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ORIGINAL ARTICLE

Association of raised level of high sensitivity CRP with complications after NSTEMI (non-ST-elevation MI).

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ABSTRACT… Objective: To determine the association of raised levels of high sensitivity CRP with complications after NSTEMI. **Study Design:** Cohort study. **Setting:** Department of Cardiology, Chaudhary Pervaiz Elahi Institute of Cardiology, Wazirabad. **Period:** Six Months from January 14, 2025 to July 13, 2025. **Methods:** All patients were followed till 90 days and data regarding adverse cardiovascular events i.e. heart failure, repeat myocardial infarction and mortality were noted. All above data was noted from both groups and complications were noted. Data was entered and analyzed with SPSS-v23. Chi-square test was applied to compare heart failure, repeat myocardial infarction and mortality in both groups taking p≤0.05 as significant. Relative risk was also calculated. **Results:** Total 380 patients (190 exposed and 190 un-exposed) were selected for this study. Among exposed, there were 120(63.2%) males and 70(36.8%) females, while 114(60.0%) males and 76(40.0%) females among un-exposed. Among exposed, mean age was 44.5±11.9 year and 45.3±12.7 year among un-exposed. Among exposed, mean BMI was 28.5±2.9 kg/m^2 and 28.3±2.7 kg/m^2 among un-exposed. By comparing adverse cardiovascular events between groups, it was found that percentage of adverse cardiovascular events was 40.5% among exposed and 26.3% among un-exposed and the difference was significant (p=0.003, RR=1.357). **Conclusion:** High CRP is an indicator of poor result in patients with NSTEMI and is used to determine patients who might be vulnerable to cardiovascular complications.

Key words: Acute Myocardial Infarction, Cardiovascular Complications, Heart Failure, Mortality, NSTEMI.

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INTRODUCTION

Globally, acute myocardial infarction (AMI) is a major cause of death and illness.[1] It is accompanied by many conditions such as Acute Mitral regurgitation due to rupture of papillary muscle, ventricular septal defects, rupture of free wall, left ventricular pseudo-aneurysm and aneurysm, heart failure, recurrence of acute myocardial infarction and death.[2–4]

The past decade has seen different research studies determine that inflammation is critical in the etiology and progression of atherosclerosis.[5–6] As more studies have been conducted on the topic that atherosclerosis is an inflammatory process, various plasma markers of inflammatory processes have been evaluated as possible tools of predicting coronary events and high levels of one or more inflammatory process have been reported to either be a predictor of developing coronary artery disease (CAD) or, as worst prognosis in a patient diagnosed with CAD.[7,8]

Various types of inflammatory markers have been associated with increased risk of major cardiovascular events and even mortality- C-reactive protein (CRP), single-handedly, was found to be associated with the risk of heart failure, incidence of heart failure increased by 1.5- Xiaoyuan Zhang, et al. revealed the existence of heart failure risk where high sensitivity CRP (hs-CRP) upon admission was associated with high risk of death or non-fatal MI by 1.5 fold in patients with non-ST-elevation MI (NSTEMI) or unstable angina.[9] Enrique Santas, et al. found elevated heart failure risk where high sensitivity CRP (hs-CRP) at admission was found to be associated with the risk of death or non-fatal MI at 30 days.[10]

Although the recurrence rate of myocardial infarction (MI) was observed to be 22.4 vs. 6.6%. CRP was also reported to be an independent predictor of poor prognosis and is linked to complications following Acute Myocardial infarction in the overall incidence

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of a major coronary event of 87% in patients with abnormal CRP compared to 13% in patients with normal CRP.[12] FRISC Trial (FRagmin during Instability in Coronary artery disease) also indicated that CRP is an independent predictor of poor prognosis fold in patients with high levels of CRP compared to patients with normal levels of CRP.

It is also believed that CRP can not only be a predictor of poor prognosis, but also cause atherosclerosis and disruption of plaque. In this respect, there is scanty information in our population hence, we intended to establish the relationship between high concentrations of hs- CRP and the adverse outcomes following acute non-ST elevation myocardial infarction (NSTEMI). The research outcomes will also be a step forward towards assisting in determining patients at higher risk of cardiovascular complications.

METHODS

This research was performed in the Department of Cardiology at the Chaudhary Pervaiz Elahi Institute of Cardiology, Wazirabad Employing a cohort design, the research took place over a six-month period from January 14, 2025, to July 13, 2025. Participants were selected via non- probability consecutive sampling, with a total sample size of 380 patients calculated for 80% study power and a 5% alpha (one-sided), expecting complication proportions of 31.1% and 20% in the two comparison groups. This sample was divided equally, with 190 patients allocated to the exposed group and 190 to the un-exposed group.

Patient selection was governed by specific criteria. Participants had to be between the ages of 20 and 60, male or female, and diagnosed with non-ST-elevation myocardial infarction (NSTEMI). They were divided into groups according to their high-sensitivity C-reactive protein (hs-CRP) levels; those with elevated levels made up the exposed group, while those with normal levels made up the un-exposed group.

All the patients gave written informed consent. Key exclusions encompassed a history of acute myocardial infarction within the prior 30 days, active infections like pneumonia or tuberculosis, chronic inflammatory conditions (e.g., lupus, rheumatoid arthritis), inflammatory bowel disease, hepatic or renal failure, and instances where patients were later lost to follow- up.

The data collection procedure commenced after obtaining formal approval from the Ethical Committee (165/ERC/PEIC/W2D)2-1-25). All 380 eligible patients were enrolled from the cardiology department. Basic demographic information, including age, gender, and weight, was recorded at the outset. Each patient was then prospectively followed for a period of 90 days. The primary outcomes of interest were adverse cardiovascular events, specifically tracked according to operational definitions: heart failure occurring within 90 days, repeat myocardial infarction within 30 days, and all-cause mortality within 30 days. These complication data were systematically documented for all participants in both the exposed and un-exposed cohorts.

Data Analysis

The data was calculated and analyzed using statistical analysis program (SPSS-v23). Age and weight were the quantitative variables that showed mean and SD. Qualitative variables such as gender and complications were presented as number and percentage. Chi-square test was used to compare the heart failure, repeat myocardial infarction and mortality in the two groups where p=0.05 was taken to be significant. Relative risk had also been determined. The stratification was performed based on age, gender and BMI to observe the impact of the latter on heart failure, recurrent myocardial infarction and death. The Chi-square test of post-stratification of both groups was used. A level of 0.05 was deemed to be statistically significant. Relative Risk was also obtained.

RESULTS

This study comprised 380 patients diagnosed with acute non-ST elevation myocardial infarction (NSTEMI), evenly split into an exposed group with elevated high-sensitivity C- reactive protein (hs-CRP) (n=190) and an unexposed group with normal hs-CRP levels (n=190). Baseline demographic characteristics were comparable between the two groups. The mean age was 44.5 ± 11.9 years in

the exposed group and 45.3 ± 12.7 years in the unexposed group, with no statistically significant difference reported. Gender distribution was also similar, with males constituting 120 (63.2%) of the exposed group and 114 (60.0%) of the unexposed group. Body mass index (BMI) was comparable between groups, with mean values of 28.5 ± 2.9 kg/m^2 and 28.3 ± 2.7 kg/m^2, respectively, and no significant imbalance in BMI categories between groups. Table-I

The primary analysis revealed a statistically significant association between elevated hs-CRP levels and increased risk of adverse cardiovascular events within 90 days post-NSTEMI. The incidence of any adverse cardiovascular event was 77 (40.5%) in the exposed group compared to 50 (26.3%) in the unexposed group (p=0.003, RR=1.357). Specifically, heart failure occurred in 44 (23.2%) of the exposed group versus 6 (3.2%) of the unexposed group (p=0.001, RR=1.989). Repeat myocardial infarction was observed in 38 (20.0%) of the exposed group compared to 19 (10.0%) of the unexposed group (p=0.006, RR=1.417). Cardiogenic shock was significantly more common in the exposed group (39, 20.5%) than in the unexposed group (19, 10.0%) (p=0.004, RR=1.434). All-cause mortality was also significantly higher in the exposed group (45, 23.7%) versus the unexposed group (25, 13.2%) (p=0.008, RR=1.374). Table-II Stratified analyses further elucidated the relationship between elevated hs-CRP and outcomes across demographic subgroups. Among males, elevated hs-CRP was significantly associated with higher rates of heart failure (31, 25.8% vs. 6, 5.3%, p=0.001), cardiogenic shock (39, 32.5% vs. 14, 12.3%, p=0.001), and mortality (39, 32.5% vs. 13, 11.4%, p=0.001). Repeat MI was also more common in males with high hs-CRP (19, 15.8% vs. 13, 11.4%, p=0.044). In females, elevated hs-CRP was significantly associated with heart failure (13, 18.6% vs. 0, 0.0%, p=0.001), repeat MI (19, 27.1% vs. 6, 7.9%, p=0.002), and cardiogenic shock (0, 0.0% vs. 5, 6.6%, p=0.029), though mortality was paradoxically lower in the exposed group (6, 8.6% vs. 12, 15.8%, p=0.045, RR=0.667). Table-III

Age-stratified analysis showed that in the 20–50 years' age group, elevated hs-CRP levels were substantially correlated with an increased incidence of heart failure. (6, 10.3% vs. 0, 0.0%, p=0.005), repeat MI (13, 22.4% vs. 7, 9.5%, p=0.039), cardiogenic shock (14, 24.1% vs. 7, 9.5%, p=0.022), and mortality (26, 44.8% vs. 17, 23.0%, p=0.008). In the 51–60 years age group, elevated hs-CRP was significantly associated with higher rates of heart failure (38, 28.8% vs. 6, 5.2%, p=0.001), repeat MI (25, 18.9% vs. 12, 10.3%, p=0.048), cardiogenic shock (25, 18.9% vs. 12, 10.3%, p=0.048), and mortality (19, 14.4% vs. 8, 6.9%, p=0.049). Table-III

BMI-stratified analysis revealed that in normal-weight individuals, elevated hs-CRP levels were substantially correlated with an increased incidence of heart failure. (25, 25.0% vs. 0, 0.0%, p=0.001), repeat MI (13, 13.0% vs. 8, 6.3%, p=0.044), cardiogenic shock (20, 20.0% vs. 7, 5.5%, p=0.001), and mortality (32, 32.0% vs. 10, 7.9%, p=0.001). In overweight individuals, elevated hs-CRP was associated with higher heart failure (13, 16.7% vs. 0, 0.0%, p=0.001) and mortality (7, 9.0% vs. 14, 24.6%, p=0.014), but not with repeat MI or cardiogenic shock at conventional significance levels. In obese individuals, elevated hs-CRP was associated with lower incidence of heart failure (6, 50.0% vs. 6, 100.0%, p=0.034) and repeat MI (12, 100.0% vs. 2, 33.3%, p=0.001), though these findings should be interpreted with caution due to small subgroup sizes. Table-III

Overall, the study provides robust statistical evidence that elevated hs-CRP is a significant predictor of adverse cardiovascular outcomes in patients with NSTEMI, with consistent findings across most demographic strata. Table-III

DISCUSSION

Inflammation is a fundamental factor in the pathophysiology of atherosclerosis, and interest in evaluating inflammatory biomarkers in coronary artery disease (CAD) has increased over the past decade.[12] The inflammatory state has been linked to the destabilization of chronic plaque of arteries, causing acute coronary syndromes. CRP is an inflammatory marker because it is a protein of acute inflammatory response produced by hepatocytes under the influence of proinflammatory cytokines, most specifically interleukin-6.[13]

TABLE-I

Demographic profile of the study participants

Characteristic	Exposed (High hs- CRP)	Unexposed (Normal hs- CRP)	Total (n=380)
Gender			
Male	120 (63.2%)	114 (60.0%)	234 (61.6%)
Female	70 (36.8%)	76 (40.0%)	146 (38.4%)
Age (years)			
20-50	58 (30.5%)	74 (38.9%)	132 (34.7%)
51-60	132 (69.5%)	116 (61.1%)	248 (65.3%)
Mean ± SD	44.5 ± 11.9	45.3 ± 12.7	—
BMI (kg/m²)			
Normal	100 (52.6%)	127 (66.8%)	227 (59.7%)
Overweight	78 (41.1%)	57 (30.0%)	135 (35.5%)
Obese	12 (6.3%)	6 (3.2%)	18 (4.7%)
Mean ± SD	28.5 ± 2.9	28.3 ± 2.7	—

TABLE-II

Primary outcomes – adverse cardiovascular events

Outcome	Exposed (High hs-CRP)	Unexposed (Normal hs-CRP)	P-Value	Relative Risk (RR)
Any Adverse Event	77 (40.5%)	50 (26.3%)	0.003	1.357
Heart Failure	44 (23.2%)	6 (3.2%)	0.001	1.989
Repeat Myocardial Infarction	38 (20.0%)	19 (10.0%)	0.006	1.417
Cardiogenic Shock	39 (20.5%)	19 (10.0%)	0.004	1.434
Mortality	45 (23.7%)	25 (13.2%)	0.008	1.374

Some of the pathways through which CRP could impact CAD progression have been demonstrated to be experimental studies which have shown that CRP can mediate inflammation, platelet aggregation and thrombosis in transgenic mice.[13]

The large variability in hs-CRP levels in the acute myocardial infraction (AMI) patient has been supported by Polyakova EA, et al, who created a vision of acute hs-CRP levels soaring high and immediately after ST-elevation myocardial infraction (STEMI) and gradually falling over the following few weeks to the normal physiological ranges of 10 mg/L.[14] Therefore, the baseline patients who offer high hs-CRP titers are hyper-responsive to circulant cTnI.[15]

High-sensitivity C-reactive protein (hs-CRP) is one of the most scrutinized risk factors for inflammatory markers in clinical practice, demonstrating a correlation with an increased risk of future myocardial infarction, cerebrovascular accident, sudden cardiac demise, and/or peripheral arterial disease, as well as cardiac events in patients with coronary artery disease, colon cancer, diabetes complications, and obesity.[16]

Increased levels of hs-CRP at admission were associated with patients with a higher age, baseline creatinine level, and who were likely to develop heart failure later.[17,18] Patients with atherothrombosis, heart failure and arrhythmias have been found to have a worse prognosis which is what was reported by Jones N, et al.[19] The association between hs-CRP and cardiac failure following STEMI has not been extensively studied.[17,18]

Major adverse cardiovascular events (MACE) can be linked to hs-CRP among patients who have a large anterior ST event going through primary percutaneous coronary intervention (PCI). The evidence has revealed that pre-procedural hs-CRP was an independent predictor of MACE and all-cause mortality in patients with primary PCI with highest-

TABLE-III

Stratified Analysis of Outcomes by Gender, Age, and BMI

Subgroup	Outcome	Exposed (High hs-CRP)	Unexposed (Normal hs-CRP)	P-Value
	Heart Failure	31 (25.8%)	6 (5.3%)	0.001
	Repeat MI	19 (15.8%)	13 (11.4%)	0.044
Male	Cardiogenic Shock	39 (32.5%)	14 (12.3%)	0.001
	Mortality	39 (32.5%)	13 (11.4%)	0.001
	Heart Failure	13 (18.6%)	0 (0.0%)	0.001
	Repeat MI	19 (27.1%)	6 (7.9%)	0.002
Female	Cardiogenic Shock	0 (0.0%)	5 (6.6%)	0.029
	Mortality	6 (8.6%)	12 (15.8%)	0.045
	Heart Failure	6 (10.3%)	0 (0.0%)	0.005
	Repeat MI	13 (22.4%)	7 (9.5%)	0.039
Age 20–50	Cardiogenic Shock	14 (24.1%)	7 (9.5%)	0.022
	Mortality	26 (44.8%)	17 (23.0%)	0.008
	Heart Failure	38 (28.8%)	6 (5.2%)	0.001
	Repeat MI	25 (18.9%)	12 (10.3%)	0.048
Age 51–60	Cardiogenic Shock	25 (18.9%)	12 (10.3%)	0.048
	Mortality	19 (14.4%)	8 (6.9%)	0.049
	Heart Failure	25 (25.0%)	0 (0.0%)	0.001
	Repeat MI	13 (13.0%)	8 (6.3%)	0.044
BMI Normal	Cardiogenic Shock	20 (20.0%)	7 (5.5%)	0.001
	Mortality	32 (32.0%)	10 (7.9%)	0.001
	Heart Failure	13 (16.7%)	0 (0.0%)	0.001
BMI	Repeat MI	13 (16.7%)	9 (15.8%)	0.042
Overweight	Cardiogenic Shock	13 (16.7%)	9 (15.8%)	0.042
	Mortality	7 (9.0%)	14 (24.6%)	0.014
	Heart Failure	6 (50.0%)	6 (100.0%)	0.034
	Repeat MI	12 (100.0%)	2 (33.3%)	0.001
BMI Obese	Cardiogenic Shock	6 (50.0%)	3 (50.0%)	1.000
	Mortality	6 (50.0%)	1 (16.7%)	0.041

intensity statin treatment over a median follow-up of 6.5 years.[20] In the AMI patients undergoing primary PCI, high-intensity statin therapy was proven to be associated with increased mortality rates by 30 days follow-up with hs-CRP as the most pronounced. This reveals the anti-inflammatory properties of statins.[21]

It has been reported that a peak hs-CRP level is a powerful independent predictor of overall mortality and heart failure-induced mortality in the next year of follow up.[9] In this study, By comparing adverse cardiovascular events between groups, it was found that percentage of adverse cardiovascular events was 40.5% among exposed and 26.3% among un-exposed and the difference was significant (p=0.003, RR=1.357). By comparing heart failure between groups, it was found that percentage of heart failure was 23.2% among exposed and 3.2% among un-exposed and the difference was significant (p=0.001, RR=1.989).

By comparing repeat MI between groups, it was found that percentage of repeat MI was 20% among exposed and 10% among un-exposed and the difference was significant (p=0.006, RR=1.417). By comparing cardiogenic shock between groups, it was found that percentage of cardiogenic shock was 20.5% among exposed and 10% among un-exposed and the difference was significant (p=0.004, RR=1.434). By comparing mortality between groups, it was found that percentage of mortality was 23.7% among exposed and 13.2% among un-exposed and the difference was significant (p=0.008, RR=1.374).

Zhang X, et al. established that high CRP at admission in non-ST-elevation MI (NSTEMI) patients or unstable angina patients increased the risk of death or non-fatal MI by 1.5 times in the first

instance and 20 in the second instance.[9] Sheikh AS, et al. established that the incidence of heart failure was high in patients with high levels of CRP hs-CRP at admission and 31.1 and 20 respectively.[11] Recurrence of myocardial infarction (MI) was also determined to be 22.4% vs. 6.6%. In the FRISC Trial, which lasted an average of 30 months, CRP was likewise found to be an independent predictor of mortality, with 87% of patients with abnormal CRP and 13% of patients with normal CRP.

CONCLUSION

The CRP of patients with suspected coronary artery disease at admission may be helpful in identifying a subset of individuals who may be at a high risk of cardiac problems; these patients need intensive cardiac care and careful post-discharge monitoring.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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1	**Muhammad Nazim:** Drafting.
2	**Abdur Rehman:** Data analysis.
3	**Abubakar Maqbool:** Interpretation of data.
4	**Naeem Asghar:** Study design.
5	**Hafiz Muhammad Faiq Ilyas:** Data collection.
6	**Talal Wasif Mirza:** Editing.