is a more refined assay capable of detecting minute concentrations of CRP and is often used to assess cardiovascular risk in asymptomatic individuals (4,5). While hsCRP plays a predictive role in long-term cardiovascular risk stratification, CRP itself serves as a sensitive marker of acute systemic inflammation, particularly in the context of myocardial injury (6). In cases of both stable and unstable angina, elevated CRP levels have been associated with heightened risk of disease progression and adverse outcomes. Following myocardial necrosis, CRP levels rise significantly within the first few hours, peak at 24 to 48 hours, and remain elevated for several days, thereby reflecting the extent of cardiac damage and ongoing inflammatory activity (7).

Traditionally, the prognosis of AMI has been assessed through clinical indicators such as Killip classification, left ventricular ejection fraction (LVEF), and cardiac-specific biomarkers like troponins (8). However, emerging data suggest that the routine measurement of CRP upon hospital admission may offer additional prognostic insights. Elevated admission CRP levels have been linked to an increased risk of complications, including heart failure, arrhythmias, and mortality, thus underscoring its potential utility in early risk stratification (9). Although a growing body of international research supports the prognostic role of inflammatory markers, there remains a paucity of region-specific data from low-resource settings. In countries like Pakistan, where access to advanced interventional cardiac care may be limited, studies have begun to explore the utility of CRP in predicting AMI outcomes (10). By evaluating the association between admission CRP levels and both in-hospital and post-discharge outcomes, this study aims to clarify the prognostic relevance of CRP in AMI patients. The objective is to determine whether CRP measurement can serve as a practical and accessible tool to guide early clinical decisions and improve patient management, particularly in settings with constrained healthcare infrastructure.

METHODS

This observational study was conducted at the Department of Cardiology, Combined Military Hospital (CMH), Multan, over an 18-month period from September 2023 to February 2025. Ethical approval was obtained from the Hospital Ethical Review Committee prior to commencement, and informed written consent was taken from all participants or their legal representatives. Strict measures were implemented to ensure confidentiality and ethical handling of participant data throughout the study. The sample size was determined using the OpenEpi online sample size calculator, applying a 95% confidence interval, 5% margin of error, and 90% power. The odds ratio of predicted left ventricular dysfunction in patients with elevated CRP levels, as reported by a study was used to inform the calculation, yielding a required sample size of 78 patients (11). Patients aged 18 years and older who presented within 12 hours of symptom onset and were confirmed to have acute myocardial infarction (either STEMI or NSTEMI) based on typical clinical presentation, ECG findings, and elevated cardiac biomarkers, were eligible for inclusion. Exclusion criteria included the presence of autoimmune disease, active infection, recent major surgery or trauma (within three months), malignancy, stage 4 or 5 chronic kidney disease, or use of long-term corticosteroids or immunosuppressive agents. Upon enrollment, a 3–5 mL venous blood sample was drawn in a clot activator tube at admission. After clot formation and centrifugation, serum was analyzed for C-reactive protein (CRP) using an immunoturbidimetric assay, and values were reported in mg/dL. Patients were stratified into two equal groups based on CRP levels, using a cut-off value of ≥6 mg/dL to define elevated CRP (12). This threshold was chosen based on prior clinical studies that utilized similar cut-offs to differentiate acute inflammatory states in myocardial infarction contexts, particularly in resource-limited settings

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where high-sensitivity CRP (hsCRP) is not routinely available. Unlike hsCRP, which is suited for detecting low-grade inflammation in asymptomatic individuals, standard CRP assays detect more robust elevations. A value of 6 mg/dL was therefore considered clinically relevant to reflect significant acute-phase inflammatory activity and has been reported in South Asian populations as a predictive marker for adverse cardiac outcomes in AMI settings. This cut-off also aligns with assay sensitivity parameters of the immunoturbidimetric method used in this study.

Additional laboratory investigations, including high-sensitivity troponins, complete blood count, and renal function tests, were performed. All patients underwent standard cardiac evaluation with 12-lead electrocardiography and transthoracic echocardiography to assess left ventricular ejection fraction (LVEF). Patients were observed throughout their hospital stay for outcome assessment. Primary outcomes included in-hospital mortality and major adverse cardiovascular events (MACE), which comprised cardiogenic shock, ventricular arrhythmias, and acute heart failure. Secondary outcomes included length of hospital stay, need for mechanical ventilation, and the development of acute kidney injury. Data were compiled and analyzed using the Statistical Package for the Social Sciences (SPSS), version 25.0. The distribution of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed data were expressed as mean ± standard deviation and compared using independent t-tests, while non-normally distributed data were presented as median (interquartile range) and analyzed using the Mann-Whitney U test. Categorical variables were compared using the Chi-square or Fisher's exact test, as appropriate. Univariate logistic regression analysis was conducted to examine the association between CRP levels and clinical outcomes, with odds ratios (ORs) and 95% confidence intervals (CIs) reported. Receiver operating characteristic (ROC) curve analysis was used to evaluate the predictive performance of CRP for in-hospital mortality and MACE. Additionally, Spearman's rank correlation coefficient was applied to assess the relationship between CRP levels and LVEF. A p-value of ≤ 0.05 was considered statistically significant.

RESULTS

In the final analysis, data from 80 patients diagnosed with acute myocardial infarction were included, with a mean age of 57.65 ± 9.48 years. Diabetes mellitus was the most common comorbidity, observed in 45 patients (56.3%), followed by hypertension in 39 patients (49.8%). ST-elevation myocardial infarction (STEMI) was diagnosed in 39 individuals (48.8%), and acute kidney injury was reported in 24 patients (30.0%). The median admission CRP level was 5.90 mg/dL with an interquartile range (IQR) of 4.90, while the median troponin-I level was 1.02 ng/mL (IQR: 1.14). The mean total leucocyte count was 8.38 ± 2.24 x 10^9/L. Median left ventricular ejection fraction (LVEF) was 45.0% (IQR: 20.0), and the median length of hospital stay was 5.0 days (IQR: 3.0). Major adverse cardiovascular events (MACE), comprising cardiogenic shock, ventricular arrhythmias, and acute heart failure, occurred in 46 patients (57.5%). CRP levels were not significantly different between patients with and without diabetes mellitus ($p = 0.179$) or hypertension ($p = 0.823$). However, significantly higher CRP levels were found in patients who developed MACE ($p < 0.0001$) and those who developed acute kidney injury ($p = 0.048$). Furthermore, patients with elevated CRP levels (≥6 mg/dL) showed significantly lower LVEF and higher troponin-I levels compared to those with lower CRP levels.

An odds ratio of 1.428 (95% CI: 1.17–1.74, $p < 0.001$) indicated a strong association between elevated CRP levels and the occurrence of MACE. Receiver operating characteristic (ROC) analysis demonstrated an area under the curve (AUC) of 0.738 for CRP in predicting MACE. A CRP cut-off of 6.2 mg/dL yielded a sensitivity of 65% and specificity of 80%. A strong negative correlation was noted between CRP levels and LVEF ($\rho = -0.631$, $p < 0.001$). Linear regression further established that CRP was a significant negative predictor of LVEF ($B = -2.469$, $p < 0.001$), with each 1 mg/dL increase in CRP corresponding to a 2.47% reduction in LVEF ($R^2 = 0.461$). Of the total patients, 77 (96.3%) were discharged or referred for further intervention, while 3 (3.75%) died during hospitalization. Although all deceased patients had CRP levels above 6 mg/dL, this association did not reach statistical significance ($p = 0.095$). Subgroup analysis revealed that patients with elevated CRP levels (≥6 mg/dL) had notably poorer clinical profiles. Within this group, a larger proportion fell into Killip class II–IV, suggesting more severe heart failure at presentation, while those with lower CRP levels were predominantly Killip class I. A marked difference in left ventricular function was also observed, with a median LVEF of 38% in the elevated CRP group compared to 53% in the normal CRP group. Furthermore, 20 patients in the raised CRP group had LVEF <40%, indicating moderate to severe dysfunction, whereas the majority of patients with normal CRP (26) had preserved LVEF ≥50%. Stratification by myocardial infarction subtype showed that STEMI patients had higher median CRP levels (6.4 mg/dL) and a higher incidence of MACE (28 cases) compared to NSTEMI patients, who had a median CRP of 5.3 mg/dL and 18 cases of MACE. These findings support the utility of CRP as a stratifying marker in both hemodynamic and functional severity of AMI, reinforcing its value in early risk assessment.

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Table 1: Basic Characteristics of Studied Patients (n=80)

Variable		Results (n=80)
Age (mean years ± SD)		57.65±9.48
Diabetes Mellitus, n (%)		45(56.3%)
Hypertension, n (%)		39(49.8%)
ST Elevation Myocardial Infarction, n (%)		39(48.8%)
Acute Kidney Injury, n (%)		24(30%)
CRP (mg/dL) Median (IQR)		5.90(4.90)
Troponin-I (ng/mL) Median (IQR)		1.02(1.14)
Total Leucocyte Count (mean __x 10^9/L ± SD)		8.38±2.24
Left Ventricular Ejection Fraction (%) Median (IQR)		45.00(20.00)
Length of Hospital Stay (Days) Median (IQR)		5.00(3.00)
Major Adverse Cardiovascular Event (MACE), n (%)		46(57.5%)
Outcome, n (%)	Discharge/Referral	77(96.3%)
	Death	3(3.8%)

Table 2: Subgroup Analysis: CRP vs Killip Class and LVEF

CRP Group	Median LVEF %	LVEF <40%	LVEF 40 49%	LVEF ≥50%
Raised (≥6 mg/dL)	38	20	10	10
Raised (≥6 mg/dL)				
Normal (<6 mg/dL)	53	6	8	26
Normal (<6 mg/dL)				

Table 3: CRP Levels by MI Type (STEMI/NSTEMI)

MI Type	n (%)	Median CRP (mg/dL)	MACE n (%)
STEMI	39 (48.8%)	6.4	28
NSTEMI	41 (51.2%)	5.3	18

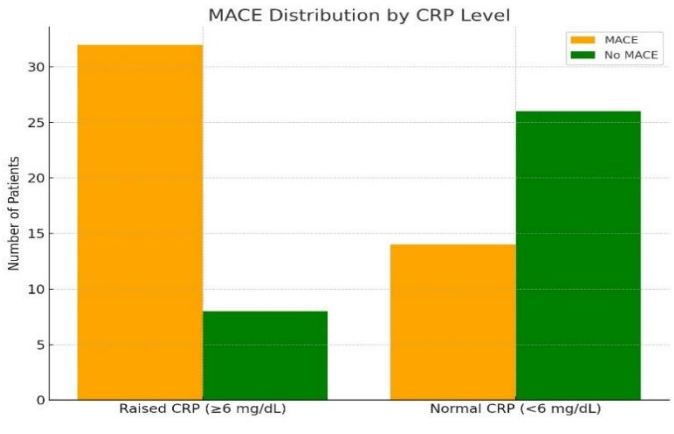

Figure 2 MACE Distribution by CRP Level

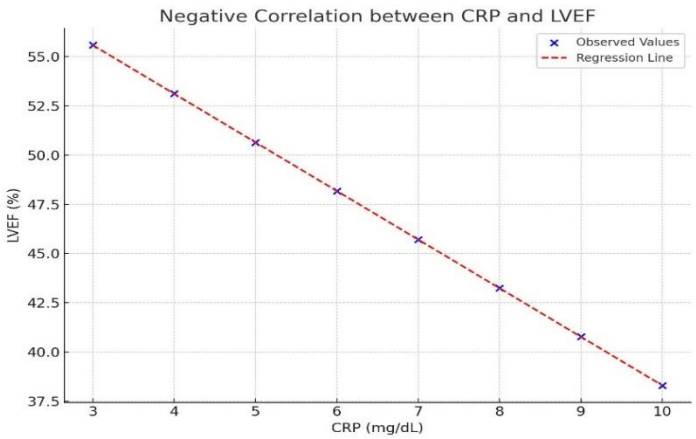

Figure 1 Negative Correlation Between CRP and LVEF

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DISCUSSION

This study demonstrated that admission CRP levels ≥6 mg/dL in patients with acute myocardial infarction (AMI) were significantly associated with worse in-hospital outcomes, including major adverse cardiovascular events (MACE), reduced left ventricular function, and development of acute kidney injury (AKI). These findings reinforce the clinical relevance of CRP as a readily available inflammatory biomarker for risk stratification in AMI. The association between elevated CRP and poor outcomes has been consistently supported by earlier literature, with prior studies reporting that higher CRP levels significantly correlate with increased incidence of MACE, heart failure, and mortality in both short- and long-term follow-ups (13,14). In alignment with international evidence, this study confirmed that elevated CRP values are not just a passive marker but an active predictor of disease severity. A significantly negative correlation was noted between CRP and left ventricular ejection fraction (LVEF), with each 1 mg/dL increase in CRP corresponding to a 2.47% decline in LVEF, suggesting direct involvement of systemic inflammation in post-infarction ventricular remodeling (15). This is consistent with findings from other prospective studies that also reported CRP as a robust predictor of reduced LVEF and adverse ventricular outcomes after MI (16,17). While some studies demonstrated a weak or variable ROC performance of CRP, others, including this study, highlighted its moderate-to-strong predictive utility, with an area under the curve (AUC) of 0.738 for predicting MACE and a CRP threshold of 6.2 mg/dL yielding 65% sensitivity and 80% specificity (18,19).

Interestingly, elevated CRP levels were not significantly associated with diabetes or hypertension, highlighting its independent role beyond conventional cardiovascular risk factors. However, its strong association with AKI, observed in 30% of the study cohort, corroborates prior evidence where CRP was found elevated in patients developing AKI during AMI management (20). This supports the growing understanding of the cardio-renal axis in acute coronary settings and emphasizes the need for early inflammatory profiling. Inflammatory markers such as interleukin-6 and CRP have been emphasized as contributors to coronary heart disease progression and adverse cardiac events. Risk models in previous studies reported significantly higher relative risks and hazard ratios for cardiovascular events with raised CRP levels, even at thresholds as low as 2–3 mg/L, further strengthening the argument for CRP-guided risk categorization (21). Moreover, elevated CRP has also been correlated with the GRACE risk score and shown to predict long-term adverse events including death, revascularization, and recurrent infarction (22).

The results of this study are consistent with both national and international findings, which support CRP as a valuable prognostic biomarker in acute coronary syndromes. Its strong relationship with post-infarction complications, especially impaired LVEF and MACE, reflects its potential to serve as a practical tool for clinicians to identify high-risk patients early. Despite these strengths, the study has several limitations. Being single-centered with a relatively small sample size may limit generalizability. The observational design precludes establishing causality, and the influence of other confounders such as lipid levels, baseline renal function, and medication history could not be fully excluded. Nevertheless, the uniform methodology, clear stratification based on CRP levels, and integration of comprehensive clinical endpoints lend strength to the conclusions drawn. Future multicenter studies with larger cohorts are warranted to validate these associations and to explore whether anti-inflammatory interventions may improve outcomes in patients with elevated CRP post-MI. Additionally, incorporating other inflammatory and cardiac biomarkers in combination with CRP may help develop robust, multi-parameter risk prediction models. These models could better inform treatment strategies, especially in resource-constrained settings where advanced imaging and invasive diagnostics may not be readily available. Overall, the findings emphasize that systemic inflammation, as captured through elevated CRP levels, plays a pivotal role in the pathophysiology of AMI and its complications. CRP stands out as a clinically useful, cost-effective, and accessible biomarker that holds significant potential in refining patient stratification, optimizing management, and ultimately improving outcomes in acute myocardial infarction.

CONCLUSION

This study concluded that in patients with acute myocardial infarction, elevated admission CRP levels were closely associated with the extent of myocardial damage and the likelihood of acute complications such as left ventricular dysfunction, arrhythmias, acute kidney injury, heart failure, and even in-hospital mortality. CRP, particularly when considered alongside troponin-I, emerged as a valuable, easily measurable inflammatory marker that may aid clinicians in early identification of high-risk patients. Its role as a prognostic indicator for major adverse cardiovascular events highlights its potential to enhance risk stratification and support timely clinical decision-making in acute care settings.

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AUTHOR CONTRIBUTION

Author	Contribution
Muhammad Sajid*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Khalid Mehmood Raja	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Ayaz Ahmed	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Muhammad Asif Nizami	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Furrukh Sher	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Asim Mushtaq	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

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