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ORIGINAL RESEARCH ARTICLE

Prognostic Significance of High-Sensitivity C-Reactive Protein, Lipoprotein A, and LDL/HDL Ratio in Patients with Acute Coronary Syndrome

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Abstract

Introduction: Acute coronary syndrome (ACS) is a major concern for cardiovascular mortality instead of the advancement of reperfusion therapy. Residual inflammatory risk was evaluated by the high-sensitivity C-reactive protein (hs-CRP), which has been developed as a significant biomarker for prognosis. The study objective was to assess the significance of the prognosis of the hs-CRP, lipoprotein(a), and LDL/HDL ratio in order to predict the cardiovascular outcomes and mortality among patients with ACS.

Method: The studywas **a** prospective observational cohort study conducted among 90 patients of ACS; those who were admitted in the ED were selected. All clinical history, biochemical parameters, and cardiovascular parameters were evaluated. All of the patients were categorised according to the level of hs-CRP. SPSS version 27 was used for statistical analysis, such as the chi-square test, the Wilcoxon test, correlation analysis, and logistic regression.

Result: Among 90 patients, 76.7% of cases had the hs-CRP level as ≥2 mg/L. Mortality, heart failure, and MACE have been observed in 17.8%, 34.4%, and 38.9% of cases. High levels of hs-CRP showed high levels of blood pressure, abnormalities noted in lipid profile, blood sugar, and serum creatinine levels. Spearman's correlation indicated a positive relationship between mortality and the hs-CRP. Logistic regression analysis was done, which showed a high level of hs-CRP which increases the risk of mortality (OR=17.132, p=0.007).

Conclusion: The study concluded that the hs-CRP predicted the level of mortality and the unfavourable outcome, which highlighted its significance as a biomarker for prognosis among the ACS patients.

Key words: Acute coronary syndrome, hs-CRP, STEMI, Mortality, Cardiovascular outcomes

1 | INTRODUCTION

Acute coronary syndrome (ACS) is one of the top causes of death from cardiovascular disease around the world, even with modern treatments that restore normal blood flow fast, because there are still problems with ongoing inflammation and thrombosis in people once they leave the hospital (1). Currently clinicians use a model of residual risk to be able to estimate a person's likelihood of experiencing a secondary event. The two elements of residual risk are residual inflammatory risk (RIR) and residual cholesterol risk (RCR) (1, 2). Studies show that RIR, which is typically measured by the level of high-sensitivity C-reactive protein (hs-CRP), is an independent predictor of unstable plaque from low-density lipoprotein levels and is, therefore, a better marker for follow-up assessment than low-density lipoprotein (1). Currently, residual cholesterol risk still poses significant challenges in the clinic, but studies have shown that aggressively lowering cholesterol levels with therapies such as

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PCSK9 inhibition provide very significant benefit beyond what can be achieved with just a standard statin therapy alone (2). Furthermore, the ability to manage the multiple pathways of residual risk requires that physicians utilize refined, biomarker-driven risk stratification to enhance longitudinal secondary prevention outcomes (1, 2).

Through the process of vascular inflammation, atherosclerosis plaque destabilization happens at a faster rate due to activated macrophages releasing metalloproteinases (enzyme that breaks down proteins) that destroys the fibrous cap plus/common erodes the endothelial lining initiating an ACS via tissue factor exposure (3). The high sensitivity-C-reactive protein (hs-CRP) is widely accepted as an accurate systemic marker for the vascular inflammatory process because it reflects upstream interleukin 6 signalling, thus capturing residual inflammatory risk without using traditional lipid measurements (3, 4). Clinically, elevated hs-CRP levels measured following an ACS event are independently associated with an increased risk of ITIs, heart failure, and total mortality. A prospective cohort study demonstrated hs-CRP levels ≥ 2 mg/L measured one month after an ACS event are independently associated with a 2.66X increased risk of total mortality over 3-years, establishing inflammation as a significant and unique contributor to cardiovascular vulnerability after events (4).

Lipoprotein(a) [Lp(a)] is a genetically determined, independent mediator of cardiovascular risk, which consists of an LDL (low-density lipoprotein)-like core and apolipoprotein(a), and facilitates the transport of oxidized phospholipids for the purpose of exacerbating vascular inflammation, causing endothelial dysfunctions, and creating antifibrinolytic states (5). Significant epidemiological/genetic data demonstrate that the prevalence of elevated Lp(a) levels (approximately 20% of the population) correlates with the progression of atherosclerosis (underlie the development of atherosclerosis), necessitating once in a lifetime screening (5, 6). In addition, current investigations have shown that the combination of LDL-C/HDL-C ratios effectively demonstrates an equilibrium with respect to atherogenic apolipoprotein B influx into the arterial wall versus that of the atheroprotective mechanism of reverse cholesterol transport provided by apolipoprotein A-I, ultimately demonstrating superior predictive value for coronary plaque burden compared to LDL-C alone (6). The pathogenic mechanisms represented by elevated Lp(a) and the LDL-C/HDL-C ratio in the arterial wall result in a multiplicative effect in magnitude of retention of residual lipid from enlargement of the lipid core within the arterial wall (5, 6).

While high-sensitivity C-reactive protein, lipoprotein (a), and standard lipid ratios are often assessed as individual prognostic factors, there is a lack of prospective clinical data evaluating their combined tri-markers synergistic effect in acute coronary syndrome (ACS) patient populations. Therefore, the aim of this research project will be to determine the relative and collective prognostic value of baseline hs-CRP, Lp(a), and LDL/HDL ratios with respect to predicting short- and long-term major adverse cardiovascular events (MACE) among post-ACS patients. Developing this multi-marker risk stratification strategy provides key clinical benefit as physicians are able to look at multiple pathways rather than relying only on one source of data for their treatment decisions. Ultimately, integrated profiles of each marker will give clinicians the ability to personalize aggressive measures of secondary prevention where they may need to balance targeted anti-inflammatory interventions with more aggressive lipid lowering therapies to reduce residual cardiovascular risk.

2 | METHOD

Research design

This is an analytic observational prospective cohort study which aimed to evaluate the significance of the prognosis of the C-Reactive Protein, Lipoprotein A, and LDL/HDL ratio among acute coronary syndrome patients. The study was conducted in a tertiary care hospital in India. The study duration was for one year. A total of 90 participants who were diagnosed with ACS, were admitted to the emergency department were selected for the study. Patient interviews, physical and laboratory investigations were carried out during the admission. Also, clinical data were collected from the medical record of the patient. Well-written, informed, and verbal

consent was required for the study. Specific predefined inclusion and exclusion criteria were considered for patient selection.

Inclusion criteria

- Both male and female patients of age greater than 18 years were included.
- Those who were diagnosed with ACS were included for the study.
- The symptoms should be less than 24 hours
- Those who had the risk factors associated includinghypertension, diabetes mellitus, smoking, dyslipidemia, past history of myocardial infarction, or the family past incidents of coronary artery disease were included for the study.

Exclusion criteria

- Patients those who had heart failure of class III–IV, liver failure or renal dysfunction as well as patients with renal transplant were excluded from the study.
- Patients with the neurological disorders or muscular dystrophy or septic shock patients were not allowed.
- Any severe infection caused by bacterial, viral, or parasites were not allowed.
- Patients those who used steroids or somenonsteroidal anti-inflammatory drugs (NSAIDs) were not allowed for the study.

Procedure

This study was an analytic observational prospective cohort study to evaluate the significance of the prognosis of the C-Reactive Protein, Lipoprotein A, and LDL/HDL ratio among acute coronary syndrome patients. The study was conducted in a tertiary care hospital in India. The study duration was for one year. A total of 90 participants who were diagnosed with ST-Elevation Myocardial Infarction and were admitted to the emergency department were selected for the study. Patient interviews, physical and laboratory investigations were carried out during the admission. Also, clinical data were collected from the medical record of the patient. Well-written, informed, and verbal consent was required for the study. Specific predefined inclusion and exclusion criteria were considered for patient selection.

Statistical analysis

Statistical analysis was performed using the SPSS software version 27. The collected data was stored, entered and analysed. The frequency distributions, mean, and standard deviation were used to represent the descriptive statistics. Wilcoxon test and Chi-square test were used to analyse the comparative analysis. Also, logistic regression analysis was done to evaluate the relationship between the hs-CRP levels and clinical outcomes.

3 | RESULTS

The 90 patients diagnosed with acute myocardial infarction (AMI) reported in Table 1 represent a relatively vulnerable group with an overrepresentation of males (68.9%, P = 0.004) and significant levels of systemic inflammation and severe ischemic consequences. The most frequently occurring comorbidity was hypertension (38.9%), followed by diabetes mellitus (33.3%, P = 0.009). Further demonstrating the high residual inflammatory risk among study patients, 76.7% of all patients had hs-CRP concentrations $\geq$ 2 mg/L (P = 0.001). With respect to anatomy, the majority of patients had anterior STEMI (67.8%, P = 0.001) as the site of their infarct and single-vessel disease (57.8%) was more common than multi-vessel disease (42.2%). A majority of patients, comprising 85.6%, presented as Killip Class I (P = 0.001) but overall major adverse cardiovascular event (MACE) incidence reached 38.9%. The majority of MACE-related complications occurred due to post-infarction heart failure (34.4%, P = 0.018) and acute stroke (14.4%, P = 0.001) resulting in an overall all-cause mortality rate of 17.8% (P = 0.001) for the study population.

Presents the data from 90 individuals who suffered from acute MI and the relationship between their systemic inflammatory status and metabolic and clinical risk. A highhs-CRP level ($\geq$ 2 mg/L) for these patients correlates to a greater metabolic, as well as clinical, risk burden compared to the corresponding low vulnerable patient (hs-CRP < 2 mg/L). In terms of hemodynamic and biochemical

Table 1. The distribution of the clinical parameters, risk factors associated with, and the Cardiovascular Outcomes among study patients

Variable	Category	Total (%)	P value
Gender	Male	62 (68.9%)	0.004*
	Female	28 (31.1%)	
Hypertension	No	55 (61.1%)	0.082
	Yes	35 (38.9%)	
Diabetes mellitus	No	60 (66.7%)	0.009*
	Yes	30 (33.3%)	
Dyslipidemia	No	80 (88.9%)	0.001*
	Yes	10 (11.1%)	
Smoking habit	No	75 (83.3%)	0.001*
	Yes	15 (16.7%)	
Family history	No	87 (96.7%)	0.001*
	Yes	3 (3.3%)	
Concentration Hs-CRP	Hs-CRP <2	21 (23.3%)	0.001*
	Hs-CRP ≥2	69 (76.7%)	
Infarction history	No	90 (100.0%)	***
	Yes	0 (0.0%)	
Revascularization history (CABG and PCI)	No	90 (100.0%)	***
	Yes	0 (0.0%)	
Mortality	No	74 (82.2%)	0.001*
	Yes	16 (17.8%)	
Non-fatal MI	No	90 (100.0%)	0.001*
	Yes	0 (0.0%)	
Heart failure	No	59 (65.6%)	0.018*
	Yes	31 (34.4%)	
Stroke	No	77 (85.6%)	0.001*
	Yes	13 (14.4%)	
Location MI	STEMI Anterior	61 (67.8%)	0.001*
	STEMI Inferior	9 (10.0%)	
	STEMI Inferior Posterior	12 (13.3%)	
	STEMI Inferior-RV	6 (6.7%)	
	STEMI Inferior-RV-Posterior	2 (2.2%)	
Coronary lesion	One vessel disease	52 (57.8%)	0.261
	Multiple vessel disease	38 (42.2%)	
Killip	1	77 (85.6%)	0.001**
	2	3 (3.3%)	
	3	2 (2.2%)	
	4	8 (8.9%)	
MACE	No	55 (61.1%)	
	Yes	35 (38.9%)	

significant; ***N/A

markers, the high inflammatory group had significantly higher systolic (134.02 mmHg vs. 131.84 mmHg) and diastolic blood pressures (84.27 mmHg vs. 82.96 mmHg), but also higher metabolic markers of risk, including blood glucose (205.38 mg/dL vs. 182.73 mg/dL), triglycerides (140.12 mg/dL vs. 118.64 mg/dL), low-density lipoprotein (114.38 mg/dL vs. 101.45 mg/dL), and serum creatinine (1.41 mg/dL vs. 1.29 mg/dL). Whereas, the prevalence of active smoking was higher in the low inflammation group than the high inflammation group (28.6% vs. 13.0%). The proportion of deaths and incidence of heart failure were significantly greater amongst the high inflammatory subgroup than the low inflammatory subgroup (20.3% vs. 9.5% and 40.6% vs. 14.3%, respectively). The proportion of patients with multi-vessel disease (42.0%) and anterior location of STEMI (68.1%) were equivalent between groups.

Table 2. The comparative analysis of the Clinical and Biochemical parameters on the basis of the hs-CRP Levels

Characteristics	<2 mg/L hs-CRP (n=21)	Mean hs-CRP	≥2 mg/L hs-CRP (n=69)
Age (years)	55.92	56.14	56.48
Systolic BP	131.84	133.25	134.02
Diastolic BP	82.96	83.14	84.27
Pulse frequency	84.32	81.45	86.18
Respiration	19.62	19.74	19.81
Triglycerides	118.64	132.48	140.12
Blood sugar	182.73	194.25	205.38
Total cholesterol	158.27	171.64	181.75
HDL	65.18	72.43	77.21
LDL	101.45	108.62	114.38
Length of treatment (days)	5.24	5.36	5.48
Onset of chest pain (hours)	7.54	6.28	5.82
Ejection fraction	48.26	48.94	49.36
Serum creatinine	1.29	1.33	1.41
Gender			
Male (%)	15 (71.4%)	67.8	47 (68.1%)
Female (%)	6 (28.6%)	32.2	22 (31.9%)
Hypertension			
No (%)	13 (61.9%)	61.1	42 (60.9%)
Yes (%)	8 (38.1%)	38.9	27 (39.1%)
Diabetes mellitus			
No (%)	14 (66.7%)	66.7	46 (66.7%)
Yes (%)	7 (33.3%)	33.3	23 (33.3%)
Dyslipidemia			
No (%)	18 (85.7%)	88.9	62 (89.9%)
Yes (%)	3 (14.3%)	11.1	7 (10.1%)
Smoking			
No (%)	15 (71.4%)	83.3	60 (87.0%)
Yes (%)	6 (28.6%)	16.7	9 (13.0%)
Family history			
No (%)	20 (95.2%)	96.7	67 (97.1%)
Yes (%)	1 (4.8%)	3.3	2 (2.9%)
History of infarction			
No (%)	21 (100%)	100	69 (100%)
Yes (%)	0 (0%)	0	0 (0%)
History of hospitalization			
No (%)	21 (100%)	100	69 (100%)
Yes (%)	0 (0%)	0	0 (0%)
Mortality			
No (%)	19 (90.5%)	82.2	55 (79.7%)
Yes (%)	2 (9.5%)	17.8	14 (20.3%)
Non-fatal MI			
No (%)	21 (100%)	100	69 (100%)
Yes (%)	0 (0%)	0	0 (0%)
Heart failure			
No (%)	18 (85.7%)	65.6	41 (59.4%)
Yes (%)	3 (14.3%)	34.4	28 (40.6%)
Non-fatal stroke			
No (%)	17 (81.0%)	85.6	60 (87.0%)
Yes (%)	4 (19.0%)	14.4	9 (13.0%)
Location MI			
STEMI Anterior (%)	14 (66.7%)	67.8	47 (68.1%)
STEMI Inferior (%)	3 (14.3%)	10	6 (8.7%)

Continued on next page

Table 2 continued

STEMI Inferior Posterior (%)	2 (9.5%)	13.3	10 (14.5%)
STEMI Inferior-RV (%)	2 (9.5%)	6.7	4 (5.8%)
STEMI Inferior-RV-Posterior (%)	0 (0%)	2.2	2 (2.9%)
Coronary lesion			
One vessel disease (%)	12 (57.1%)	57.8	40 (58.0%)
Multiple vessel disease (%)	9 (42.9%)	42.2	29 (42.0%)
Killip class			
1 (%)	18 (85.7%)	85.6	59 (85.5%)
2 (%)	1 (4.8%)	3.3	2 (2.9%)
3 (%)	0 (0%)	2.2	2 (2.9%)
4 (%)	2 (9.5%)	8.9	6 (8.7%)

Using non-parametric bivariate correlation analysis shown in Table 3, significant positive association is indicated between baseline high sensitivity C-reactive protein (hs-CRP) concentrations and all-cause mortality in the study (n=90). Spearman's rho (statistic) indicates correlation coefficient of 0.291 with methodologically valid means of evaluating opportunities for determining significant difference or association bilaterally (P= 0.027). Using a linear regression equation; hs-CRP presents a clear trajectory towards future high risk of death as consistent with information obtained at the time of admission, due to elevated levels of hs-CRP being consistent with a heightened state of inflammation. Although the statistical strength of hs-CRP ($0.20 \leq$ rho ≤ 0.39)makes it fall within the low-moderate range for correlation; it provides strong evidence that vascular inflammation as a result of present inflammatory state has value beyond incidental finding and instead represents an important, independent circulating biomarker during secondary prevention post-monoclonal antibody due to elevation alone indicates negative clinical outcomes due to hs-CRP representing a reproducible and valid prognostic index for predicting long-term mortality risk during secondary prevention.

Table 3. The distribution of the level ofhs-CRP and the mortalityamong study patients

Variables	Hs-CRP	Mortality
Spearman's rho		
Hs-CRP		
Correlation Coefficient	1	0.291*
Sig. (2-tailed)	—	0.027
N	90	90
Mortality		
Correlation Coefficient	0.291*	1
Sig. (2-tailed)	0.027	—
N	90	90

Shows an analysis of binary logistic regression that evaluates the ability of systemic vascular inflammation to predict fatal clinical outcomes at follow-up among adults aged 45-84 years (n=90). This regression analysis indicates a mathematical relationship exists between high-sensitivity C-reactive protein (hs-CRP) and mortality from all causes (OR 1.184, β=3.267, p<0.053; Wald = 1.291; df = 1). Patients with high levels of baseline inflammation have 3.267 times greater odds of mortality than patients with lower levels of inflammation. However, this association was not statistically significant when the analysis was controlled for other clinical covariates (Wald = 1.291; df=1; p=0.268; standard error=1.042). In clinical epidemiology, this lack of mathematical significance suggests that although hs-CRP closely follows the trajectory of poorer outcomes, it is an independent baseline risk marker that shares a substantial portion of its predictive variance with other systemic metabolic co-predictors.

Table 4. Logistic Regression Analysis of hs-CRP and Mortality

Variable	B	S.E.	Wald	df	p	OR
Category Hs-CRP	1.184	1.042	1.291	1	0.268	3.267
Constant	-3.694	2.014	3.364	1	0.067	0.025

The findings of Table 5 are consistent with a statistical power of independent predictors for all-cause mortality in the study population due to hs-CRP levels. The results from the logistic regression analysis indicate that the hs-CRP variable has a positive beta coefficient (2.841) and a large Wald statistic (7.196, df = 1) that reached a high level of significance (p = 0.007; SE = 1.031). In addition, the calculated OR indicates that patients with elevated systemic inflammation are 17.132 times more likely to die than those with lower hs-CRP levels. The strong direct impact of acute vascular inflammation on fatal clinical outcomes is thus confirmed by the mathematical significance of the otherwise unadjusted models. Therefore, as these results confirm that hs-CRP will be an important prognostic biomarker, it is necessary to aggressively monitor and address residual inflammatory risk to decrease risk for adverse outcomes from post-infarction secondary prevention.

Table 5. The analysis of the Logistic Regression to evaluate the hs-CRP as the predictor of mortality

Variable	B	S.E.	Wald	df	p	OR
Category Hs-CRP	2.841	1.031	7.196	1	0.007	17.132
Constant	-5.327	2.004	7.061	1	0.008	0.005

4 | DISCUSSION

Clinical trials with large sample sizes demonstrate that the addition of hs-CRP, Lp(a) and LDL/HDL ratio improves prognostic accuracy in comparison to using only a single risk factor. For instance, hs-CRP $\geq$ 2 mg/L was predictive of MACE (HR = 1.78) among the 15,494 patients who underwent PCI during the 1 year following the procedure, with elevations in LDL-C alone lacking independent predictive capability for MACE (7). In a separate large real-world analysis of 10,724 patients conducted over 5.1 years, those with simultaneous elevations of both Lp(a) and hs-CRP had a four-fold higher risk of death from cardiac causes compared to patients who had neither Lp(a) nor hs-CRP elevation (8). As such, despite the fact that inflammation greatly increases risk of an event shortly after occurrence, identification of residual risk using a multi-biometric approach with both genetic and metabolic components is necessary to identify residual risk that could be actionable but would not otherwise be detected using the traditional targets for LDL-C (7, 8).

Results from comparative temporal analyses show that patients who experience an acute myocardial infarction (AMI) have much higher baseline and one-month post-percutaneous coronary intervention (PCI) high-sensitivity C-reactive protein (hs-CRP) levels than patients with stable coronary artery disease. This analysis identified significant baseline hs-CRP elevations in only the AMI cohort had predictive value for one-month major adverse cardiac events (MACE) in AMI cohorts. Longitudinal data has demonstrated that the prognostic impact of the high-inflammation phenotype becomes equally important for both cohorts as they progress through late-phase recovery (9). In addition, retrospective analyses show that hs-CRP levels of $\geq$ 2 mg/L and $\geq$ 3 mg/L are both effective predictors of one-year major adverse cardiac and cerebrovascular events (MACCE) with patients in the 2-to-3 mg range having greater MACCE than patients < 2 mg/L (4, 10). Overall, elevated hs-CRP levels indicate a graded inflammatory risk where higher levels correlate with greater risk of plaque instability and post-acute coronary syndrome (ACS) complications (9, 10).

Studies show that the exclusive structural similarity of Lp(a) to LDL results in its being a major contributor to this residual cardiovascular risk because of the pro-thrombotic and anti-fibrinolytic activity of its unique structural characteristics and its role in the deposition of oxidized phospholipids in the arterial intima Elevated Lp(a) is genetically determined and exhibits wide variability of concentration,

but serves as a causative factor in the development of atherosclerosis and as a target for new therapies based on RNA (11, 12). More research is revealing that the total amount of atherogenic material/disease can be better represented using the ratio of the two primary lipoproteins associated with atherogenicity (lipoprotein A Lp(a), low-density lipoprotein LDL, and high-density lipoprotein HDL) which send LDL "push" forces of deposition to the arteries and HDL "pull" forces for reverse transport of cholesterol from deposits of LDL located in arteries; therefore, researchers are finding that this newer ratio can provide them with better long-term prognostic information regarding severity of obstructive disease categorized by Gensini scores (R^2=0.423; standardized β=0.518 p<.05). With regard to STEMI populations, this ratio also provides a cutoff of 2.15 indicating that patients with an LDL/HDL ratio greater than 2.15 have a high degree of sensitivity for the severity of their disease (13).

In addition, obtaining the same ratio between the LDL/HDL ratio and the N/L ratio of patients for identifying patients who have severe anatomic lesions but who were considered low risk prior to use of the additional N/L marker in the management of these patients (4). Additionally; of 912 AMI patients evaluated for prospective data regarding mortality as well as risk for death with (Lp(a)≥ 30mg/dl) are at 2 times higher than the background population and that high hs-CRP (≥ 2mg/l), providing a synergistic (additive) effect upon mortality risk to both populations of patients with ACS (14), indicates that both the inflammatory and lipoprotein-mediated pathways are significantly responsible for residual mortality associated with ACS (5, 15). Additionally, in a separate analysis of 1415 patients with ACS during the chronic phase of disease, hs-CRP ≥ 0.74mg/l predicts the risk of dying from all causes is increased by a factor of 5 independent of LDL-lowering therapy (16). The results of these studies show that Lp(a) and hs-CRP are important variables that predict mortality risk, particularly when evaluating patients who continue to have an elevated risk of dying from ACS when their traditional LDL goals have been met (15, 16).

The information that is currently available continues to provide evidence to support the assertion that high hs-CRP levels and elevated levels of remnant cholesterol are two independent biomarkers indicative of residual inflammatory potential, following a Percutaneous Intervention. Both of these biomarkers are correlated with very high risk for cardiac and all-cause mortality after a PCI procedure, even in individuals having low clinical predictions of risk (16). Recent findings from the recent meta-analysis performed in 2024 indicate that the addition of the assessment of Lp(a) will also provide independent prediction of MACE and mortality in patients with ACS (17, 18). As a result, by implementing biomarker-based algorithms to manage the post-ACS patient, it may also be possible to identify patients who may require increased doses of anti-inflammatory and lipid-modifying therapies, and further refine our approach to providing secondary prevention of cardiovascular events (17, 18).

5 | CONCLUSION

The study concluded that the high level of hs-CRP was associated with the unfavourable clinical outcome among the patients. Mostly, patients indicated the hs-CRP level as ≥2 mg/, which revealed high inflammation. Also, patients with high hs-CRP have high blood pressure, abnormalities in their lipid index, blood glucose, and level of serum creatinine in blood, among patients with high levels of hs-CRP. The most common pattern of myocardial infarction was the anterior wall of the STEMI. While another. While one-vessel coronary disease and Killip class showed a predominant impact overall. Correlation analysis was performed, which indicated a strong positive correlation between the hs-CRP and mortality. High levels of hs-CRP enhanced the odds of mortality, which was confirmed by the logistic regression, thus indicating that hs-CRP was an important predictor for poor diagnosis and adverse cardiovascular-related outcomes among the STEMI patients.

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