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HIGH SENSITIVITY C- REACTIVE PROTEINS AS A MARKER OF IN- HOSPITAL MORBIDITY & MORTALITY IN PATIENTS WITH MYOCARDIAL INFARCTION

Cardiology

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ABSTRACT

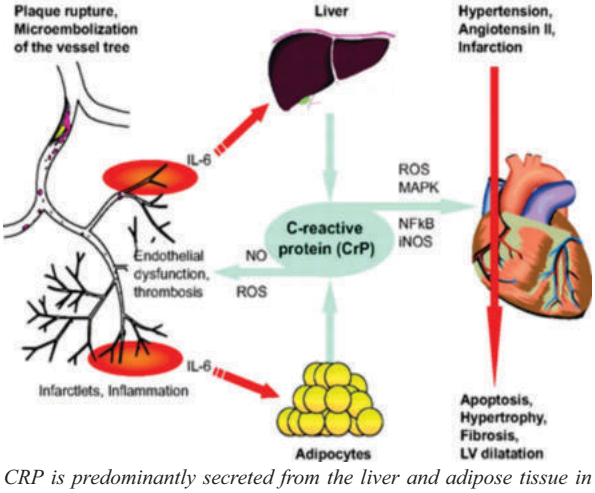
High-sensitivity C-reactive protein (hsCRP) is a well-established marker of systemic inflammation and has increasingly been recognized as an important prognostic indicator in acute myocardial infarction (AMI). Numerous studies have demonstrated that elevated CRP correlates with plaque instability, myocardial injury, and risk of recurrent events.[1,2] This prospective observational study evaluated 103 patients with ST-elevation AMI to determine the relationship between admission hsCRP levels, traditional cardiovascular risk factors, left-ventricular function, and in-hospital outcomes. Elevated hsCRP values were strongly associated with diabetes, hypertension, smoking, dyslipidemia, and previous ischemic heart disease, [3] reflecting the chronic inflammatory burden linked to these conditions. Patients with higher hsCRP also demonstrated significantly reduced left-ventricular ejection fraction, suggesting a direct correlation between inflammatory activity, myocardial injury, and impaired ventricular performance. Furthermore, elevated hsCRP was significantly associated with major in-hospital complications, including reinfarction, arrhythmias, congestive cardiac failure, stroke, and cardiac death.[4] These findings support the role of hsCRP as a robust, inexpensive, and easily measurable biomarker that provides meaningful prognostic information in AMI. Incorporating hsCRP into routine clinical evaluation may help identify high-risk patients who require closer monitoring and more intensive therapeutic strategies to improve early outcomes.

KEYWORDS

High-sensitivity C-reactive protein; Acute myocardial infarction; Left ventricular dysfunction; In-hospital complications; Cardiovascular risk factors

INTRODUCTION

Acute myocardial infarction (AMI) remains a leading global cause of mortality, with inflammation now recognized as a central driver in atherogenesis and plaque rupture.[1] CRP, the classical acute-phase reactant first described by Tillet and Francis in 1930,[5] rises rapidly following tissue injury and serves as a sensitive biomarker of systemic inflammation. Its synthesis is primarily regulated by IL-6 released during inflammatory states.[6] High-sensitivity assays (hsCRP) permit detection of low-grade inflammation and facilitate early cardiovascular risk stratification.[7] Evidence indicates that CRP not only marks inflammation but also contributes pathogenically by binding modified LDL, activating complement, inducing tissue factor, impairing nitric oxide activity, and destabilizing plaques[1]. Given these mechanistic links and its strong prognostic value in prior studies[8], evaluating hsCRP levels in AMI patients may provide critical insight into risk prediction and clinical outcomes. The present study therefore examined admission hsCRP values in AMI and their association with complications and risk factors.



CRP is predominantly secreted from the liver and adipose tissue in response to inflammatory stress resulting from plaque rupture and subsequent microembolization. High levels of CRP induce endothelial dysfunction, activate platelets, and aggravate stress-induced alterations in cardiac function and morphology

Figure 1: C-reactive Protein And Inflammatory Response

MATERIALS AND METHODS

This study was carried out at MUMBAI PORT TRUST HOSPITAL, WADALA Mumbai, Department of Medicine from August 2005 to August 2006.

Selection Criteria:

The study included all cases presenting with an episode of Acute myocardial infarction being admitted in Intensive Cardiac Care Unit (ICCU) having chest pain of less than 6 hrs. duration. The diagnosis of acute myocardial infarction was based on:

1. Typical history of an acute coronary syndrome and
2. ECG Criteria based on WHO recommendations - > 0.1 mV ST segment elevation in at least one standard or two precordial leads
3. Elevation of Cardiac enzymes (CPK-MB > 2 times normal range).

The presence of any two of these criteria were considered as indicative of Acute Myocardial Infarction.

Pts. of Myocardial Infarction were thrombolysed with Streptokinase if they had no contraindication to thrombolysis. Only those pts who were thrombolysed were included in the study.

Exclusion Criteria:

Conditions known to be associated with raised CRP levels formed the basis for exclusion in most cases.

1) Advanced Liver disease.
2) Collagen Vascular diseases.
3) Major injury or surgery within last one month.
4) Any acute or Chronic infections.
5) Renal Failure, acute or chronic
6) Malignancy.

Study Protocol:

All patients diagnosed to have myocardial infarction admitted to intensive cardiac care unit were studied.

The details regarding chest pain in relation to duration, type and site, onset, frequency and radiation were noted. In the past history, particular attention was paid to previous myocardial infarction, angina pectoris, hypertension, and diabetes mellitus. Family history of ischemic heart disease, hypertension and diabetes mellitus in immediate blood relations were noted.

Personal history was asked carefully as regards to diet and addictions

to alcohol and smoking. Detailed physical examination was carried out in all cases. Height and weight were recorded. Waist circumference was measured one inch above umbilicus. The maximum circumference over the buttock with minimum clothing was recorded as hip circumference.

Waist - hip ratio was calculated.

In all cases, baseline 12 lead ECG was recorded immediately on admission. The acute coronary syndrome was classified as per **Schamroth**[9] as:
1. Anterior wall myocardial infarction if changes noted in lead I, aVL, V2-V6
2. Inferior wall infarction if changes were noted in II, III, aVF.
3. Others - including either of this combination with right ventricular or posterior wall infarction or right ventricular or posterior wall infarction alone.

Blood samples were taken for estimation of Haemogram with total leucocyte count, differential leucocyte count and ESR were estimated. Blood sugar (Glucose oxidase method) and serum lipid profile was estimated. Patients having Total cholesterol > 200 mg/dl and Serum Triglycerides > 160 mg/dl were taken to have dyslipidemia.

hsCRP estimation was done immediately on admission and analyzed by the immunoturbidimetry method. The normal value of hsCRP by this method is 0.1 to 6 mg/ml.

Patients were examined in detail for 7 days for development of complications. ECGs were done daily. 2D ECHO was done during in hospital stay. The complications monitored were reinfarction, congestive cardiac failure, arrhythmia, stroke and sudden cardiac death.

Statistical Analysis:
Statistical analysis was done on a personal computer. Student t test was applied for continuous variables and Chi square test for categorical variables. Multilogistic regression analysis between C reactive protein, risk factors and complications was done using statistical software STATA ver 70, STATA Corporation, USA.

OBSERVATION AND RESULTS.
A total of 103 patients diagnosed with acute myocardial infarction were included in this prospective observational study. The study cohort demonstrated a clear male predominance, with **80% males** and **20% females**, reflecting established epidemiological patterns in AMI where men experience higher event rates at comparatively younger ages.[8] The mean age of the participants was **55 years**, consistent with observations from earlier Indian and global cohorts indicating a shift toward younger AMI presentation.[8]

Clinical Presentation:
Anterior-wall MI (AWMI) was the most common presentation, occurring in **56%** of patients, whereas **44%** presented with inferior-wall MI (IWMI). This distribution is relevant as AWMI is frequently associated with larger infarct size, heightened inflammatory response, and worse prognosis, which aligns with previous findings demonstrating greater CRP elevations in anterior infarctions.[10]

Risk Factor Profile:
A substantial proportion of patients exhibited traditional cardiovascular risk factors. Smoking was the most prevalent risk factor (**56.3%**), followed by abnormal waist–hip ratio (**55.3%**), previous ischemic heart disease (**32%**), dyslipidemia (**24.3%**), diabetes mellitus (**22.3%**), and hypertension (**21.3%**). The clustering of these metabolic and lifestyle-related factors is strongly supported by population-based evidence linking each of these conditions to elevated systemic inflammation and high CRP levels.[3]

Figure 2: Risk Factors And hsCRP On Admission In pts. Of Acute Myocardial Infarction:

Risk Factor	No. of pts (n=103)	Mean hsCRP (mg/dl)	t value	p-value
Previous h/o IHD	33	14.0	3.97	0.0001
No Previous h/o IHD	70	4.31		
Diabetic	23	14.9	5.40	< 0.0001
Non Diabetic	80	3.47		
Hypertensive	22	9.67	2.48	0.015
Non - Hypertensive	81	4.43		
Smoker	58	9.03	3.65	0.0004
Non Smoker	45	2.12		
Dyslipidemia ++	47	9.09	2.95	0.0039
Dyslipidemia --	56	3.42		
Abnormal WHR	57	6.99	1.10	0.280
Normal WHR	46	4.80		

Various risk factors and relation of risk factors with hsCRP in pts of MI was studied. It was found that serum hsCRP levels were significantly higher in diabetics (p-value < 0.0001), smokers (p-value =0.0004), pts with h/o hypertension (p-value = 0.015), with h/o previous IHD (p-value=0.0001), pts with dyslipidemia (p-value =0.0039), but not in pts of abnormal waist - hip ratio (p-value = 0.28)

hsCRP Measurements And Associations:
Admission hsCRP levels were distributed across a broad spectrum, with a marked trend toward significantly higher values among individuals with established cardiovascular risk factors. Statistically significant elevations in hsCRP were noted among diabetics ($p < 0.0001$), hypertensives ($p = 0.015$), smokers ($p = 0.0004$), dyslipidemic patients ($p = 0.0039$), and those with a history of ischemic heart disease ($p = 0.0001$). These findings closely reflect large cohort studies that found increased hsCRP levels in patients with metabolic syndrome, smoking, and insulin resistance.[11]

Additionally, hsCRP levels were comparatively higher in patients presenting with AWMI versus IWMI, suggesting an association between infarct severity and magnitude of inflammatory activation. Although this difference did not reach statistical significance in this cohort, the trend parallels earlier reports observing greater CRP rises in larger infarcts and in those involving the anterior wall.[10]

Left Ventricular Function:
A clear inverse relationship was noted between hsCRP and left-ventricular ejection fraction (LVEF). Patients with hsCRP > 3 mg/dL demonstrated significantly lower LVEF at presentation for both AWMI and IWMI subgroups. This observation underscores the established mechanistic links between heightened inflammation, myocardial necrosis, and impaired ventricular recovery.[4]

Figure 3: Relationship Between hsCRP And Ejection Fraction

Type of IHD	hsCRP	N	Mean hsCRP (mg/dl)	EF% on 2D Echo	p-value
AWMI	>3 mg/dl	34	10.29	34.3%	0.0004
AWMI	<3 mg/dl	24	1.28	42.8%	
IWMI	>3 mg/dl	22	8.18	39.8%	0.0014

Pts with AWMI and high hsCRP (>3 mg/dl) had a lower ejection fraction than those with a lower hsCRP level (<3 mg/dl) (34.3% vs. 42.8%, p value = 0.004). Same findings were seen in pts with IWMI (39.8% vs. 47.2%, p-value = 0.0014). The association between high hsCRP value and low Ejection Fraction was statistically significant and was seen in all types of Ischemic heart disease.

In-hospital Complications:
Complications developed in a clinically meaningful proportion of patients:
- **Congestive cardiac failure (18 patients)**
- **Arrhythmias (13 patients)**
- **Stroke (6 patients)**
- **Reinfarction (5 patients)**
- **Cardiac death (5 patients)**

Higher hsCRP levels were significantly associated with the presence of these complications. This trend mirrors evidence from previous studies showing that elevated CRP predicts recurrent ischemia, pump failure, arrhythmic events, and short-term mortality.[4] Taken together, the results demonstrate that admission hsCRP serves not only as a biochemical marker but also as a prognostic indicator identifying individuals at elevated risk for clinical deterioration.

Figure 4: hsCRP And Individual Complications

Complication		Mean hsCRP	p-value
Reinfarction (Present / Absent)	Present	17.1	0.0001
	Absent	4.55	

CCF (Present / Absent)	Present	10.8	0.0001
	Absent	3.72	
Arrhythmia (Present / Absent)	Present	9.35	0.034
	Absent	4.64	
Stroke (Present / Absent)	Present	23.5	0.0001
	Absent	4.41	
Sudden Cardiac Death (Present / Absent)	Present	14.9	0.0012
	Absent	4.63	

On correlating complications with hsCRP levels, there was significant correlation between hsCRP and in-hospital complications like Reinfarction (p-value =0.0001), Congestive cardiac failure (p-value = 0.0001), arrhythmias (p-value = 0.034), stroke (p-value = 0.0001) and sudden cardiac death (p-value =0.0012).

DISCUSSION

The present study reinforces the role of hsCRP as a powerful marker of inflammation and a prognostic indicator in acute myocardial infarction (AMI). The significant association of elevated hsCRP with multiple traditional cardiovascular risk factors-including diabetes, hypertension, smoking, dyslipidemia, and abdominal obesity-is consistent with prior findings from large-scale population studies.[3] Chronic exposure to these risk factors contributes to low-grade inflammation, oxidative stress, endothelial dysfunction, and lipid abnormalities, all of which are reflected in elevated hsCRP levels.

The higher hsCRP trend observed in AWMI compared to IWMI can be attributed to larger infarct size and more extensive myocardial injury typically associated with anterior-wall involvement. This correlation aligns with findings from Pietilä et al., who demonstrated that CRP levels tend to mirror infarct magnitude and peak simultaneously with myocardial necrosis markers.[10] This relationship is clinically important because AWMI patients often experience higher complication rates, and hsCRP can act as a rapid surrogate marker for infarct burden.

The strong inverse association between hsCRP and LVEF observed in this study further supports the hypothesis that inflammatory activation contributes directly to ventricular dysfunction. Previous studies have shown that elevated CRP can impair myocardial contractility through various mechanisms, including increased oxidative stress, complement activation, endothelial dysfunction, and microvascular obstruction.[4] These mechanistic pathways help explain the deteriorated left-ventricular performance and delayed functional recovery in patients with elevated inflammatory markers.

The significant association of hsCRP with in-hospital complications-including reinfarction, arrhythmias, stroke, congestive cardiac failure, and mortality-reflects its broad clinical relevance. Studies by Tomoda, Anzai, Mishra, and others have shown that elevated hsCRP at admission or within the first 24 hours of MI is a robust predictor of early and late adverse events.[4] Mechanistically, CRP contributes to plaque instability, thrombogenesis, endothelial dysfunction, and microvascular inflammation, all of which promote recurrent ischemia and cardiac complications.[2,12]

These findings underscore that hsCRP is not merely a passive marker of inflammation but may actively participate in the pathogenesis of atherosclerotic progression and adverse outcomes. This is supported by extensive mechanistic evidence showing that CRP enhances monocyte adhesion, promotes tissue factor expression, activates complement pathways, suppresses nitric oxide production, and directly influences vascular smooth-muscle behavior.[12]

Given its rapid rise following myocardial injury, lack of diurnal variation, reproducibility, and cost-effectiveness, hsCRP represents a valuable adjunctive tool in early risk stratification of AMI patients.[13,14] Incorporating hsCRP into standard assessment protocols may help identify high-risk individuals who require more intensive monitoring, earlier invasive evaluation, or more aggressive therapeutic strategies, including anti-inflammatory and lipid-lowering agents.[14]

Overall, the observed relationships between hsCRP, risk factors, infarct characteristics, ventricular dysfunction, and adverse clinical outcomes strongly support its role in routine AMI evaluation. These findings complement the growing body of global evidence positioning hsCRP as an essential biomarker in cardiovascular risk prediction and management.

CONCLUSION

Elevated hsCRP levels at admission are strongly associated with adverse cardiovascular risk profiles, impaired ventricular function, and increased in-hospital complications in patients with acute myocardial infarction. These findings align with extensive global evidence demonstrating the prognostic value of CRP in acute coronary syndromes. Routine measurement of hsCRP can therefore enhance early risk stratification and inform clinical decision-making. Incorporating hsCRP into standard evaluation protocols may improve outcomes by identifying patients at heightened risk for complications and guiding timely, aggressive management.

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