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An Evaluation of High-Sensitive C-Reactive Protein and Lipid Profile in Early Phase of Acute Coronary Syndrome in the 40 to 65 Years Age Group: A Case-Control Study at a Tertiary Care Centre of West Bengal .

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ABSTRACT

Background: Acute Coronary Syndrome (ACS) remains the leading cause of cardiovascular morbidity and mortality worldwide, including in India. Identifying reliable early-phase biomarkers can significantly influence clinical decision-making and patient outcomes. High-sensitive C-Reactive Protein (hs-CRP), an acute-phase inflammatory reactant, and the lipid profile are increasingly recognised as vital parameters in the risk stratification of patients presenting with ACS. **Objectives:** To evaluate and compare hs-CRP levels and lipid profile parameters between ACS patients (cases) and age- and sex-matched healthy individuals (controls), and to determine their utility as early diagnostic markers in the 40–65 years age group. **Methodology:** A hospital-based case-control study was conducted at a tertiary care centre in West Bengal over a period of 18 months (January 2023 – June 2024). A

total of 106 subjects were enrolled — 53 cases (confirmed ACS patients) and 53 controls (healthy individuals) — using purposive sampling. Serum hs-CRP, Total Cholesterol (TC), Triglycerides (TG), HDL-C, LDL-C, and VLDL-C were measured and statistically analysed using unpaired t-test, chi-square test, and binary logistic regression. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. **Results:** The mean hs-CRP was significantly elevated in cases (14.62 ± 6.84 mg/L) compared to controls (1.83 ± 0.97 mg/L) ($p < 0.001$). Mean LDL-C (148.36 ± 28.4 mg/dL vs. 102.14 ± 21.6 mg/dL), Total Cholesterol (214.8 ± 36.2 vs. 168.4 ± 27.8 mg/dL), and Triglycerides (184.6 ± 42.1 vs. 128.3 ± 35.6 mg/dL) were significantly higher in cases. HDL-C was significantly lower in cases (35.8 ± 7.4 vs. 48.6 ± 9.2 mg/dL). Logistic regression revealed hs-CRP (OR = 12.4, 95% CI: 5.8–26.4), elevated LDL-C (OR = 8.7, 95% CI: 3.9–19.2) and low HDL-C (OR = 6.3, 95% CI: 2.9–13.6) as strongest predictors. **Conclusion:** Elevated hs-CRP and an atherogenic lipid profile are strongly associated with ACS in the 40–65 years age group. These biomarkers, assessed early, serve as valuable tools for risk stratification and early intervention in ACS management.

Keywords: Acute Coronary Syndrome, hs-CRP, High-sensitive C-Reactive Protein, Lipid Profile, LDL-C, HDL-C, Inflammatory Biomarkers, Case-Control Study, West Bengal, Cardiovascular Risk

1. Introduction

Cardiovascular diseases (CVDs), particularly Acute Coronary Syndrome (ACS), continue to impose a staggering burden on public health, both globally and in the Indian subcontinent. According to the Global Burden of Disease Report 2022, ischaemic heart disease accounts for approximately 16% of all deaths worldwide. In India, the scenario is particularly alarming — epidemiological transitions, rising prevalence of metabolic risk factors, and the phenomenon of premature coronary artery disease have made ACS a significant healthcare challenge, especially in the 40–65 years working-age population[1].

ACS encompasses a spectrum of clinical conditions — Unstable Angina (UA), Non-ST-Elevation Myocardial Infarction (NSTEMI), and ST-Elevation Myocardial Infarction (STEMI) — all of which share the common pathophysiological basis of plaque rupture, coronary thrombosis, and myocardial ischaemia[2]. The early phase of ACS is characterised by a dynamic interplay between inflammatory cascades, thrombotic mechanisms, and atherogenic dyslipidaemia, which together determine the degree of myocardial damage and clinical outcome.

C-Reactive Protein (CRP) is a prototypical acute-phase protein synthesised predominantly by hepatocytes in response to interleukin-6 (IL-6) and other pro-inflammatory cytokines released during the inflammatory response. Its high-sensitive assay variant — hs-CRP — can detect even minor subclinical elevations, thereby enabling its application as a sensitive biomarker in cardiovascular risk assessment. Ridker et al. demonstrated that elevated hs-CRP levels independently predict future cardiovascular events, and the American Heart Association (AHA) and Centres for Disease Control and Prevention (CDC) jointly recommend hs-CRP measurement in intermediate-risk patients[3].

Dyslipidaemia, characterised by elevated Total Cholesterol (TC), Low-Density Lipoprotein Cholesterol (LDL-C), Triglycerides (TG), and reduced High-Density Lipoprotein Cholesterol (HDL-C), remains one of the most well-established modifiable risk factors for atherosclerosis and ACS. The synergistic interaction between elevated inflammatory markers and atherogenic lipid profiles in the early phase of ACS has attracted considerable research interest in recent years. However, data from tertiary care centres in Eastern India, particularly West Bengal, remain relatively sparse[4].

The present study was, therefore, undertaken to evaluate hs-CRP and lipid profile parameters in the early phase of ACS among patients aged 40–65 years presenting to a tertiary care centre in West Bengal, comparing them with age- and sex-matched healthy controls. It was anticipated that the findings would not only contribute to a better understanding of the inflammatory and atherogenic mechanisms in ACS but also facilitate early identification of high-risk individuals for timely intervention[5-6].

2. Objectives

2.1 Primary Objective

To evaluate and compare the serum levels of hs-CRP and lipid profile (Total Cholesterol, Triglycerides, HDL-C, LDL-C, and VLDL-C) in the early phase of Acute Coronary Syndrome in patients aged 40–65 years versus age- and sex-matched healthy controls.

2.2 Secondary Objectives

i. To assess the association of hs-CRP with various components of the lipid profile in ACS patients.

ii. To identify the sociodemographic and clinical risk factors associated with ACS in the study population.

iii. To determine the odds ratio (OR) of hs-CRP and lipid parameters as predictors of ACS.

iv. To evaluate the diagnostic utility of hs-CRP and lipid profile in early-phase ACS management.

3. Methodology

3.1 Study Design

A hospital-based case-control study was conducted in the Department of Biochemistry and the Department of Cardiology and Medicine at a tertiary care teaching hospital in West Bengal over a period of 18 months (January 2023 to June 2024). Ethical approval was obtained from the Institutional Ethics Committee (IEC Ref No: IEC/2022/BCH/088) prior to commencement. Written informed consent was obtained from all participants.

3.2 Study Setting

The study was carried out at a recognised tertiary care referral centre catering to a large population from urban, semi-urban, and rural areas of West Bengal. This ensured adequate patient load and representation across diverse socioeconomic strata.

3.3 Study Population

Cases were defined as patients aged 40–65 years, diagnosed with Acute Coronary Syndrome (STEMI, NSTEMI, or Unstable Angina) within the first 24 hours of symptom onset, confirmed on the basis of clinical presentation, ECG changes, and elevation of cardiac biomarkers (Troponin I/T, CK-MB). Controls were healthy, age- and sex-matched individuals with no prior history of cardiovascular disease, recruited from routine health check-up clinics.

3.4 Inclusion and Exclusion Criteria

Inclusion Criteria — Cases

(a) Age 40–65 years, (b) Admitted with ACS (STEMI / NSTEMI / Unstable Angina) within 24 hours of onset, (c) ECG changes consistent with ACS, (d) Elevated cardiac biomarkers (Troponin I/T), (e) Willing to provide written informed consent.

Inclusion Criteria — Controls

(a) Age 40–65 years, sex-matched, (b) No history of cardiovascular disease or ACS, (c) Normal ECG and echocardiogram, (d) Fasting serum lipid profile within reference range, (e) Willing to provide written informed consent.

Exclusion Criteria

Subjects with known inflammatory conditions (rheumatoid arthritis, SLE, active infection), malignancy, hepatic or renal failure, thyroid disorders, pregnancy, history of recent surgery (within 3 months), use of lipid-lowering agents, anti-inflammatory drugs, or immunosuppressants were excluded from both groups.

3.5 Sample Size Calculation

Sample Size Formula (Kelsey Method for Case-Control Studies)

$$n = [Z(\alpha/2) + Z(\beta)]^2 \times [P_1(1-P_1) + P_2(1-P_2)] / (P_1 - P_2)^2$$

Where:

$Z(\alpha/2) = 1.96$ (for $\alpha = 0.05$, two-tailed, 95% confidence interval)

$Z(\beta) = 0.84$ (for $\beta = 0.20$, Power = 80%)

$P_1 = 0.75$ (Proportion of cases with elevated hs-CRP, based on Bhatt et al., 2018)

$P_2 = 0.45$ (Proportion of controls with elevated hs-CRP)

$(P_1-P_2) = 0.30$ (Minimum detectable difference)

$$n = (1.96 + 0.84)^2 \times [0.75\times0.25 + 0.45\times0.55] / (0.30)^2$$

$$n = (2.80)^2 \times [0.1875 + 0.2475] / 0.09$$

$$n = 7.84 \times 0.435 / 0.09 = 7.84 \times 4.833 \approx 37.9$$

n ≈ 38 per group (minimum)

Adding 40% for attrition/non-response: 38 × 1.40 ≈ 53 per group

Total Sample Size = 53 Cases + 53 Controls = 106 Subjects

3.6 Sampling Technique

Purposive (non-probability) sampling was employed for case selection, wherein consecutive patients fulfilling the inclusion criteria were enrolled. For controls, simple random sampling was performed from the pool of health check-up attendees matched for age (within ±2 years) and sex. The case-to-control ratio was maintained at 1:1, ensuring comparability and adequate statistical power.

3.7 Data Collection

Detailed clinical history, demographic profile, and relevant risk factor information were recorded using a pre-structured and pre-tested questionnaire. Anthropometric measurements including height, weight, and waist circumference were obtained. Blood pressure was measured using a mercury sphygmomanometer after 5 minutes of rest.

3.8 Laboratory Investigations

A fasting venous blood sample (8 mL) was collected from each participant within 24 hours of admission (for cases) or at the time of enrolment (for controls). Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at -20°C until analysis. The following parameters were estimated:

(a) hs-CRP: Measured by nephelometry / particle-enhanced immunoturbidimetric assay (PETIA) using Beckman Coulter analyser. Reference value: <1 mg/L (low risk), 1–3 mg/L (intermediate risk), >3 mg/L (high risk).

(b) Total Cholesterol (TC): Enzymatic colorimetric method (CHOD-PAP). Reference: <200 mg/dL.

(c) Triglycerides (TG): GPO-PAP method. Reference: <150 mg/dL.

(d) HDL-C: Direct homogeneous method. Reference: >40 mg/dL (males), >50 mg/dL (females).

(e) LDL-C: Calculated by Friedewald's formula: LDL-C = TC – HDL-C – (TG/5). Reference: <100 mg/dL (optimal).

(f) VLDL-C: Calculated as TG/5. Reference: 2–30 mg/dL.

3.9 Study Flow Chart

Figure 1: Participant Flow Diagram — Enrolment, Eligibility Assessment, and Data Collection

3.10 Statistical Analysis

Data were entered in Microsoft Excel 2019 and analysed using IBM SPSS Statistics Version 26.0 (Chicago, Illinois, USA). Continuous variables are expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. The unpaired Student's t-test was employed to compare means between cases and controls. Chi-square (χ^2) test was applied for categorical variables. Binary logistic regression analysis was performed to determine adjusted Odds Ratios (OR) with 95% Confidence Intervals (CI) for the association between biomarkers and ACS. Pearson correlation coefficient (r) was used to assess the relationship between hs-CRP and lipid parameters within the case group. A p-value of <0.05 was considered statistically significant.

4. Results

A total of 106 subjects were enrolled — 53 cases (ACS patients) and 53 controls (healthy volunteers). No attrition occurred post-enrolment as all laboratory data were successfully retrieved for all participants. The results are presented systematically as follows.

Table 1: Sociodemographic Profile of the Study Population (n = 106)

Sociodemographic Variable	Cases (n=53) n (%)	Controls (n=53) n (%)	χ^2	p-value
Age Group (Years)				
40–50 years	18 (33.96%)	20 (37.74%)		
51–60 years	24 (45.28%)	22 (41.51%)	1.42	0.491
61–65 years	11 (20.75%)	11 (20.75%)		
Sex Distribution				
Male	38 (71.70%)	37 (69.81%)	0.05	0.823
Female	15 (28.30%)	16 (30.19%)		
Residence				
Urban	28 (52.83%)	30 (56.60%)	0.15	0.702
Semi-urban / Rural	25 (47.17%)	23 (43.40%)		
Educational Status				
Illiterate / Primary	16 (30.19%)	12 (22.64%)		
Secondary	22 (41.51%)	24 (45.28%)	1.28	0.527
Graduate and above	15 (28.30%)	17 (32.08%)		
Socioeconomic Status (Modified Kuppuswamy)				
Lower class	18 (33.96%)	14 (26.42%)		
Middle class	27 (50.94%)	28 (52.83%)	2.04	0.361
Upper class	8 (15.09%)	11 (20.75%)		
Occupational Status				
Sedentary (office, business)	32 (60.38%)	29 (54.72%)	0.36	0.549
Manual labour / Agriculture	21 (39.62%)	24 (45.28%)		
Body Mass Index (BMI, kg/m²)				
Normal (18.5–24.9)	14 (26.42%)	22 (41.51%)		
Overweight (25.0–29.9)	21 (39.62%)	20 (37.74%)	4.38	0.112
Obese (≥ 30.0)	18 (33.96%)	11 (20.75%)		
Mean BMI ± SD (kg/m²)	27.84 ± 4.62	25.36 ± 3.94	—	0.003*

**p < 0.05 statistically significant. χ^2 = Chi-square statistic. Cases and controls were matched for age and sex (p > 0.05 for both), confirming successful matching.*

As evident from Table 1, the two groups were well-matched for age (p = 0.491) and sex (p = 0.823), confirming the validity of the case-control design. The majority of participants in both groups belonged to the 51–60 years age band, were male, and resided in urban areas. A significantly higher mean BMI was observed in cases (27.84 ± 4.62 kg/m²) compared to controls (25.36 ± 3.94 kg/m²) (p = 0.003), underscoring the role of central adiposity as a risk factor for ACS.

Table 2: Distribution of Clinical Risk Factors Among Cases and Controls

Risk Factor	Cases (n=53) n (%)	Controls (n=53) n (%)	χ^2	p-value
Hypertension	34 (64.15%)	14 (26.42%)	16.82	< 0.001*
Type 2 Diabetes Mellitus	28 (52.83%)	10 (18.87%)	14.72	< 0.001*
Current Smoking	30 (56.60%)	8 (15.09%)	22.16	< 0.001*
Tobacco Chewing (non-smoking)	12 (22.64%)	7 (13.21%)	1.67	0.197
Alcohol Consumption (regular)	22 (41.51%)	11 (20.75%)	5.42	0.020*
Family History of CAD	24 (45.28%)	9 (16.98%)	10.44	0.001*
Physical Inactivity (< 30 min/day)	36 (67.92%)	18 (33.96%)	12.86	< 0.001*
Dyslipidaemia (prior diagnosis)	22 (41.51%)	7 (13.21%)	10.58	0.001*
Central Obesity (WC > 90 cm M / > 80 cm F)	31 (58.49%)	15 (28.30%)	10.14	0.001*
Prior ACS History	8 (15.09%)	0 (0.00%)	8.71	0.003*
Stress / Type A Personality	27 (50.94%)	12 (22.64%)	8.86	0.003*

**p < 0.05 statistically significant. WC = Waist Circumference. CAD = Coronary Artery Disease.*

Table 2 reveals a strikingly higher prevalence of classical cardiovascular risk factors in cases compared to controls. Smoking (56.60% vs 15.09%), hypertension (64.15% vs 26.42%), Type 2 Diabetes Mellitus (52.83% vs 18.87%), family history of CAD (45.28% vs 16.98%), and physical inactivity (67.92% vs 33.96%) were all significantly more prevalent in ACS cases ($p < 0.001$ for most). These findings are consistent with established risk factor profiles in Indian ACS literature.

Table 3: Comparison of hs-CRP and Lipid Profile Parameters Between Cases and Controls

Parameter	Cases (n=53) Mean ± SD	Controls (n=53) Mean ± SD	t-value	p-value
hs-CRP (mg/L)	14.62 ± 6.84	1.83 ± 0.97	13.88	< 0.001*
Total Cholesterol (mg/dL)	214.8 ± 36.2	168.4 ± 27.8	7.41	< 0.001*
Triglycerides (mg/dL)	184.6 ± 42.1	128.3 ± 35.6	7.43	< 0.001*
HDL-C (mg/dL)	35.8 ± 7.4	48.6 ± 9.2	–8.08	< 0.001*
LDL-C (mg/dL)	148.36 ± 28.4	102.14 ± 21.6	9.51	< 0.001*
VLDL-C (mg/dL)	36.92 ± 8.42	25.66 ± 7.12	7.43	< 0.001*
TC / HDL-C Ratio	6.0 ± 1.28	3.5 ± 0.94	11.52	< 0.001*
LDL-C / HDL-C Ratio	4.1 ± 1.06	2.1 ± 0.74	11.40	< 0.001*

**p < 0.001 — highly statistically significant. SD = Standard Deviation. Values compared using unpaired Student's t-test.*

Table 3 demonstrates a highly significant and consistent pattern of derangement in both hs-CRP and all components of the lipid profile in ACS cases relative to controls. The hs-CRP mean in cases (14.62 ± 6.84 mg/L) was nearly 8-fold higher than controls (1.83 ± 0.97 mg/L), indicating a pronounced acute inflammatory response in the early phase of ACS. LDL-C, Total Cholesterol, Triglycerides, and VLDL-C were all significantly elevated, while HDL-C was significantly depressed in cases — collectively depicting a classic atherogenic dyslipidaemic profile ($p < 0.001$ for all parameters).

Table 4: Odds Ratio Analysis — Predictors of Acute Coronary Syndrome (Binary Logistic Regression)

Predictor Variable	Unadjusted OR	95% CI (Lower)	95% CI (Upper)	Adj. OR*	p-value
hs-CRP > 3 mg/L	12.4	5.8	26.4	10.8	< 0.001*
LDL-C > 130 mg/dL	8.7	3.9	19.2	7.2	< 0.001*
HDL-C < 40 mg/dL (M) / < 50 mg/dL (F)	6.3	2.9	13.6	5.4	< 0.001*
Total Cholesterol > 200 mg/dL	5.8	2.7	12.4	4.9	< 0.001*
Triglycerides > 150 mg/dL	5.2	2.4	11.2	4.3	< 0.001*
Hypertension (Yes vs No)	5.0	2.3	10.8	3.8	< 0.001*
Smoking (Current vs Non-smoker)	7.2	3.1	16.8	6.0	< 0.001*
Diabetes Mellitus (Yes vs No)	4.8	2.1	10.6	3.6	< 0.001*
Family History of CAD (Yes vs No)	3.9	1.7	8.8	3.1	0.001*
Physical Inactivity (Yes vs No)	4.1	1.9	8.9	3.2	0.001*
Obesity (BMI ≥ 30, Yes vs No)	3.2	1.4	7.3	2.6	0.006*
Regular Alcohol Use (Yes vs No)	2.7	1.2	6.2	2.1	0.020*
TC/HDL-C Ratio > 5 (Yes vs No)	9.1	4.2	20.0	7.8	< 0.001*
LDL/HDL-C Ratio > 3.5 (Yes vs No)	8.4	3.8	18.5	6.9	< 0.001*

**Adjusted OR: Adjusted for age, sex, BMI, and co-morbidities in binary logistic regression model. OR = Odds Ratio; CI = Confidence Interval; CAD = Coronary Artery Disease.*

Table 4 presents the results of binary logistic regression analysis. Elevated hs-CRP (>3 mg/L) emerged as the single strongest predictor of ACS with an unadjusted OR of 12.4 (95% CI: 5.8–26.4, p < 0.001), followed by a high TC/HDL-C ratio >5 (OR 9.1; 95% CI: 4.2–20.0), elevated LDL-C >130 mg/dL (OR 8.7; 95% CI: 3.9–19.2), and current smoking (OR 7.2; 95% CI: 3.1–16.8). Low HDL-C had an OR of 6.3 (95% CI: 2.9–13.6), reflecting its strong protective function in cardiovascular homeostasis. Even after adjustment for age, sex, BMI, and comorbidities, all biomarkers retained statistical significance, establishing their independent predictive value.

Table 5: Pearson Correlation of hs-CRP with Lipid Parameters in ACS Cases

Lipid Parameter	Pearson r	p-value
hs-CRP vs Total Cholesterol	+0.612	< 0.001*
hs-CRP vs Triglycerides	+0.584	< 0.001*
hs-CRP vs LDL-C	+0.638	< 0.001*
hs-CRP vs VLDL-C	+0.492	< 0.001*
hs-CRP vs HDL-C	–0.541	< 0.001*
hs-CRP vs TC/HDL-C Ratio	+0.671	< 0.001*

**Statistically significant positive/negative correlation. r = Pearson correlation coefficient.*

Table 5 demonstrates statistically significant positive correlations between hs-CRP and atherogenic lipid parameters (TC, TG, LDL-C, VLDL-C) and a significant negative correlation with HDL-C. The strongest correlation was observed between hs-CRP and TC/HDL-C ratio (r = +0.671), suggesting that systemic inflammation synergistically amplifies the atherogenic effect of dyslipidaemia. The inverse correlation with HDL-C (r = –0.541) supports the concept that inflammation suppresses reverse cholesterol transport.

5. Discussion

The present hospital-based case-control study was conducted with the objective of evaluating hs-CRP and lipid profile parameters in the early phase of ACS among patients aged 40–65 years at a tertiary care centre in West Bengal. The findings provide compelling evidence for the role of both inflammation and dyslipidaemia as intertwined pathophysiological mechanisms in the genesis and early evolution of ACS[7-10].

5.1 Sociodemographic Findings

In the present study, males outnumbered females in both the case and control groups (71.70% vs 28.30%), which is consistent with existing literature reporting a higher predisposition of males to premature and early ACS in India. A study by Gupta et al. (2020) from North India similarly reported male predominance (approximately 72%) in ACS patients below 65 years. The predominance of the 51–60 years age group in cases mirrors published epidemiological trends indicating peak ACS incidence in the sixth decade of life in the Indian population. Notably, though cases and controls were comparable in age and sex distribution, the mean BMI was significantly higher in cases (27.84 ± 4.62 vs 25.36 ± 3.94; $p = 0.003$), reinforcing the metabolic underpinnings of ACS. Overweight and obesity have been increasingly recognised as pro-inflammatory states, driving both hs-CRP elevation and dyslipidaemia[11-12].

5.2 Clinical Risk Factors

The risk factor analysis in Table 2 revealed that hypertension, Type 2 Diabetes Mellitus, current smoking, physical inactivity, family history of CAD, and central obesity were significantly more prevalent among cases. Current smoking was reported in 56.60% of cases — a finding corroborated by the INTERHEART study, which identified smoking as one of the nine modifiable risk factors collectively accounting for over 90% of attributable risk for acute myocardial infarction globally. The high prevalence of diabetes (52.83%) in cases reflects the compounding role of hyperglycaemia in promoting endothelial dysfunction, oxidative stress, and platelet hyper-reactivity — all of which accelerate the pathway to ACS.

Physical inactivity was prevalent in 67.92% of cases versus 33.96% of controls — a finding of particular public health significance in the context of the rapidly changing sedentary

lifestyle pattern in West Bengal's urban and semi-urban population. Habitual physical activity is well-established as a cardioprotective factor that reduces CRP levels, improves HDL-C, and maintains vascular endothelial integrity[13-16].

5.3 hs-CRP in ACS — Discussion

The most striking biochemical finding of this study was the markedly elevated mean hs-CRP in cases (14.62 ± 6.84 mg/L) compared to controls (1.83 ± 0.97 mg/L) — a nearly 8-fold difference ($p < 0.001$). This elevation reflects the intense systemic inflammatory response accompanying plaque rupture and myocardial necrosis that characterises the acute phase of ACS. CRP is not merely an epiphenomenon of inflammation; it actively participates in complement activation, opsonisation of LDL particles, and endothelial dysfunction — all of which perpetuate vascular injury[17].

Our findings are in strong concordance with those of Bhatt et al. (2018, n = 80) from Ahmedabad, who reported a mean hs-CRP of 13.4 ± 5.6 mg/L in STEMI patients. Similarly, Sharma et al. (2019) from AIIMS Delhi found hs-CRP to be independently predictive of major adverse cardiovascular events (MACE) at 30 days in ACS patients, with an OR of 9.8 (95% CI: 4.2–22.6). Ridker's landmark Jupiter Trial firmly established hs-CRP as an independent cardiovascular risk predictor beyond LDL-C. In the present study, hs-CRP yielded an unadjusted OR of 12.4 (95% CI: 5.8–26.4) — the highest among all predictors assessed — validating its centrality in ACS pathophysiology[18].

The significant positive correlations of hs-CRP with LDL-C ($r = +0.638$), TC ($r = +0.612$), TG ($r = +0.584$), and the strong negative correlation with HDL-C ($r = –0.541$) suggest that inflammation and dyslipidaemia amplify each other's effects, creating a vicious cycle of atherosclerosis acceleration. The TC/HDL-C ratio demonstrated the strongest correlation with hs-CRP ($r = +0.671$), indicating that systemic inflammation most powerfully reflects the overall atherogenic burden rather than any single lipid fraction in isolation.

5.4 Lipid Profile in ACS — Discussion

All lipid parameters were significantly deranged in ACS cases compared to controls ($p < 0.001$ for all). Elevated LDL-C (148.36 ± 28.4 vs 102.14 ± 21.6 mg/dL) is a well-established

driver of atherosclerosis. LDL particles, particularly the small, dense LDL (sdLDL) subfraction, infiltrate the subendothelial space, undergo oxidation, and initiate the macrophage-driven foam cell formation that forms the nidus of the atherosclerotic plaque. The Framingham Heart Study and subsequent trials have robustly established the linear relationship between LDL-C and coronary risk. In the present study, LDL-C >130 mg/dL yielded an OR of 8.7 (95% CI: 3.9–19.2), underscoring its strong independent predictive power.

Low HDL-C (35.8 ± 7.4 vs 48.6 ± 9.2 mg/dL; $p < 0.001$) was the second most significant lipid predictor (OR 6.3; 95% CI: 2.9–13.6). HDL-C mediates reverse cholesterol transport — the critical mechanism by which peripheral tissues, including arterial walls, return excess cholesterol to the liver for catabolism and excretion. Furthermore, HDL-C exerts anti-inflammatory, anti-oxidant, and anti-thrombotic effects. Its depletion thus removes multiple layers of cardioprotection. This finding aligns with data from the PROCAM Study and the Emerging Risk Factors Collaboration (ERFC), both of which demonstrated that low HDL-C independently and substantially increases coronary event risk.

Hypertriglyceridaemia (184.6 ± 42.1 vs 128.3 ± 35.6 mg/dL; $p < 0.001$) was also significantly associated with ACS. Elevated TG promotes the formation of VLDL and IDL particles, shifts the LDL distribution towards smaller, denser, more atherogenic particles, and increases hepatic CRP synthesis — thus establishing a direct mechanistic link between hypertriglyceridaemia and both atherogenesis and inflammation. The elevated VLDL-C (36.92 ± 8.42 vs 25.66 ± 7.12 mg/dL) in cases further corroborates these findings.

The composite atherogenic indices — TC/HDL-C ratio (6.0 ± 1.28 vs 3.5 ± 0.94) and LDL/HDL-C ratio (4.1 ± 1.06 vs 2.1 ± 0.74) — were markedly elevated in cases and yielded OR values of 9.1 and 8.4, respectively, establishing them as superior predictors compared to individual lipid fractions alone. Several studies, including that of Millán et al. (2009), have advocated for the routine use of lipid ratios in cardiovascular risk assessment due to their superior discriminative ability.

5.5 Strengths and Limitations

The study has several notable strengths: (a) rigorous 1:1 matching for age and sex, (b) exclusion of confounding inflammatory conditions, (c) standardised laboratory methods with quality controls, and (d) use of logistic regression to adjust for potential confounders. The study is, however, subject to certain inherent limitations. As a single-centre study, findings may not be universally generalisable to all settings across West Bengal or India. The cross-sectional nature of biomarker assessment precludes temporal causal inference — it is not possible to determine whether elevated hs-CRP preceded or followed plaque rupture. Additionally, detailed dietary assessment and assessment of sdLDL subfraction and Lipoprotein(a) were beyond the scope of this investigation.

6. Conclusion

The present case-control study conclusively demonstrates that elevated hs-CRP and an atherogenic lipid profile — characterised by high LDL-C, high Total Cholesterol, high Triglycerides, elevated VLDL-C, and low HDL-C — are strongly and independently associated with Acute Coronary Syndrome in the 40–65 years age group at a tertiary care centre in West Bengal.

hs-CRP emerged as the single most powerful predictor of ACS (OR = 12.4; 95% CI: 5.8–26.4), followed by the TC/HDL-C ratio (OR 9.1) and LDL-C (OR 8.7). The significant positive correlations between hs-CRP and atherogenic lipid parameters, and its inverse relationship with HDL-C, highlight the intricate crosstalk between systemic inflammation and lipid metabolism in the pathogenesis of ACS. Smoking, hypertension, diabetes, and physical inactivity were the predominant modifiable risk factors in this population.

Early measurement of hs-CRP alongside a complete fasting lipid profile, particularly in patients presenting with chest pain or those with multiple cardiovascular risk factors, can significantly enhance risk stratification, guide therapeutic decisions, and facilitate timely institution of anti-inflammatory and lipid-lowering therapy. These findings carry important implications for preventive cardiology practice in the Indian setting.

7. Recommendations

Based on the findings of this study, the following recommendations are made:

Routine hs-CRP measurement should be incorporated into the standard biochemical panel for all patients presenting with suspected ACS, particularly in the 40–65 years age group, to enable early risk stratification and guide intensity of anti-inflammatory therapy.A complete fasting lipid profile — including TC/HDL-C and LDL/HDL-C ratios — should be obtained at the earliest possible opportunity in ACS patients. Lipid ratios, rather than individual fractions alone, should be considered as part of the cardiovascular risk algorithm.Aggressive multi-factorial risk factor modification — encompassing cessation of smoking, glycaemic control in diabetics, antihypertensive therapy, dietary modification, and structured physical activity — should be instituted at the community level through targeted health education and preventive cardiology programmes.Cardiac rehabilitation programmes in West Bengal should incorporate monitoring of hs-CRP and lipid fractions as objective markers of therapeutic efficacy and residual cardiovascular risk.

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