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Original Research Article

High-Sensitivity C-Reactive Protein and Lipid Profiles in Early Acute Coronary Syndrome

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Abstract

Background: Acute coronary syndrome is associated with inflammation and lipid abnormalities that influence plaque instability and myocardial injury. Early biomarker assessment may improve risk stratification and clinical outcomes.

Objectives: To evaluate the association of high-sensitive C-reactive protein and lipid profile within 24 hours of symptom onset in patients with acute coronary syndrome.

Material and Methods: A hospital-based observational study was conducted on 150 participants comprising 100 ACS patients and 50 controls. ACS patients were subdivided based on symptom onset into <6 hours and ≥6–24 hours groups. hs-CRP and lipid profile were analyzed and compared.

Results: hs-CRP levels were significantly higher in ACS patients and increased with delayed presentation. ACS patients showed significantly higher triglycerides, LDL-C, VLDL-C, and lipid ratios with lower HDL-C compared to controls. Late presenters exhibited more pronounced inflammatory and lipid abnormalities.

Conclusion: hs-CRP and lipid profile assessment within 24 hours of ACS onset provides valuable insight into inflammatory burden and disease severity and may aid in early risk stratification and therapeutic planning.

Keywords: Acute coronary syndrome; hs-CRP; Lipid profile; Inflammation.

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Introduction

Acute coronary syndrome (ACS) represents a spectrum of clinical conditions resulting from acute myocardial ischemia, including unstable angina, non–ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). Despite major advances in diagnostic modalities and therapeutic strategies, ACS continues to be a leading cause of morbidity and mortality worldwide.

Early identification of high-risk patients during the initial hours of symptom onset remains crucial for optimizing clinical outcomes and guiding therapeutic decision-making [1]. Inflammation plays a central role in the initiation, progression, and destabilization of atherosclerotic plaques. Among inflammatory biomarkers, high-sensitive C-reactive protein (hs-CRP) has emerged as a robust and reproducible marker reflecting low-grade systemic inflammation. Elevated hs-CRP levels have been shown to correlate with endothelial dysfunction, plaque vulnerability, and thrombus formation, thereby contributing to the pathophysiology of ACS [2]. Measurement of hs-CRP allows detection of subtle inflammatory changes that may not be evident with conventional CRP assays, making it particularly valuable in the early phase of ACS [3].

Dyslipidemia is a well-established modifiable risk factor for coronary artery disease and ACS. Abnormal lipid parameters, including elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides, and reduced high-density lipoprotein cholesterol (HDL-C), contribute to atherogenesis and plaque instability. However, lipid levels are known to fluctuate during the acute phase of myocardial ischemia due to metabolic and inflammatory responses, potentially masking baseline lipid abnormalities if not assessed early

[4]. Therefore, lipid profile estimation within 24 hours of symptom onset is recommended to accurately reflect the patient's underlying lipid status [5].

Recent studies have highlighted a significant association between inflammatory markers and lipid abnormalities in ACS patients. Elevated hs-CRP levels have been independently associated with adverse lipid patterns, suggesting an interaction between inflammation and lipid metabolism in acute ischemic events [6]. This interplay accelerates plaque rupture and worsens myocardial injury, emphasizing the importance of combined evaluation of inflammatory and lipid biomarkers in ACS [7].

Early-phase assessment of hs-CRP and lipid profile may provide incremental prognostic information beyond traditional risk factors. Elevated hs-CRP measured within the first 24 hours of ACS has been linked to increased risk of recurrent ischemic events, heart failure, and mortality [8]. Similarly, specific lipid abnormalities identified early in ACS have been associated with worse short- and long-term outcomes [9]. Integrating these parameters may improve early risk stratification and help tailor aggressive therapeutic interventions.

Despite growing evidence, data on the combined evaluation of hs-CRP and lipid profile specifically within the first 24 hours of ACS onset remain limited, particularly in diverse clinical settings. Understanding this association in the early phase of ACS could enhance diagnostic accuracy, refine prognostic assessment, and support timely initiation of anti-inflammatory and lipid-lowering strategies [10]. Therefore, the present study aims to examine the relationship between high-sensitive C-reactive protein and lipid profile within 24 hours of symptom onset in patients presenting with acute coronary syndrome.

Material and Methods

This hospital-based, observational, case–control study was conducted in the Department of Cardiology of a tertiary care teaching hospital over a defined study period after obtaining approval from the Institutional Ethics Committee. A total of 150 participants were included in the study, comprising 100 patients diagnosed with acute coronary syndrome (ACS) and 50 age- and sex-matched healthy controls. Written informed consent was obtained from all participants prior to enrolment, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Patients presenting to the emergency department with clinical features suggestive of ACS were evaluated and diagnosed based on standard criteria, including typical ischemic chest pain, electrocardiographic changes, and elevated cardiac biomarkers. The ACS group included patients with ST-elevation myocardial infarction, non–ST-elevation myocardial infarction, and unstable angina. The 100 ACS patients were further categorized into two subgroups based on the time of onset of symptoms: Group 1 included patients presenting within less than 6 hours of onset of ACS symptoms, and Group 2 included patients presenting at or after 6 hours but within 24 hours of symptom onset.

The control group consisted of 50 apparently healthy individuals without any clinical evidence of coronary artery disease. Controls were selected from hospital staff and patient attendants who had no history of cardiovascular disease, diabetes mellitus, hypertension, dyslipidemia, or chronic inflammatory conditions, and who were not on lipid-lowering or anti-inflammatory medications.

Venous blood samples were collected from all ACS patients within 24 hours of symptom onset and prior to initiation of statin or anti-inflammatory therapy. For controls, blood samples were collected under fasting conditions. Serum was separated by centrifugation and analyzed for high-sensitive C-reactive protein (hs-CRP) and lipid profile parameters, including total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol, using standardized enzymatic methods in the central clinical biochemistry laboratory.

Data were recorded systematically and subjected to statistical analysis using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as frequencies and percentages. Comparisons between ACS patients and controls, as well as between the two ACS subgroups based on symptom onset time, were performed using appropriate parametric or non-parametric tests. A p-value of less than 0.05 was considered statistically significant.

Results

The demographic characteristics of the study population are summarized in Table 1. The mean age of ACS patients was 58.6 ± 7.2 years, which was comparable to controls (57.9 ± 6.8 years), with no statistically significant difference ($p > 0.05$). Male predominance was observed in both groups, with 64 males (64%) among ACS patients and 31 males (62%) among controls ($p > 0.05$).

Body mass index was significantly higher in ACS patients (26.8 ± 3.6 kg/m²) compared to controls (25.2 ± 3.4 kg/m²) ($p < 0.01$). Mean hemoglobin levels were lower in ACS patients (13.2 ± 1.9 g/dL) than in controls (14.0 ± 1.7 g/dL), showing a statistically significant difference ($p < 0.05$). The

prevalence of hypertension (42% vs 40%), diabetes mellitus (28% vs 26%), smoking (31% vs 30%), alcohol consumption (29% vs 28%), and family history of coronary heart disease (19% vs 18%) did not differ significantly between ACS patients and controls ($p > 0.05$). However, a significantly higher proportion of ACS patients (27%) had a personal history of coronary heart disease compared to controls ($p < 0.001$).

Mean levels of hs-CRP and lipid profile parameters in ACS patients and controls are shown in Table 2. ACS patients demonstrated markedly elevated hs-CRP levels (8.6 ± 4.9 mg/L) compared to controls (1.1 ± 0.6 mg/L), which was highly significant ($p < 0.001$). Total cholesterol levels were higher in ACS patients (171.2 ± 23.1 mg/dL) than controls (162.4 ± 18.5 mg/dL) ($p < 0.01$). Triglyceride levels were significantly elevated in ACS patients (146.8 ± 45.6 mg/dL) compared to controls (116.2 ± 26.1 mg/dL) ($p < 0.001$). LDL-C levels were also higher among ACS patients (101.6 ± 21.3 mg/dL) than controls (90.1 ± 18.2 mg/dL) ($p < 0.001$), whereas HDL-C levels were significantly reduced in ACS patients (41.3 ± 9.8 mg/dL) compared to controls (49.6 ± 8.9 mg/dL) ($p < 0.001$). VLDL-C levels and lipid ratios, including TC:HDL-C and LDL-C:HDL-C ratios, were significantly higher in ACS patients than controls ($p < 0.001$).

Comparison of hs-CRP and lipid profile parameters among ACS subgroups and controls is depicted in Table 3. Patients presenting ≥6 hours to <24 hours after symptom onset (Group II) exhibited significantly higher hs-CRP levels (12.1 ± 4.3 mg/L) compared to those presenting within 6 hours (Group I: 3.6 ± 1.9 mg/L) and controls (1.1 ± 0.6 mg/L) ($p < 0.001$). Triglyceride, LDL-C, VLDL-C, TC:HDL-C, and LDL-C:HDL-C ratios were progressively higher from controls to Group I and highest in Group II, while HDL-C levels showed a declining trend, with the lowest values observed in Group II (38.9 ± 8.7 mg/dL). Total cholesterol levels showed a mild increase across the groups but did not reach statistical significance between ACS subgroups ($p > 0.05$).

Table 1: Demographic characteristics of ACS patients and controls (n = 150)

Parameters	ACS patients (n = 100)	Controls (n = 50)	p value
Age (years)	58.6 ± 7.2	57.9 ± 6.8	>0.05
Female/Male	36/64	19/31	>0.05
Body mass index (kg/m²)	26.8 ± 3.6	25.2 ± 3.4	<0.01
Hemoglobin (g/dL)	13.2 ± 1.9	14.0 ± 1.7	<0.05
Hypertension, n (%)	42 (42)	20 (40)	>0.05
Diabetes mellitus, n (%)	28 (28)	13 (26)	>0.05
Smoking, n (%)	31 (31)	15 (30)	>0.05
Alcohol intake, n (%)	29 (29)	14 (28)	>0.05
Family history of CHD, n (%)	19 (19)	9 (18)	>0.05
Personal history of CHD, n (%)	27 (27)	–	<0.001

Table 2: Mean levels of hs-CRP and lipid profile in ACS patients and controls

Parameters	ACS patients (n = 100)	Controls (n = 50)	p value
hs-CRP (mg/L)	8.6 ± 4.9	1.1 ± 0.6	<0.001
Total cholesterol (mg/dL)	171.2 ± 23.1	162.4 ± 18.5	<0.01
Triglycerides (mg/dL)	146.8 ± 45.6	116.2 ± 26.1	<0.001
LDL-C (mg/dL)	101.6 ± 21.3	90.1 ± 18.2	<0.001
HDL-C (mg/dL)	41.3 ± 9.8	49.6 ± 8.9	<0.001
VLDL-C (mg/dL)	29.4 ± 8.9	23.2 ± 5.1	<0.001
TC: HDL-C ratio	4.38 ± 1.21	3.44 ± 0.82	<0.001
LDL-C:HDL-C ratio	2.61 ± 0.96	1.94 ± 0.71	<0.001

Table 3: Comparison of studied parameters between ACS subgroups and controls

Parameters	<6 hours (Group I) (n = 45)	≥6 to <24 hours (Group II) (n = 55)	Controls (n = 50)
hs-CRP (mg/L)	3.6 ± 1.9	12.1 ± 4.3	1.1 ± 0.6
Total cholesterol (mg/dL)	168.7 ± 21.9	173.4 ± 24.2	162.4 ± 18.5
Triglycerides (mg/dL)	141.2 ± 42.8	150.9 ± 47.1	116.2 ± 26.1
LDL-C (mg/dL)	97.4 ± 19.6	104.9 ± 22.7	90.1 ± 18.2
HDL-C (mg/dL)	44.1 ± 9.6	38.9 ± 8.7	49.6 ± 8.9
VLDL-C (mg/dL)	28.2 ± 8.5	30.1 ± 9.2	23.2 ± 5.1
TC: HDL-C ratio	4.11 ± 1.18	4.59 ± 1.26	3.44 ± 0.82
LDL-C:HDL-C ratio	2.43 ± 0.89	2.79 ± 1.01	1.94 ± 0.71

Discussion

The present study evaluated the association between high-sensitive C-reactive protein (hs-CRP) and lipid profile within 24 hours of symptom onset in patients with acute coronary syndrome (ACS) and demonstrated a strong relationship between inflammatory burden, dyslipidemia, and timing of presentation. The markedly elevated hs-CRP levels observed in ACS patients compared with controls, and the progressive rise in hs-CRP from early presenters (<6 hours) to late presenters (≥6–24 hours), highlight the dynamic inflammatory response following plaque rupture and myocardial injury. Similar temporal increases in hs-CRP during the early phase of ACS have been reported, supporting its role as a sensitive marker of plaque instability and myocardial necrosis rather than merely a passive inflammatory bystander [11].

In the present study, hs-CRP levels were significantly higher in patients presenting after 6 hours, suggesting ongoing inflammatory amplification with delayed presentation.

This observation aligns with evidence demonstrating that inflammatory cytokine release intensifies several hours after ischemic insult, contributing to endothelial dysfunction, prothrombotic state, and adverse remodeling [12]. The low hs-CRP levels seen in early presenters (<6 hours) emphasize the importance of timing when interpreting inflammatory markers in ACS and reinforce the clinical value of early biomarker assessment for risk stratification.

Dyslipidemia was a prominent finding among ACS patients, with significantly higher triglycerides, LDL-C, VLDL-C, and atherogenic lipid ratios, along with reduced HDL-C levels, compared with controls. These findings corroborate previous studies showing that atherogenic lipid patterns accelerate plaque vulnerability and thrombus formation in ACS [13]. Notably, HDL-C levels were lowest in late presenters, supporting the inverse relationship between HDL-C and systemic inflammation. HDL-C is known to exert anti-inflammatory, antioxidative, and endothelial-protective effects; therefore, reduced levels may amplify inflammatory cascades during acute ischemic events [14].

The progressive worsening of lipid ratios, particularly TC:HDL-C and LDL-C:HDL-C, from controls to Group I and highest in Group II underscores the synergistic interaction between lipid abnormalities and inflammation in ACS pathophysiology. These ratios have been shown to predict cardiovascular risk more accurately than isolated lipid parameters, especially during acute events when lipid levels may fluctuate [15]. The lack of significant difference in total cholesterol between ACS subgroups further supports the concept that lipid ratios and qualitative lipid changes are more informative than absolute cholesterol values in the acute setting. Taken together, the findings of this study emphasize that hs-CRP and lipid profile assessment within the first 24 hours of ACS provides valuable insights into disease severity and inflammatory status. Early presenters exhibit comparatively lower inflammatory burden, whereas delayed presentation is associated with intensified inflammation and more adverse lipid patterns. This highlights the clinical importance of early hospital presentation, prompt biomarker evaluation, and early initiation of anti-inflammatory and lipid-lowering therapies to mitigate myocardial damage and improve outcomes.

Conclusion

The present study demonstrates that hs-CRP levels are significantly elevated in ACS patients and increase progressively with delayed presentation, reflecting an evolving inflammatory response. Dyslipidemia, characterized by elevated triglycerides, LDL-C, VLDL-C, and adverse lipid ratios with reduced HDL-C, is significantly associated with ACS and is more pronounced in patients presenting after 6 hours of symptom onset. Combined early assessment of hs-CRP and lipid profile within 24 hours of ACS onset serves as a valuable tool for early risk stratification, understanding disease severity, and guiding timely therapeutic interventions.

References

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40(3):237–269.
2. Ridker PM. Inflammation, C-reactive protein, and cardiovascular disease: moving past the marker versus mediator debate. Circ Res. 2014;114(4):594–595.
3. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO, Criqui M, et al. Markers of inflammation and cardiovascular disease: application to clinical and public health practice. Circulation. 2003;107(3):499–511.
4. Rosenson RS, Brewer HB Jr, Davidson WS, Fayad ZA, Fuster V, Goldstein J. Cholesterol efflux and atheroprotection: advancing the concept of reverse cholesterol transport. Circulation. 2012;125(15):1905–1919.
5. Nordestgaard BG, Langsted A, Mora S, Kolovou G, Baum H, Bruckert E. Fasting is not routinely required for determination of a lipid profile: clinical and laboratory implications. Eur Heart J. 2016;37(25):1944–1958.

6. Li JJ, Fang CH. C-reactive protein is not only an inflammatory marker but also a direct cause of cardiovascular diseases. Med Hypotheses. 2004;62(4):499–506.
7. Libby P. Inflammation in atherosclerosis. Nature. 2002;420(6917):868–874.
8. Kaptoge S, Di Angelantonio E, Lowe G, Pepys MB, Thompson SG, Collins R, et al. C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: an individual participant meta-analysis. Lancet. 2010;375(9709):132–140.
9. Schwartz GG, Steg PG, Szarek M, Bhatt DL, Bittner VA, Diaz R, et al. Alirocumab and cardiovascular outcomes after acute coronary syndrome. N Engl J Med. 2018;379(22):2097–2107.
10. Tawakol A, Ishai A, Li D, Takx RAP, Hur S, Kaiser Y, et al. Association of arterial and lymph node inflammation with distinct inflammatory pathways in patients with acute coronary syndromes. Circulation. 2017;135 (24):2373–2384.
11. Biasucci LM, Liuzzo G, Grillo RL, Caligiuri G, Rebuzzi AG, Buffon A. Elevated levels of C-reactive protein at discharge in patients with unstable angina predict recurrent instability. Circulation. 1999;99(7):855–860.
12. Haverkate F, Thompson SG, Pyke SDM, Gallimore JR, Pepys MB. Production of C-reactive protein and risk of coronary events in stable and unstable angina. Lancet. 1997;349(9050):462–466.
13. Miller M, Stone NJ, Ballantyne C, Bittner V, Criqui MH, Ginsberg HN. Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2011;123(20):2292–2333.
14. Besler C, Heinrich K, Riwanto M, Lüscher TF, Landmesser U. High-density lipoprotein-mediated anti-inflammatory effects in coronary artery disease. Eur Heart J. 2011;32(12):1441–1449.
15. Castelli WP, Anderson K, Wilson PW, Levy D. Lipids and risk of coronary heart disease: the Framingham Study. Ann Epidemiol. 1992;2(1–2):23–28.