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Original Research

Association of High-Sensitivity C-Reactive Protein with Dyslipidaemia and Cardiovascular Risk Factors in Adults: A Cross-Sectional Observational Study.

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Abstract
Background: Dyslipidaemia and low-grade inflammation contribute jointly to atherosclerotic cardiovascular disease. High-sensitivity C-reactive protein (hs-CRP) detects subclinical inflammation, but its relationship with lipid abnormalities and clustered cardiovascular risk factors requires further evaluation in Indian adults. *Objectives:* To assess the association of hs-CRP with dyslipidaemia, individual lipid parameters, and cardiovascular risk factors among adults. *Methods:* This hospital-based cross-sectional observational study included 100 adults at Government Medical College Khammam, Telangana, India, from October 2024 to April 2025. Demographic, clinical, anthropometric, fasting lipid, glucose, and hs-CRP data were recorded. hs-CRP was classified as low (<1 mg/L), intermediate (1-3 mg/L), or high (>3 mg/L). Correlation and multivariable logistic regression analyses were performed. *Results:* The mean age was 48.2 ± 11.6 years, and 58.0% were males. Dyslipidaemia was present in 62.0%, central obesity in 48.0%, hypertension in 38.0%, and diabetes in 24.0%. Median hs-CRP was 2.7 mg/L; 35.0% had hs-CRP >3 mg/L. Participants with dyslipidaemia had higher median hs-CRP than those without dyslipidaemia (3.4 versus 1.8 mg/L; p<0.001). hs-CRP correlated positively with waist circumference, body mass index, triglycerides, LDL cholesterol, total cholesterol, systolic blood pressure, and fasting glucose, and inversely with HDL cholesterol. Dyslipidaemia, central obesity, and hypertension were independently associated with high hs-CRP. *Conclusion:* Elevated hs-CRP was strongly associated with an adverse lipid profile, central obesity, and hypertension. Integrating hs-CRP with conventional risk-factor assessment can improve identification of adults with combined metabolic and inflammatory cardiovascular risk.

Keywords: *Cardiovascular risk; dyslipidaemia; high-sensitivity C-reactive protein; inflammation; lipid profile; obesity.*

Introduction

Cardiovascular disease remains a major cause of premature morbidity and mortality, and its development reflects the cumulative influence of dyslipidaemia, hypertension, diabetes, smoking, obesity, and physical inactivity. Although these conventional factors explain a substantial proportion of vascular risk, atherosclerosis is now understood as a lipid-driven inflammatory disorder rather than a passive process of cholesterol deposition. Modified lipoproteins, endothelial dysfunction, monocyte recruitment, cytokine signalling, and plaque inflammation interact throughout the evolution of atherosclerotic lesions and contribute to plaque instability and thrombosis.[1]

C-reactive protein is an acute-phase reactant produced predominantly by hepatocytes in response to interleukin-6 and other inflammatory mediators. High-sensitivity assays quantify low circulating CRP concentrations that are below the detection limits of conventional assays and permit assessment of chronic, low-grade systemic inflammation. Clinical and epidemiological studies have demonstrated that high-sensitivity C-reactive protein (hs-CRP) provides cardiovascular prognostic information beyond several traditional risk factors.[2,3] Concentrations below 1 mg/L, between 1 and 3 mg/L, and above 3 mg/L have commonly been used to identify lower, intermediate, and higher relative cardiovascular risk, respectively.[2]

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Dyslipidaemia promotes atherogenesis through the retention and oxidation of apolipoprotein B-containing lipoproteins within the arterial wall. Elevated low-density lipoprotein cholesterol (LDL-C), hypertriglyceridaemia, and reduced high-density lipoprotein cholesterol (HDL-C) frequently coexist with obesity, insulin resistance, and hypertension. These abnormalities also stimulate inflammatory pathways. Conversely, systemic inflammation can alter lipoprotein metabolism, impair HDL function, and increase triglyceride-rich particles, creating a bidirectional relationship between inflammation and an adverse lipid profile.

The combined presence of metabolic syndrome and elevated CRP has been associated with greater cardiovascular event rates than either condition alone.[4] Large-scale evidence further indicates that CRP concentrations are continuously associated with coronary heart disease, ischaemic stroke, and vascular mortality, although part of this relationship is explained by conventional risk factors.[5]

The clinical relevance of hs-CRP is particularly important in South Asian populations, who develop central adiposity, insulin resistance, dyslipidaemia, and cardiovascular disease at comparatively younger ages.[7] Population-based studies have reported higher hs-CRP concentrations among individuals with obesity, diabetes, abnormal lipid concentrations, and elevated blood pressure.[8,9]

Detailed clinical reviews indicate that hs-CRP contributes information comparable with other established cardiovascular variables, while current prevention guidelines recognise persistently elevated hs-CRP as a risk-enhancing factor in selected adults whose treatment decisions remain uncertain after conventional assessment.[6,10,11] However, local evidence linking hs-CRP with individual lipid components and clustered cardiovascular risk factors remains limited.

The present study aimed to determine the distribution of hs-CRP concentrations among adults, assess their association with dyslipidaemia and individual lipid parameters, examine correlations with anthropometric and cardiometabolic risk variables, and identify factors independently associated with hs-CRP concentrations above 3 mg/L.

Materials and Methods

Study design and setting: This hospital-based cross-sectional observational study was conducted at Government Medical College Khammam, Telangana, India, from October 2024 to April 2025. Adults attending the associated outpatient services were screened consecutively. Men and women aged 18-70 years who provided written informed consent and completed clinical, anthropometric, and biochemical assessment were eligible.

Individuals with acute infection, recent fever, chronic inflammatory or autoimmune disease, active malignancy, recent major surgery or trauma, pregnancy, severe hepatic or renal dysfunction, or corticosteroid or immunosuppressive therapy were excluded. Participants with incomplete data were also excluded.

Sample size and sampling: The minimum sample size was estimated using the single-proportion formula with 50% expected prevalence, 95% confidence, and 10% absolute precision. The calculated sample of 96 was rounded to 100. Consecutive eligible adults were enrolled until the target was achieved.

Clinical and anthropometric assessment: Demographic information, smoking, physical activity, relevant medical history, medication use, hypertension, diabetes mellitus, and family history of premature cardiovascular disease were recorded using a structured form. Current smoking indicated active tobacco smoking at enrolment. Insufficient physical activity was defined as less than 150 minutes of moderate-intensity activity weekly.

Blood pressure was measured after five minutes of seated rest; the mean of two readings was recorded. Weight and height were measured using standard procedures, and body mass index was calculated as kg/m². Waist circumference was measured midway between the lowest rib and iliac crest. Central obesity was defined as waist circumference ≥90 cm in men or ≥80 cm in women.

Biochemical assessment: After an overnight fast of 8-12 hours, venous blood was collected. Serum total cholesterol, triglycerides, and high-density lipoprotein cholesterol (HDL-C) were measured using enzymatic methods.

Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation when triglycerides were below 400 mg/dL and measured directly when required. Serum hs-CRP was determined using a validated high-sensitivity immunoturbidimetric assay with routine internal quality control.

Operational definitions: Dyslipidaemia was defined by at least one abnormality: total cholesterol ≥200 mg/dL, LDL-C ≥130 mg/dL, triglycerides ≥150 mg/dL, HDL-C <40 mg/dL in men, or HDL-C <50 mg/dL in women.[12] hs-CRP was categorised as low (<1 mg/L), intermediate (1-3 mg/L), or high (>3 mg/L).[2,6]

Statistical analysis: Normally distributed variables were expressed as mean ± standard deviation and skewed variables as median with interquartile range. Categorical data were presented as frequencies and percentages. Comparisons used one-way analysis of variance, Kruskal-Wallis, chi-square, or Fisher's exact tests, as appropriate.

Spearman correlation assessed associations between hs-CRP and continuous variables. Multivariable logistic regression generated adjusted odds ratios with 95% confidence intervals for hs-CRP >3 mg/L. A two-sided p-value <0.05 indicated statistical significance.

Ethical considerations: Necessary Permissions were obtained before starting the study. Written informed consent was obtained, and participant confidentiality was protected.

Results

Participant recruitment and baseline characteristics

A total of 108 adults were assessed for eligibility during the study period. Eight individuals were excluded: five did not fulfil the eligibility criteria and three had incomplete biochemical information. The remaining 100 participants were included in the final analysis, with complete clinical and laboratory data available for all study variables.

The mean age was 48.2 ± 11.6 years, with a range of 25-70 years; 58 (58.0%) participants were males. The mean body mass index was 27.1 ± 4.5 kg/m², and the mean waist circumference was 92.3 ± 11.8 cm. Overweight and obesity were identified in 38 (38.0%) and 28 (28.0%) participants, respectively, while central obesity was present in 48 (48.0%).

Hypertension was documented in 38 (38.0%), diabetes mellitus in 24 (24.0%), current smoking in 22 (22.0%), and insufficient physical activity in 54 (54.0%). Baseline characteristics are presented in Table 1.

Table 1. Baseline demographic and cardiovascular risk characteristics of the participants

Characteristic	Frequency/Mean	Percentage
Age, years, mean ± SD	48.2 ± 11.6	–
Age ≥50 years	47	47.0
Male sex	58	58.0
Female sex	42	42.0
Body mass index, kg/m², mean ± SD	27.1 ± 4.5	–
Normal body mass index	34	34.0
Overweight	38	38.0
Obesity	28	28.0
Waist circumference, cm, mean ± SD	92.3 ± 11.8	–
Central obesity	48	48.0
Hypertension	38	38.0
Diabetes mellitus	24	24.0
Current smoking	22	22.0
Insufficient physical activity	54	54.0
Family history of premature cardiovascular disease	29	29.0

SD: standard deviation.

Lipid profile and prevalence of dyslipidaemia

The mean total cholesterol concentration was 199.6 ± 41.8 mg/dL, the mean LDL-C concentration was 126.4 ± 35.2 mg/dL, and the mean HDL-C concentration was 43.8 ± 10.4 mg/dL. The median triglyceride concentration was 164 mg/dL (interquartile range: 122-218 mg/dL).

Overall, dyslipidaemia was identified in 62 (62.0%) participants. Hypertriglyceridaemia was the most frequent lipid abnormality, affecting 44 (44.0%), followed by elevated LDL-C in 41 (41.0%), reduced HDL-C in 39 (39.0%), and elevated total cholesterol in 31 (31.0%). Thirty-six (36.0%) participants had two or more abnormal lipid components (Table 2).

Table 2. Lipid profile and dyslipidaemia patterns among the participants

Lipid variable	Value
Total cholesterol, mg/dL, mean ± SD	199.6 ± 41.8
LDL-C, mg/dL, mean ± SD	126.4 ± 35.2
HDL-C, mg/dL, mean ± SD	43.8 ± 10.4
Triglycerides, mg/dL, median (IQR)	164 (122-218)
Any dyslipidaemia, n (%)	62 (62.0)
Elevated total cholesterol, n (%)	31 (31.0)
Elevated LDL-C, n (%)	41 (41.0)
Reduced HDL-C, n (%)	39 (39.0)
Hypertriglyceridaemia, n (%)	44 (44.0)
Two or more lipid abnormalities, n (%)	36 (36.0)

HDL-C: high-density lipoprotein cholesterol; IQR: interquartile range; LDL-C: low-density lipoprotein cholesterol; SD: standard deviation.

Distribution of high-sensitivity C-reactive protein

The median hs-CRP concentration was 2.7 mg/L (interquartile range: 1.4-4.6 mg/L). Based on cardiovascular risk categories, 27 (27.0%) participants had low hs-CRP concentrations of <1 mg/L, 38 (38.0%) had intermediate concentrations of 1-3 mg/L, and 35 (35.0%) had high concentrations of >3 mg/L.

Participants with dyslipidaemia had significantly higher median hs-CRP concentrations than those without dyslipidaemia: 3.4 mg/L (interquartile range: 2.0-5.2) versus 1.8 mg/L (interquartile range: 0.8-3.0), respectively (p<0.001). High hs-CRP concentrations occurred in 28 of 62 participants with dyslipidaemia (45.2%), compared with 7 of 38 participants without dyslipidaemia (18.4%; p=0.007).

Association between hs-CRP categories and lipid parameters

A progressive increase in atherogenic lipid concentrations was observed across low, intermediate, and high hs-CRP categories. Mean total cholesterol increased from 181.3 ± 34.6 mg/dL in the low hs-CRP group to 216.1 ± 42.5 mg/dL in the high hs-CRP group (p<0.001). LDL-C and triglycerides showed similar patterns. In contrast, mean HDL-C declined from 48.9 ± 9.6 mg/dL to 40.0 ± 9.4 mg/dL (p<0.001).

The prevalence of dyslipidaemia increased from 40.7% in the low hs-CRP category to 80.0% in the high category (p=0.006). Higher hs-CRP categories were also associated with greater body mass index, waist circumference, systolic blood pressure, and higher prevalences of hypertension, diabetes mellitus, and central obesity (Table 3).

Table 3. Clinical and lipid parameters according to hs-CRP category

Variable	Low hs-CRP, <1 mg/L (n=27)	Intermediate hs-CRP, 1-3 mg/L (n=38)	High hs-CRP, >3 mg/L (n=35)	p-value
Age, years	43.9 ± 10.8	48.1 ± 11.2	51.6 ± 11.8	0.041
Body mass index, kg/m²	24.4 ± 3.5	27.0 ± 4.0	29.2 ± 4.4	<0.001
Waist circumference, cm	85.4 ± 9.6	91.7 ± 10.2	98.1 ± 11.7	<0.001
Systolic blood pressure, mmHg	125.6 ± 14.3	132.8 ± 16.2	140.2 ± 17.8	0.002
Total cholesterol, mg/dL	181.3 ± 34.6	197.8 ± 38.5	216.1 ± 42.5	<0.001
LDL-C, mg/dL	111.2 ± 29.1	124.6 ± 31.8	140.2 ± 37.1	0.001
HDL-C, mg/dL	48.9 ± 9.6	43.7 ± 10.1	40.0 ± 9.4	<0.001
Triglycerides, mg/dL, median (IQR)	126 (101-166)	161 (124-204)	201 (154-258)	<0.001
Dyslipidaemia, n (%)	11 (40.7)	23 (60.5)	28 (80.0)	0.006
Hypertension, n (%)	5 (18.5)	14 (36.8)	19 (54.3)	0.012
Diabetes mellitus, n (%)	3 (11.1)	8 (21.1)	13 (37.1)	0.038
Central obesity, n (%)	6 (22.2)	17 (44.7)	25 (71.4)	<0.001

Continuous normally distributed variables are presented as mean ± SD. Triglycerides are presented as median (IQR). HDL-C: high-density

lipoprotein cholesterol; hs-CRP: high-sensitivity C-reactive protein; IQR: interquartile range; LDL-C: low-density lipoprotein cholesterol; SD: standard deviation.

Correlation of hs-CRP with lipid and cardiovascular risk variables

hs-CRP showed moderate positive correlations with triglycerides (ρ=0.49, p<0.001), LDL-C (ρ=0.45, p<0.001), and total cholesterol (ρ=0.42, p<0.001), together with an inverse correlation with HDL-C (ρ=-0.38, p<0.001). Among anthropometric variables, hs-CRP correlated with waist circumference (ρ=0.55, p<0.001) and body mass index (ρ=0.52, p<0.001). Weaker positive correlations were found with systolic blood pressure, fasting blood glucose, and age (Table 4).

Table 4. Correlation between hs-CRP and selected clinical and biochemical variables

Variable	Spearman correlation coefficient (ρ)	p-value
Total cholesterol	0.42	<0.001
LDL-C	0.45	<0.001
HDL-C	-0.38	<0.001
Triglycerides	0.49	<0.001
Body mass index	0.52	<0.001
Waist circumference	0.55	<0.001
Systolic blood pressure	0.31	0.002
Fasting blood glucose	0.34	0.001
Age	0.23	0.021

HDL-C: high-density lipoprotein cholesterol; hs-CRP: high-sensitivity C-reactive protein; LDL-C: low-density lipoprotein cholesterol.

Multivariable analysis of factors associated with elevated hs-CRP

In multivariable logistic regression, dyslipidaemia remained independently associated with hs-CRP >3 mg/L after adjustment for the included covariates. Participants with dyslipidaemia had approximately three-fold higher odds of high hs-CRP than those without dyslipidaemia (adjusted odds ratio [aOR]: 3.08; 95% confidence interval [CI]: 1.20-7.92; p=0.019).

Central obesity was independently associated with high hs-CRP (aOR: 3.44; 95% CI: 1.35-8.76; p=0.010), while hypertension was associated with a 2.61-fold increase in odds (95% CI: 1.04-6.56; p=0.041). Diabetes mellitus and current smoking showed positive associations that did not reach statistical significance after adjustment (Table 5).

Table 5. Multivariable logistic regression analysis of factors associated with hs-CRP >3 mg/L

Variable	Adjusted odds ratio	95% confidence interval	p-value
Dyslipidaemia	3.08	1.20-7.92	0.019
Central obesity	3.44	1.35-8.76	0.010
Hypertension	2.61	1.04-6.56	0.041
Diabetes mellitus	2.48	0.92-6.68	0.073
Current smoking	2.32	0.82-6.60	0.112
Age, per one-year increase	1.03	0.99-1.07	0.138
Male sex	1.29	0.52-3.21	0.581

hs-CRP: high-sensitivity C-reactive protein.

Discussion

This cross-sectional study demonstrated a substantial overlap between low-grade systemic inflammation, dyslipidaemia, and conventional cardiovascular risk factors. Nearly two-thirds of participants had dyslipidaemia, and more than one-third had hs-CRP concentrations above 3 mg/L. Participants with dyslipidaemia had a markedly higher median hs-CRP than those without dyslipidaemia, while the prevalence of dyslipidaemia increased stepwise across the low, intermediate, and high hs-CRP categories. These findings support the concept that adverse lipid metabolism and inflammation coexist within the cardiometabolic risk continuum.

The lipid pattern associated with increasing hs-CRP was consistently atherogenic. Total cholesterol, LDL-C, and triglycerides rose across hs-CRP categories, whereas HDL-C declined. Significant positive correlations were identified between hs-CRP and triglycerides, LDL-C, and total cholesterol, with an inverse correlation for HDL-C. Similar clustering has been described among adults with obesity and other cardiovascular risk factors. Ebrahimi et al. reported higher hs-CRP concentrations in individuals with obesity and diabetes,[8] while Ramos-Arellano et al. linked elevated hs-CRP risk categories with obesity, dyslipidaemia, and high blood pressure.[9] The present findings extend these observations by demonstrating both categorical and graded relationships between hs-CRP and individual lipid components.

Waist circumference and body mass index showed the strongest correlations with hs-CRP. Central obesity also remained independently associated with hs-CRP >3 mg/L. Visceral adipose tissue is metabolically active and releases interleukin-6, tumour necrosis factor-alpha, and other mediators that stimulate hepatic CRP synthesis. This mechanism provides a biologically plausible explanation for the progressive rise in hs-CRP across increasing levels of adiposity. The Indian perspective is clinically relevant because central obesity and metabolic abnormalities frequently occur at lower levels of general adiposity in South Asian adults.[7]

Hypertension was independently associated with high hs-CRP, while diabetes and smoking showed positive but statistically non-significant adjusted associations. The lack of statistical significance for the latter variables probably reflects the modest sample and limited number of high hs-CRP outcomes rather than the absence of biological relationships. Previous prospective evidence has shown that CRP adds prognostic information to established risk factors,[3,5] and the combined presence of metabolic syndrome and elevated CRP identifies individuals with particularly high event risk.[4] Contemporary prevention guidelines therefore consider elevated hs-CRP a risk-enhancing factor when conventional assessment leaves uncertainty regarding preventive therapy.[10,11]

The clinical implications are twofold. First, lipid evaluation should remain central because dyslipidaemia was common and independently related to high hs-CRP. Second, hs-CRP can provide complementary information about residual inflammatory risk, particularly in adults with central obesity or multiple cardiometabolic abnormalities. The JUPITER trial demonstrated cardiovascular benefit from statin therapy among selected adults with elevated hs-CRP despite non-elevated LDL-C,[13] while CANTOS established that targeted inflammation reduction can lower recurrent cardiovascular events independently of lipid lowering.[14] The present study does not establish treatment effects, but it reinforces the value of integrated assessment of lipid and inflammatory risk.

LIMITATIONS

The cross-sectional design prevents determination of temporal or causal relationships between hs-CRP and dyslipidaemia. Participants were recruited from a single hospital, which limits external validity. A single hs-CRP measurement could be influenced by unrecognised transient inflammation. Dietary intake, detailed medication exposure, socioeconomic status, and calculated ten-year cardiovascular risk were not comprehensively assessed. The modest sample also reduced precision in the multivariable model.

Conclusion

High hs-CRP concentrations were frequent among the studied adults and showed a clear association with dyslipidaemia and clustered cardiovascular risk factors. Increasing hs-CRP was accompanied by higher total cholesterol, LDL-C, triglycerides, body mass index, waist circumference, and systolic blood pressure, together with lower HDL-C. Dyslipidaemia, central obesity, and hypertension remained independently associated with hs-CRP concentrations above 3 mg/L. These findings indicate that hs-CRP provides complementary information regarding the inflammatory component of cardiometabolic risk. Combined assessment of lipid abnormalities, abdominal adiposity, blood pressure, glycaemic status, and hs-CRP can improve risk characterisation and support timely preventive counselling and clinical follow-up in adults without overt acute inflammatory illness across routine adult clinical settings.

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