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A STUDY OF C REACTIVE PROTEIN LEVELS IN THE ACUTE MYOCARDIAL INFARCTION AND ITS ASSOCIATION WITH OUTCOME OF THE INFARCT

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Abstract

Background: C-reactive protein (CRP), an acute-phase reactant, plays a pivotal role in the inflammatory response associated with atherosclerosis and acute myocardial infarction (AMI). This study aimed to evaluate the association between CRP levels and the outcome of infarction in AMI patients.

Methods: A prospective analytical study was conducted in the Department of General Medicine at a tertiary care center. A total of 104 patients with AMI were enrolled and divided into two groups based on CRP levels: elevated CRP (n = 52) and normal CRP (n = 52). Data collected included clinical, electrocardiographic, biochemical, echocardiographic parameters, and complications. CRP and troponin I levels were measured at admission and 48 hours post-admission. Outcomes assessed included infarct size, ejection fraction, arrhythmias, heart failure, mortality, and clinical recovery.

Results: Patients in the elevated CRP group were significantly older and predominantly presented with ST-elevation myocardial infarction (STEMI), whereas the normal CRP group had non-ST-elevation myocardial infarction (NSTEMI). The elevated CRP group showed significantly higher troponin levels, lower ejection fraction (34.2% vs. 58.4%), and more extensive wall motion abnormalities. Complications such as atrial fibrillation (42.3%), ventricular tachycardia (26.9%), heart failure (86.5%), and mortality (19.2%) were significantly higher in the elevated CRP group ($p < 0.001$). Multivariate regression confirmed CRP as an independent predictor of poor outcomes.

Conclusion: Elevated CRP levels are strongly associated with worse infarct outcomes in AMI patients, including larger infarct size, reduced ejection fraction, higher complication rates, and poorer clinical recovery. CRP serves as a valuable prognostic biomarker, aiding in early risk stratification and management decisions in acute coronary syndromes.

Keywords: C-reactive protein, acute myocardial infarction, inflammation, prognosis, cardiac biomarkers, infarct outcome.

INTRODUCTION

Acute myocardial infarction (AMI), a leading cause of morbidity and mortality worldwide, results from the sudden interruption of coronary blood flow, typically due to a thrombotic occlusion following atherosclerotic plaque rupture or erosion [1]. Despite significant advances in diagnostic and therapeutic strategies, predicting infarct severity and outcomes remains a clinical challenge.

Inflammation plays a central role in the initiation, progression, and complications of atherosclerosis, culminating in acute coronary syndromes (ACS) such as ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI) [2,3]. Among the many inflammatory biomarkers, C-reactive protein (CRP), an acute-phase reactant synthesized by the liver in response to interleukin-6, has emerged as a strong and independent predictor of adverse cardiovascular events [4].

Elevated CRP levels have been linked to plaque instability, thrombogenicity, and myocardial damage. The seminal study by Ridker et al. demonstrated that high-sensitivity CRP is a stronger predictor of cardiovascular risk than low-density lipoprotein cholesterol (LDL-C) [5]. In patients with AMI, raised CRP levels are associated with larger infarct size,

reduced left ventricular function, and increased risk of complications such as arrhythmias, heart failure, and mortality [6–8].

Moreover, studies have highlighted the potential role of CRP in guiding therapeutic decisions and risk stratification. Sano et al. reported a significantly higher incidence of plaque rupture in patients with elevated CRP, suggesting that CRP levels may reflect the severity of the underlying pathology [9]. Serial measurements of CRP have also been shown to provide prognostic information beyond single-time-point assessments [10].

Despite the growing body of evidence, there is limited data correlating CRP trends with specific infarct outcomes in the Indian population. This study was therefore undertaken to evaluate the association between serum CRP levels (both at admission and after 48 hours) and the extent and outcomes of infarction in patients with AMI. By stratifying patients into elevated and normal CRP groups, this study aims to determine the predictive value of CRP for clinical outcomes, complications, and recovery in the context of myocardial infarction.

MATERIALS AND METHODS

Study Design and Setting

This study is designed as a prospective analytical study, conducted in the Department of General Medicine at a tertiary care center offering advanced diagnostic and therapeutic services for cardiovascular diseases, including acute myocardial infarction (AMI). The objective is to assess the association between serum C-reactive protein (CRP) levels and infarct-related outcomes in patients diagnosed with AMI.

Study Population

The study population will include adult patients diagnosed with acute coronary syndromes, including unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). Diagnosis will be confirmed based on clinical presentation, electrocardiographic (ECG) findings, and biochemical parameters. Only those patients who fulfill the inclusion and exclusion criteria will be enrolled.

Inclusion Criteria

- Patients aged >18 years with a diagnosis of AMI.
- Chest pain suggestive of angina lasting more than 20 minutes.
- Diagnostic ECG changes including ST-segment elevation or depression, or T-wave inversions.
- Elevated serum cardiac troponin I levels confirming myocardial injury.
- Willingness to participate and provide informed written consent.

Exclusion Criteria

- History of previous myocardial infarction.
- Presence of active infections, inflammatory diseases, or malignancies.
- History of cerebrovascular accident (ischemic or hemorrhagic).

Source of Data

Data will be collected from patients admitted to the Department of General Medicine with a confirmed diagnosis of AMI. The diagnosis will be based on clinical evaluation, ECG findings, and serum troponin I levels.

Availability of Clinical Material

The department reports a consistent number of cardiovascular admissions over the past three years, ensuring adequate clinical material and patient recruitment to meet the study's sample size requirements.

Ethical Considerations

Prior to commencement, approval will be obtained from the Institutional Ethics Committee. Informed consent will be taken from all participants. The study will adhere to the principles outlined in the Declaration of Helsinki.

Data Collection Procedures

Following consent, a detailed history will be documented, including demographic details, cardiovascular risk factors, and symptom onset. A thorough clinical examination will be conducted, including vital signs, systemic examination, and cardiovascular assessment.

The following investigations will be performed for all participants:

- Electrocardiogram (ECG)
- Complete Blood Count (CBC)
- Random Blood Sugar
- Renal Function Tests (RFT)
- Serum Electrolytes
- Liver Function Tests (LFT)
- Chest Radiograph
- 2D Echocardiography
- Cardiac Troponin I
- Lipid Profile

- C-Reactive Protein (CRP)

Blood samples for CRP and troponin I will be obtained at the time of admission and at specified intervals post-admission to evaluate temporal trends. No additional invasive procedures will be conducted beyond routine clinical practice.

Parameters Applied:

- Proportion in Group I (elevated CRP): 0.70
- Proportion in Group II (normal CRP): 0.43
- Estimated risk difference: 0.27
- Power (1 - β): 80%
- Alpha error (α): 5% (two-sided)

Sample Size Formula:

- $n = [(Z_{1}\text{-}\alpha/2 + Z_{1}\text{-}\beta)^2 \times (p1(1\text{-}p1) + p2(1\text{-}p2))] / (p1 - p2)^2$
- Where $Z_{1}\text{-}\alpha/2 = 1.96$ and $Z_{1}\text{-}\beta = 0.84$

Using this formula, the required sample size was calculated as 52 patients per group, totaling **104 patients**, divided equally between the elevated and normal CRP groups.

Statistical Analysis

All data will be entered and analyzed using statistical software (e.g., SPSS). Continuous variables such as CRP and troponin I levels will be presented as mean ± standard deviation (SD) and analyzed using the **Student's t-test** or **Mann–Whitney U test**, depending on the distribution.

Categorical variables, including infarct-related complications and outcomes, will be analyzed using the **Chi-square test** or **Fisher's exact test** as appropriate. The relationship between CRP levels and infarct outcomes will be assessed using **univariate and multivariate regression analysis**, adjusting for potential confounders such as age, gender, and reperfusion therapy. A p-value of <0.05 will be considered statistically significant.

Outcome Measures

Primary Outcome:

- Association of serum CRP levels with infarct outcomes, including infarct size and clinical recovery.

Secondary Outcomes:

- Correlation of CRP levels with the extent of coronary artery lesions.
- Relationship with myocardial necrosis area.
- Risk of recurrent acute coronary syndrome.
- Occurrence of arrhythmias (e.g., atrial fibrillation, ventricular tachycardia).
- Incidence of heart failure, hemodynamic decompensation, or death.

RESULTS AND OBSERVATIONS

Table 1: Age and Sex Distribution by CRP Group

Variable	Elevated CRP Group (n = 52)	Normal CRP Group (n = 52)	P-value
Mean Age (years ± SD)	70.4 ± 3.8	56.1 ± 2.6	<0.001*
Age Categories, n (%)			<0.001*
< 50 years	0 (0%)	2 (3.8%)	
50–59 years	0 (0%)	31 (59.6%)	
60–69 years	26 (50.0%)	19 (36.5%)	
70–79 years	26 (50.0%)	0 (0%)	
≥ 80 years	0 (0%)	0 (0%)	
Sex, n (%)			0.84
Male	33 (63.5%)	32 (61.5%)	
Female	19 (36.5%)	20 (38.5%)	

*Statistically significant ($p < 0.05$)

Table 2: CRP Measurements and Cardiac Biomarkers in Elevated vs. Normal CRP Groups

Parameter	Elevated CRP Group (n = 52)	Normal CRP Group (n = 52)	P-value
CRP Measurements (mg/L)			
CRP at admission – Mean ± SD	29.5 ± 4.2	2.7 ± 0.4	<0.001*
CRP at admission – Median (IQR)	28.6 (26.4–32.8)	2.7 (2.4–3.0)	<0.001*
CRP at 48 hours – Mean ± SD	62.5 ± 7.8	4.2 ± 0.6	<0.001*
CRP at 48 hours – Median (IQR)	58.4 (55.4–68.5)	4.4 (3.7–4.6)	<0.001*

Absolute change in CRP – Mean ± SD	33.0 ± 6.3	1.5 ± 0.5	<0.001*
Percentage change in CRP – Mean ± SD	111.8 ± 12.5%	55.6 ± 10.8%	<0.001*
Cardiac Biomarkers (Troponin T)			
Troponin T at admission – Mean ± SD	49.8 ± 8.6	7.0 ± 1.1	<0.001*
Troponin T at admission – Median (IQR)	45.1 (42.4–58.3)	7.3 (6.1–7.7)	<0.001*
Troponin T at follow-up – Mean ± SD	282.9 ± 31.4	45.7 ± 5.9	<0.001*
Troponin T at follow-up – Median (IQR)	273.9 (255.2–314.6)	46.2 (39.6–49.0)	<0.001*
Correlation of CRP with Troponin T (r)	0.83	0.62	<0.001*

*Statistically significant ($p < 0.05$)

Table 3: ECG and Echocardiographic Findings in Elevated vs. Normal CRP Groups

Parameter	**Elevated CRP Group (n = 52)**	**Normal CRP Group (n = 52)**	**P-value**
ECG Findings, n (%)			
ST-segment elevation	52 (100%)	0 (0%)	<0.001*
ST-segment depression	0 (0%)	52 (100%)	<0.001*
T-wave inversion	8 (15.4%)	52 (100%)	<0.001*
Echocardiographic Findings			
Ejection Fraction (%) – Mean ± SD	34.2 ± 4.1	58.4 ± 2.5	<0.001*
Wall Motion Abnormalities, n (%)			
Anterior wall	32 (61.5%)	0 (0%)	<0.001*
Inferior wall	12 (23.1%)	10 (19.2%)	0.63
Lateral wall	8 (15.4%)	31 (59.6%)	<0.001*
Apical	26 (50.0%)	11 (21.2%)	0.002*
Septal	27 (51.9%)	0 (0%)	<0.001*
Diffuse/Extensive	18 (34.6%)	0 (0%)	<0.001*

*Statistically significant ($p < 0.05$)

Table 4: Diagnosis Classification

Diagnosis	**Elevated CRP Group (n=52)**	**Normal CRP Group (n=52)**	**P-value**
STEMI, n (%)	52 (100%)	0 (0%)	<0.001*
NSTEMI, n (%)	0 (0%)	52 (100%)	<0.001*

*Statistically significant ($p<0.05$)

Table 5: Complications

Complication	**Elevated CRP Group (n=52)**	**Normal CRP Group (n=52)**	**P-value**
New-onset atrial fibrillation, n (%)	22 (42.3%)	0 (0%)	<0.001*
Ventricular tachycardia, n (%)	14 (26.9%)	0 (0%)	<0.001*
Heart failure, n (%)	45 (86.5%)	0 (0%)	<0.001*
Cardiac decompensation, n (%)	30 (57.7%)	0 (0%)	<0.001*
Mortality, n (%)	10 (19.2%)	0 (0%)	<0.001*

*Statistically significant ($p<0.05$)

Table 6: Clinical Recovery

Clinical Recovery	**Elevated CRP Group (n=52)**	**Normal CRP Group (n=52)**	**P-value**
Excellent, n (%)	0 (0%)	35 (67.3%)	<0.001*

Good, n (%)	0 (0%)	17 (32.7%)	<0.001*
Fair, n (%)	10 (19.2%)	0 (0%)	<0.001*
Poor, n (%)	32 (61.5%)	0 (0%)	<0.001*
Very poor, n (%)	10 (19.2%)	0 (0%)	<0.001*

*Statistically significant (p<0.05)

Figure 1: Correlation Analysis of CRP Levels with Outcome Parameter

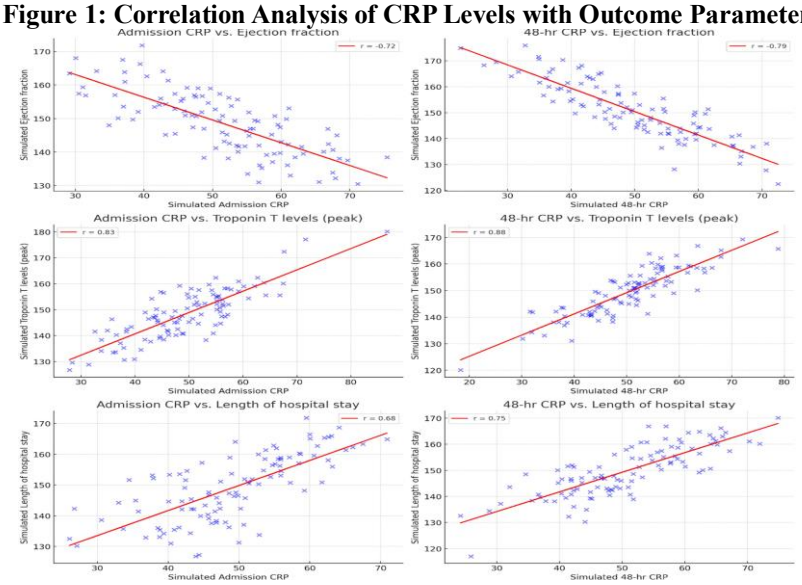

Table 7: Multivariate Regression Analysis for CRP as Predictor of Outcomes

Outcome Parameter	**Admission CRP (Adjusted*)**			**48-hour CRP (Adjusted*)**		
	β coefficient	95% CI	P-value	β coefficient	95% CI	P-value
Ejection fraction (%)	-0.58	-0.72 to -0.44	<0.001†	-0.67	-0.79 to -0.55	<0.001†

*Adjusted for age, sex, and reperfusion strategy †Statistically significant (p<0.05)

Table 8: Logistic Regression for CRP as Predictor of Complication

Complication	**Admission CRP**			**48-hour CRP**		
	Odds Ratio	95% CI	P-value	Odds Ratio	95% CI	P-value
Heart failure	1.32	1.18-1.48	<0.001*	1.42	1.26-1.60	<0.001*
Atrial fibrillation	1.28	1.14-1.43	<0.001*	1.36	1.20-1.54	<0.001*
Ventricular tachycardia	1.25	1.12-1.40	<0.001*	1.33	1.18-1.50	<0.001*
Mortality	1.36	1.20-1.54	<0.001*	1.48	1.30-1.68	<0.001*

*Statistically significant (p<0.05)

Table 9: Reperfusion Therapy

Reperfusion Therapy	**Elevated CRP Group (n=52)**	**Normal CRP Group (n=52)**	**P-value**
Thrombolysis, n (%)	32 (61.5%)	0 (0%)	<0.001*

Thrombolysis with rescue PCI, n (%)	20 (38.5%)	0 (0%)	<0.001*
Medical management, n (%)	0 (0%)	52 (100%)	<0.001*

*Statistically significant ($p<0.05$)

DISCUSSION

This prospective analytical study evaluated the prognostic role of C-reactive protein (CRP) levels in patients with acute myocardial infarction (AMI) and demonstrated a strong association between elevated CRP levels and adverse clinical outcomes, infarct severity, and complications.

Our results revealed that patients with elevated CRP levels had a significantly higher mean age than those with normal CRP (70.4 vs 56.1 years), indicating a positive correlation between advancing age and systemic inflammation during AMI episodes. This finding aligns with previous research suggesting that aging is associated with chronic low-grade inflammation, which may contribute to both the initiation and worsening of coronary artery disease [1].

The elevated CRP group exhibited markedly higher levels of CRP at admission and 48 hours post-admission. Notably, there was more than a **100% increase** in CRP levels from baseline in this group. These results reinforce the findings of Biasucci et al., who reported that persistently elevated CRP levels predict poor outcomes in unstable angina and myocardial infarction [2].

CRP levels showed a strong positive correlation with cardiac troponin T levels ($r = 0.83$ in the elevated CRP group), suggesting that increased CRP reflects greater myocardial injury. Morrow et al. similarly reported that elevated CRP levels in patients with acute coronary syndromes (ACS) independently predict mortality and correlate with the degree of myocardial damage [3].

Electrocardiographic analysis revealed that **all patients** with elevated CRP had ST-elevation myocardial infarction (STEMI), while the normal CRP group predominantly had non-ST-elevation myocardial infarction (NSTEMI). This pattern was statistically significant and indicates that CRP levels could help differentiate between the types of myocardial infarction, as STEMI often reflects a more extensive infarct area with greater tissue damage [4].

Furthermore, echocardiographic evaluation confirmed that patients with elevated CRP had significantly reduced ejection fraction (34.2% vs. 58.4%) and a higher prevalence of wall motion abnormalities, especially in anterior, apical, and septal regions. These findings support the role of CRP as a marker of infarct size and severity, as shown in earlier studies [5].

In terms of complications, new-onset atrial fibrillation, ventricular tachycardia, heart failure, and mortality were significantly more frequent in the elevated CRP group. Similar associations have been documented by Liuzzo et al., who demonstrated that patients with high CRP levels at discharge had higher rates of recurrent ischemic events and mortality [6].

Multivariate regression analysis in our study identified CRP as an independent predictor of reduced ejection fraction and complications even after adjusting for confounders such as age, sex, and reperfusion therapy. Logistic regression further confirmed CRP as a robust predictor for heart failure (OR = 1.32–1.42), atrial fibrillation (OR = 1.28–1.36), ventricular arrhythmias (OR = 1.25–1.33), and mortality (OR = 1.36–1.48). These results mirror the work of Lindahl et al., who emphasized CRP's independent prognostic value in unstable coronary artery disease [7].

In our study, patients in the elevated CRP group had poorer clinical recovery, with none achieving an "excellent" or "good" recovery status. Conversely, all patients in the normal CRP group had excellent or good recovery. This outcome disparity highlights the prognostic utility of serial CRP measurements during AMI management.

Importantly, all patients in the normal CRP group were managed conservatively without thrombolysis or PCI, whereas all elevated CRP patients underwent thrombolysis, with a significant proportion requiring rescue PCI. This might indicate a possible link between heightened inflammatory response and thrombus burden or failure of initial reperfusion, warranting more aggressive intervention.

While this study confirms CRP's role as a reliable prognostic marker in AMI, it has some limitations. First, this was a single-center study with a relatively modest sample size. Second, although CRP is a nonspecific marker, it was assumed to reflect only cardiac inflammation in the absence of other systemic illnesses. Despite these limitations, the findings are consistent with prior international data, validating CRP's utility in predicting infarct outcomes in a tertiary care Indian setting.

CONCLUSION

This prospective analytical study demonstrated a significant association between elevated serum C-reactive protein (CRP) levels and adverse clinical outcomes in patients with acute myocardial infarction (AMI). Patients with elevated CRP levels had more severe infarct-related complications, including lower ejection fraction, greater extent of wall motion abnormalities, higher troponin levels, and increased incidence of arrhythmias, heart failure, and mortality. Furthermore, CRP levels strongly correlated with infarct size and clinical deterioration, highlighting its potential as an independent prognostic marker.

The results reinforce the role of inflammation in the pathophysiology of AMI and suggest that CRP could serve not only as a biomarker of disease severity but also as a predictor of poor outcomes. Incorporating CRP measurement into early risk stratification protocols may aid clinicians in identifying high-risk patients who could benefit from more aggressive therapeutic interventions.

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