Original Article

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Correlation of C-reactive Protein with Cardiac Enzymes and Left Ventricular Function in Patients with Acute Myocardial Infarction: A Cross-sectional Study

Internal Medicine Section

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ABSTRACT

Introduction: Coronary Artery Disease (CAD) is a leading cause of morbidity and mortality worldwide, arising from atherosclerotic narrowing or acute occlusion of the coronary arteries. Acute Myocardial Infarction (AMI), a severe manifestation of CAD, is influenced by risk factors such as hypertension, diabetes, smoking, and hyperlipidemia. C-Reactive Protein (CRP), an inflammatory marker produced by the liver in response to Interleukin-6, is elevated in AMI and may reflect the degree of myocardial damage and ventricular dysfunction. CRP not only reflects the underlying inflammatory processes that contribute to plaque instability and rupture, but may also play a direct role in promoting endothelial dysfunction and thrombosis.

Aim: To assess the levels of serum CRP in patients with AMI and to determine the correlation of CRP with cardiac enzymes and left ventricular function.

Materials and Methods: This cross-sectional study was conducted at the Department of Medicine, Regional Institute of Medical Sciences (RIMS), Imphal, Manipur, from May 2022 to July 2024. A total of 81 patients aged≥18 years with AMI were included. Clinical data, Electrocardiogram (ECG) findings, serum CRP, cardiac enzymes {Creatine Kinase-Myocardial Band (CK-MB), High-sensitivity Troponin I (hs-TnI)}, and 2D echocardiographic parameters were recorded. Pearson's correlation was used for statistical analysis, with significance set at $p<0.05$.

Results: The mean age of patients was 63.6±12.4 years, with a male-to-female ratio of 1.5:1. Chest pain (93.8%) and shortness of breath (43.1%) were common symptoms. Risk factors included smoking (63%), hypertension (46.9%), and diabetes (33.3%). Mean CRP was 20.8±20 mg/L, CK-MB 74.1±53.5 U/L, and hs-TnI 7.42±9.39 ng/mL. CRP negatively correlated with Left Ventricular Ejection Fraction (LVEF) ($r=-0.605$, $p<0.001$) and positively with Regional Wall Motion Abnormalities (RWMA) segments ($r=0.664$, $p<0.001$) and Left Ventricular Diastolic Dysfunction (LVDD) grade ($r=0.462$, $p<0.001$). hs-TnI showed similar correlations: negative with LVEF ($r=-0.591$, $p<0.001$) and positive with RWMA ($r=0.651$, $p<0.001$) and LVDD ($r=0.462$, $p<0.001$). CK-MB was positively correlated with RWMA ($r=0.384$, $p<0.001$), but not significantly with LVEF or LVDD.

Conclusion: Elevated serum CRP levels correlated significantly with impaired Left Ventricular (LV) function in AMI patients. CRP may serve as a useful biomarker for assessing the severity and prognosis of myocardial infarction.

Keywords: Atherosclerosis, Coronary artery disease, Inflammatory marker, Troponin I

INTRODUCTION

Coronary Artery Disease (CAD) is a spectrum of disorders causing reduced blood flow to the myocardium. CAD accounted for around eight million deaths worldwide in 2013, and its incidence has been increasing [1]. Inflammation plays an important role in the development of atherosclerosis and ischaemic heart disease. Marked serum CRP elevation after AMI is associated with adverse outcomes such as cardiac rupture, LV remodeling, LV mural thrombosis, and cardiac death [2]. In addition, CRP levels in the acute phase of Myocardial Infarction (MI) are powerful independent markers of heart failure and long-term mortality [3]. The diagnosis of myocardial infarction is established when typical symptoms are present along with supportive evidence of ECG changes, a rise or fall of cardiac biomarkers, or echocardiographic evidence of recent loss of viable myocardium or newly detected RWMA [4].

Inflammation plays an important role in the development of atherosclerosis and ischaemic heart disease. Vascular inflammation precedes the clinical syndromes of cardiovascular disease and has an important role in the pathogenesis of atherosclerosis by mediating different stages of plaque development from lipid streak formation to rupture and destabilisation of the plaque [5]. AMI is associated with a systemic inflammatory response with augmented production of nonspecific plasma acute-phase proteins, including CRP [6]. Myocardial necrosis, which follows abrupt occlusion of a coronary artery, leads to regional as well as systemic inflammatory responses. The early phase of AMI shows an increase in cytokine activity aimed at promoting the local myocardial healing process. However, increasing cytokine levels persisting for a long duration increases damage to the myocardial tissue. Cytokines such as CRP cause necrosis of myocardial cell membranes and facilitate complement activation, leading to expansion of necrosis [7].

The marked serum CRP elevation after acute MI is associated with adverse outcomes such as cardiac rupture, LV remodeling, LV mural thrombosis, and cardiac death. In addition, CRP in the acute phase of AMI is a powerful independent marker of heart failure and long-term mortality [3]. Therefore, various inflammatory serum biomarkers are being assessed as potential tools for predicting acute coronary disease [8-10]. It has been shown that CRP may not only be a biomarker of generalised inflammation but may also have a direct, active role in both atherogenesis and plaque disruption [9]. These inflammatory biomarkers have become valuable tools for studying acute coronary events and the prognosis of various therapeutic interventions.

Several studies have reported an independent association between CRP and the recurrence of myocardial ischemia and death during follow-up [10,11]. The effect of CRP on postinfarct LV dysfunction has been attributed to increased rates of apoptosis, macrophage infiltration, monocyte chemoattractant protein-1 expression, and matrix metalloproteinase-9 activity in adjoining myocardial tissue, which relate to the severity of long-term sequelae [12].

No study has been conducted in this region to evaluate the correlation of CRP with parameters of LV function in patients with myocardial infarction. Therefore, this study was undertaken to assess CRP levels and analyse the correlation between CRP, cardiac enzymes, and LV function in patients with myocardial infarction.

MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted at the Department of Medicine, Regional Institute of Medical Sciences (RIMS), a tertiary care hospital in Imphal, Manipur, India, over two years and two months, from May 2022 to July 2024. Ethical approval was obtained from the Research Ethics Board (REB) of the Regional Institute of Medical Sciences, Imphal, with ethical clearance number No. A/206/REB-Comm(SP)/RIMS/2015/901/239/2022. Participants were informed about the nature of the study and provided written consent. They were assured of their right to withdraw from the study at any time. Privacy and confidentiality were maintained by coding the data and restricting access to the investigators, study staff, and REB members. The study was self-sponsored, with no conflicts of interest.

Sample size calculation: Using the prevalence value given in the study "Association between CRP velocity and left ventricular function in patients with ST-elevated myocardial infarction" by Banai A et al., (2021) [13], sample size was calculated as follows. $n=4PQ/L^2$, where P is the prevalence of LV dysfunction in patients with raised CRP in myocardial infarction (16.02%), Q=100 – P=83.98%, and L is the absolute allowable error (taken as 8%). This yields $n \approx 80.64 \approx 81$.

Inclusion and exclusion criteria: Patients aged 18 years and above who were admitted with AMI to the Medicine Ward or ICCU of RIMS Hospital, Imphal, Manipur, India, were included in the study, diagnosed according to the World Health Organisation (WHO) criteria and the joint ESC/ACCF/AHA/WHF Fourth Universal Definition of Myocardial Infarction [4]. Exclusion criteria included pre-existing heart failure, acute infections, or recent surgery within the preceding two weeks. Additional exclusions included the presence of malignancy, liver failure, renal failure, thyroid disorders, or any chronic inflammatory diseases.

Study Procedure

Methodology: Participants were recruited using purposive-convenience sampling, and those fulfilling the inclusion criteria were selected. Recruitment occurred on all working days from all subunits of the Department of Medicine.

Study variables:

- Independent variables: Age, sex, hypertension, diabetes mellitus, smoking history, ECG findings, and serum CRP.
- Outcome variables: Cardiac enzymes, 2D echocardiography parameters (LVEF, number of RWMA segments), and LV diastolic dysfunction (LVDD) grades 0-3 [14].

Data collection methods:

- Blood samples
- Serum CRP: Collected at admission in standardised plain vials
- Cardiac enzymes: Collected four hours after the onset of chest pain in plain vials
- 2D echocardiography: Conducted during the patient's hospital stay

Laboratory methods:

- Serum CRP was measured using Meril ProViso (manufactured in India)
- Cardiac enzymes and lipid parameters were quantified using a miniVIDAS machine (manufactured in India)
- 12-lead electrocardiograph was recorded using a CARDIART Gen X3 machine (manufactured in India)
- Echocardiography was performed using an Esaote MyLab X6 echocardiography machine (manufactured in India) by a certified cardiologist

STATISTICAL ANALYSIS

- Descriptive statistics: Summarised the characteristics of the study population and key variables (means, standard deviations, minimum and maximum values, and frequencies for categorical variables)
- Inferential statistics: Pearson correlation was used to examine linear relationships between continuous variables, with correlation coefficients ranging from -1 to 1
- Significance levels: Set at 5% (alpha=0.05) for most analyses, with p-values between 0.05 and 0.10 considered suggestive, between 0.01 and 0.05 considered moderate, and ≤0.01 considered strong
- All analyses were conducted using Statistical Package for the Social Sciences (SPSS) version 26.0.

RESULTS

Demographic and clinical characteristics: The study included 81 consecutive patients diagnosed with AMI. The demographic and clinical characteristics are as follows. The mean age was 63.6 years, with males affected more than females. The older age group was affected more than the younger age group [Table/Fig-1]. Chest pain was the most common presenting complaint, while palpitations were the least common presenting complaint. This is consistent with the clinical features of acute coronary syndrome [Table/Fig-2]. Hypertension, diabetes mellitus, and smoking, traditional risk factors for acute coronary syndrome, were also significantly present in the study population [Table/Fig-3]. Mean CRP was 20.8±20 mg/L, CK-MB 74.1±53.5 U/L, and hs-Troponin I 7.42±9.39 ng/mL [Table/Fig-4].

Demographics	Frequency N (%)
Mean age	63.6 (±12.4) years
Age <40 years	2 (2.5)
Age >40 years	79 (97.5)
Male	49 (60.4)
Female	32 (39.6)
Male:Female ratio	1.5:1

[Table/Fig-1]: Demographics of the study population.

Presenting complaints	Frequency N (%)
Chest pain	76 (93.8)
Shortness of breath	35 (43.1)
Epigastric pain	19 (23)
Syncope	16 (19.6)
Sweating	9 (11)
Nausea/vomitting	8 (10.8)
Palpitations	5 (6.1)

[Table/Fig-2]: Primary complaints of the study population.

Risk factors	Yes	No
Hypertension	38 (46.9%)	43 (53.1%)
Diabetes mellitus	27 (33.3%)	54 (66.7%)
Smoking	51 (63%)	30 (37%)

[Table/Fig-3]: Risk factors in the study population.

Parameters	Mean±SD	Minimum (Maximum)	25th percentile	50th percentile	75th percentile	Shapiro-Wilk W	Shapiro-Wilk p
CRP (mg/L)	20.8±20.0	5.08 (86.02)	7.58	10.7	25.7	0.735	<0.001
CK-MB (U/L)	74.1±53.5	21 (422)	40.0	68.0	90.0	0.715	<0.001
HsTroponinI (ng/mL)	7.42±9.39	0.20 (40.0)	1.60	3.20	7.90	0.685	<0.001
LVEF (%)	40.2±7.93	28 (60)	34	40	46	0.930	<0.001

[Table/Fig-4]: Table showing frequency distribution of CRP, CK-MB, HsTropI, LVEF.

Using the 17-segment model for the assessment of RWMA, the maximum number of RWMA segments observed was four. Five patients had no RWMA, while only one patient had the maximum number of RWMA segments, i.e., 10 (out of the 17 segments of the RWMA model) [Table/Fig-5]. As seen in [Table/Fig-4,6], myocardial infarction affected the systolic function of the LV, as shown by a decrease in LVEF, and it also affected LV diastolic function, as shown by the presence of grade 1-2 diastolic dysfunction. Correlations [Table/Fig-7] show that CRP had a significant positive correlation with the cardiac biomarkers CK-MB and hs-Troponin I. [Table/Fig-8] shows that CRP had a significant positive correlation with the number of RWMA segments and with LVDD. A significant negative correlation existed between CRP and LVEF. [Table/Fig-9] shows that CK-MB exhibited no significant correlation with LVEF. CK-MB significantly correlated positively with the number of RWMA segments and showed no statistically significant correlation with LVDD grade. [Table/Fig-10] shows that hs-Troponin I had a significant negative correlation with LVEF and significant positive correlations with the number of RWMA segments and with LVDD grade.

No. of RWMA segments	Frequency (%)
0	5 (6.2)
1	0 (0)
2	3 (3.7)
3	11 (13.6)
4	28 (34.6)
5	17 (21)
6	11 (13.6)
7	2 (2.5)
8	1 (1.2)
9	2 (2.5)
10	1 (1.2)

[Table/Fig-5]: Table showing the frequency distribution of number of RWMA segments in the study population.

LVDD (grade)	Frequency (%)
0	0 (0.0%)
1	36 (44.4%)
2	45 (55.6%)
3	0 (0.0%)

[Table/Fig-6]: Table showing frequency distribution of Left Ventricular Diastolic Dysfunction (LVDD).

Variables		CRP (mg/L)	CKMB (U/L)	HsTrop I (ng/ml)
CRP (mg/L)	Pearson Correlation	1	0.363**	0.992**
	Sig. (2-tailed)		<0.001	<0.001
CKMB (U/L)	Pearson Correlation	0.363**	1	0.344**
	Sig. (2-tailed)	<0.001		0.002
HsTrop I (ng/mL)	Pearson Correlation	0.992**	0.344**	1
	Sig. (2-tailed)	<0.001	0.002	

[Table/Fig-7]: Table showing the correlation of CRP with CKMB and HsTrop I.
**. Correlation is significant at the 0.01 level (2-tailed)

Variables		CRP (mg/L)	LVEF (%)	RWMA No. of Segments	LVDD (grade)
CRP (mg/L)	Pearson Correlation	1	-0.605**	0.664**	0.462**
	Sig. (2-tailed)		<0.001	<0.001	<0.001
LVEF (%)	Pearson Correlation	-0.605**	1	-0.763**	-0.668**
	Sig. (2-tailed)	<0.001		<0.001	<0.001
RWMA No. of Segments	Pearson Correlation	0.664**	-0.763**	1	0.638**
	Sig. (2-tailed)	<0.001	<0.001		<0.001
LVDD (grade)	Pearson Correlation	0.462**	-0.668**	0.638**	1
	Sig. (2-tailed)	<0.001	<0.001	<0.001	

[Table/Fig-8]: Table showing the correlation of CRP with various LV parameters.
**Correlation is significant at the 0.01 level (2-tailed)

Variables		CKMB (U/L)	LVEF (%)	RWMA No. of Segments	LVDD (grade)
CKMB (U/L)	Pearson correlation	1	-0.140	0.384**	0.195
	Sig. (2-tailed)		0.214	<0.001	0.080
LVEF (%)	Pearson correlation	-0.140	1	-0.763**	-0.691**
	Sig. (2-tailed)	0.214		<0.001	<0.001
RWMA No. of Segments	Pearson correlation	0.384**	-0.763**	1	0.641**
	Sig. (2-tailed)	<0.001	<0.001		<0.001
LVDD (grade)	Pearson correlation	0.195	-0.691**	0.641**	1
	Sig. (2-tailed)	0.080	<0.001	<0.001	

[Table/Fig-9]: Table showing the correlation of CKMB and various LV parameters.
**Correlation is significant at the 0.05 level (2-tailed)

Variables		HsTrop I (ng/mL)	LVEF (%)	RWMA No. of Segments	LVDD (grade)
HsTrop I (ng/mL)	Pearson Correlation	1	-0.591**	0.651**	0.462**
	Sig. (2-tailed)		<0.001	<0.001	<0.001
LVEF (%)	Pearson Correlation	-0.591**	1	-0.763**	-0.691**
	Sig. (2-tailed)	<0.001		<0.001	<0.001
RWMA No. of Segments	Pearson Correlation	0.651**	-0.763**	1	0.641**
	Sig. (2-tailed)	<0.001	<0.001		<0.001
LVDD (grade)	Pearson Correlation	0.462**	-0.691**	0.641**	1
	Sig. (2-tailed)	<0.001	<0.001	<0.001	

[Table/Fig-10]: Table showing the correlation of HsTrop I with various LV parameters.
**Correlation is significant at the 0.01 level (2-tailed)

DISCUSSION

The mean age of the participants in the present study, as shown in [Table/Fig-1], was 63.6±12.4 years, whereas the mean age in a previous study by Tomoda H et al., was 64±10 years [15]. Myocardial infarction is more common in the older age group. However, recent trends in cardiovascular disease show that younger age groups are also experiencing MI. The risk factors in these younger groups differ from the traditional risk factors seen in older age groups [9].

As shown in [Table/Fig-1], the sex distribution was 60.4% males and 39.6% females. In a study by Toss H et al., females accounted for 35% and males for 65%, which is comparable to our present study [16]. In a previous study by Ferreirós ER et al., there were 64% males and 36% females, showing male preponderance in AMI [17]. This may be attributed to the higher prevalence of traditional risk factors among men compared with women. Women are also protected to some extent by estrogen [18].

The most common presenting complaint in our study, as referenced in [Table/Fig-2], was chest pain (93.8%), and the least common was palpitations (6.1%), which is similar to the study conducted by Sinha SK et al., where chest pain and palpitations were seen in 94.8% and 4.2% of the participants, respectively [19]. The usual presenting complaint of AMI is chest pain described classically as "Angina Pectoris," which is not relieved by rest or medication. Liuzzo G et al., demonstrated that hypertension was present as a risk factor in 45% of the participants, which is similar to our study, where hypertension is present in 46.9% of the participants as shown in [Table/Fig-3] [20].

In the study conducted by Shukla AN et al., the prevalence of tobacco use among myocardial infarction patients was 49.7%, which is slightly lower than our present study, where smoking is seen in 63% of the patients, as shown in [Table/Fig-3] [21]. This can be explained by the broader age range in the present study, whereas Shukla AN et al., studied a younger age group [21].

Arnold SV et al., in their study, showed diabetes mellitus in 38% of participants with AMI [22]. This finding is similar to our present study, where type 2 diabetes mellitus (T2DM) was seen in 33.3% of the participants, as shown in [Table/Fig-3]. Similar results were reported by Tenerz A et al., where one in four patients with myocardial infarction had diabetes [23].

As shown in [Table/Fig-4], the CRP value in our present study had a mean of 20.8±20 mg/L. This finding is similar to the value reported by Berk BC et al., who found a mean CRP of 22±29 mg/L [24]. In our study, the median hs-Troponin I was 3.2 ng/mL, which is similar to the study by Daniel FS et al., where the median hs-Troponin I was 2.063 ng/mL [25]. In a previous study by Chew DS et al., the mean LVEF was 40% with a range of 36–55%, roughly similar to our mean LVEF of 40.2±7.93% [26].

Poulsen SH et al., demonstrated that LVDD was present in 62% of AMI patients, of which 38% had impaired left ventricular relaxation; this is similar to our study, which showed 44.4% with impaired relaxation as shown in [Table/Fig-6] [27]. Myocardial injury during the event also affects the relaxation properties of the ventricles, contributing to diastolic dysfunction.

Aseri ZA et al., showed that CRP and cardiac biomarkers have a positive correlation in patients with AMI and can help predict long-term outcomes such as heart failure and death [28]. Morrow DA et al., also reported that CRP levels rise in parallel with biomarker levels, indicating the extent of myocardial necrosis [29]. Ulucay A et al., however, demonstrated opposing results and showed no relation between CRP and the presence or severity of CAD [30].

Our present study showed a positive correlation not only between CRP and cardiac biomarkers as indicated in [Table/Fig-7], but also between CRP and the degree of left ventricular dysfunction as shown in [Table/Fig-8]. Left ventricular dysfunction was assessed as systolic dysfunction and diastolic dysfunction. LVEF and the number of RWMA segments were used to define left ventricular dysfunction. LVDD was graded into four grades (0, 1, 2, 3) according to the 2D echocardiographic finding of the E/A ratio.

CRP levels correlated positively with cardiac biomarker levels. CRP was also positively correlated with the number of RWMA segments; higher CRP values were associated with a greater number of RWMA segments, whereas lower CRP values were associated with fewer RWMA segments. By contrast, CRP was negatively correlated with LVEF. In addition, higher CRP and higher cardiac biomarker values were associated with greater grades of diastolic dysfunction, while higher cardiac biomarker values were associated with greater degrees of systolic dysfunction, as shown in [Table/Fig-9,10].

This is concordant with the extensive study by Swiatkiewicz I et al., which showed that in-hospital CRP values correlated with 2D echocardiographic findings and predicted postinfarct left ventricular remodeling at six months after a first STEMI [31]. Similar results were reported by Mather AN et al., in a smaller subset of participants. Ørn S et al., also demonstrated a significant correlation between CRP and the predictive power of CRP for LV remodeling at two months [32,33].

CRP is an acute-phase reactant and one of the best-studied inflammatory biomarkers in acute coronary syndromes. CRP levels rise in response to infection, inflammation, or tissue injury. It not only serves as a biomarker but also actively participates in the inflammatory processes of atherogenesis and the pathogenesis of unstable angina and other coronary syndromes [34]. CRP is produced at the site of the culprit plaque, is involved in plaque development, and contributes to plaque vulnerability. CRP has also been shown to be related to restenosis after coronary intervention and is thought to have both prothrombotic and proapoptotic properties [35].

Circulating CRP levels have been shown to relate to other traditional risk factors such as hypertension, T2DM, smoking, and dyslipidemia. Several studies have demonstrated an independent association between CRP and cardiac-related mortality and long-term outcomes after CAD events [36-38]. Moreover, CRP has been associated with infarct size and the extent of myocardial damage. Some studies have also linked CRP levels to recurrent coronary events [36].

Our study adds to the previous evidence of a significant increase in CRP during AMI. Myocardial necrosis, which occurs after abrupt occlusion of a coronary artery, triggers regional and systemic inflammatory responses. CRP, an inflammatory marker, is involved in processes that can lead to necrosis of myocardial cell membranes, and the expansion of necrosis and loss of myocardial tissue can result in long-term sequelae such as heart failure, left ventricular remodeling, left ventricular dysfunction, rupture of the ventricles, reinfarction, restenosis, etc.

Limitation(s) and Future Directions

Despite its contributions, our study is limited by its cross-sectional and single-center design, potentially limiting generalisability. Future research should incorporate larger, multicenter cohorts with longitudinal follow-up to elucidate the temporal relationships between serum CRP and clinical outcomes post-AMI. Addressing confounding factors such as comorbidities, genetic variations, and therapeutic interventions will enhance the clinical applicability of serum CRP in AMI management. Furthermore, a follow-up study will be required to ascertain the predictability of CRP and other parameters for long-term sequelae. A regression model to predict the outcome of myocardial infarction based on CRP and other parameters could be valuable for predicting the long-term sequelae of AMI.

CONCLUSION(S)

In our study of AMI patients, elevated CRP levels were significantly associated with increased cardiac biomarker levels (hs-Troponin I and CK-MB), reduced LVEF, greater regional wall motion abnormalities, and more severe diastolic dysfunction. Among the biomarkers assessed, high-sensitivity Troponin I exhibited the strongest correlations with echocardiographic parameters of myocardial injury. In contrast, CK-MB showed a limited correlation with functional cardiac impairment. These findings highlight CRP as a valuable inflammatory marker with potential utility in assessing the severity and prognosis of myocardial infarction.

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