



RESEARCH ARTICLE

Role of High Sensitivity CRP as an Early Biomarker of Subclinical Inflammation in Metabolic Syndrome

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ABSTRACT

Background: Metabolic syndrome (MetS) is a major public health concern characterized by central obesity, insulin resistance, dyslipidemia, and hypertension. Chronic low-grade inflammation has been recognized as a central mechanism underlying the pathogenesis of MetS. High sensitivity C-reactive protein (hs-CRP) is an established inflammatory biomarker that may help in early detection of subclinical inflammation and cardiovascular risk.

Aim: To evaluate the role of hs-CRP as an early biomarker of subclinical inflammation in metabolic syndrome.

Materials and Methods: This hospital-based observational cross-sectional study was conducted at Jawaharlal Nehru Medical College, Bhagalpur, Bihar, from 2024–2026 among 180 participants. Ninety diagnosed cases of metabolic syndrome and ninety healthy controls were included. Anthropometric measurements, blood pressure, fasting blood glucose, lipid profile, and hs-CRP levels were assessed. Statistical analysis was performed using SPSS version 26.0. Student's t-test, chi-square test, Pearson correlation analysis, and multivariate regression were used. A p-value <0.05 was considered statistically significant.

Results: Mean hs-CRP levels were significantly elevated among MetS patients (5.92 ± 2.14 mg/L) compared to controls (1.48 ± 0.76 mg/L) ($p < 0.001$). hs-CRP showed positive correlation with BMI ($r = 0.58$), waist circumference ($r = 0.61$), fasting blood glucose ($r = 0.52$), triglycerides ($r = 0.49$), and systolic blood pressure ($r = 0.46$). hs-CRP levels progressively increased with increasing number of metabolic syndrome components.

Conclusion: hs-CRP is significantly elevated in metabolic syndrome and strongly correlates with various metabolic risk factors, suggesting its role as an early biomarker of subclinical inflammation and cardiovascular risk assessment.

Keywords: Metabolic syndrome, hs-CRP, Inflammation, Obesity, Cardiovascular risk, Biomarker

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INTRODUCTION

Metabolic syndrome (MetS) refers to a cluster of metabolic abnormalities including abdominal obesity, insulin resistance, dyslipidemia, hypertension, and impaired glucose metabolism that collectively increase the risk of cardiovascular disease and type 2 diabetes mellitus. [1] The prevalence of metabolic syndrome has increased substantially worldwide due to rapid urbanization, unhealthy dietary patterns, physical inactivity, and increasing obesity rates. [2]

In India, metabolic syndrome has emerged as a major healthcare challenge, particularly in urban populations, with prevalence estimates ranging from 20% to 35% among adults. [3] Individuals with metabolic syndrome are at increased risk of coronary artery disease, cerebrovascular disease, and all-cause mortality. [4]

The pathophysiology of metabolic syndrome is complex and multifactorial. Insulin resistance and central obesity are considered the primary pathogenic mechanisms. [5] Adipose tissue functions as an active endocrine organ

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that secretes inflammatory cytokines such as tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), leptin, and resistin, which contribute to systemic inflammation and endothelial dysfunction.[6]

Chronic low-grade inflammation has been recognized as a hallmark feature of metabolic syndrome and plays an important role in the development of atherosclerosis and cardiovascular complications.[7] Inflammatory mediators released from visceral adipose tissue stimulate hepatic synthesis of acute phase reactants including C-reactive protein (CRP).[8]

C-reactive protein is a sensitive marker of systemic inflammation synthesized by hepatocytes under stimulation by IL-6 and other cytokines.[9] High sensitivity CRP assays can detect very low concentrations of CRP and are therefore useful for identifying subclinical inflammation.[10]

Elevated hs-CRP levels have been associated with obesity, insulin resistance, diabetes mellitus, hypertension, and dyslipidemia.[11] Several prospective studies have demonstrated that hs-CRP independently predicts future cardiovascular events including myocardial infarction and stroke.[12]

Previous studies have also shown that hs-CRP levels correlate positively with waist circumference, fasting blood glucose, triglycerides, and body mass index among patients with metabolic syndrome.[13] Some investigators have even proposed inclusion of hs-CRP as an additional component of metabolic syndrome because of its strong association with cardiometabolic risk.[14]

However, limited data are available regarding the association between hs-CRP and metabolic syndrome in eastern India. Therefore, the present study was conducted to evaluate the role of hs-CRP as an early biomarker of subclinical inflammation in metabolic syndrome among patients attending a tertiary care hospital in Bihar.

MATERIALS AND METHODS

Study Design

Hospital-based observational cross-sectional study.

Study Place

Jawaharlal Nehru Medical College, Bhagalpur, Bihar – 812001.

Study Duration

Two years (2024–2026).

Sample Size

180 participants.

- Cases: 90 metabolic syndrome patients
- Controls: 90 healthy individuals

Inclusion Criteria

- Age between 20–65 years
- Patients fulfilling modified NCEP ATP III criteria for metabolic syndrome
- Individuals willing to provide informed consent

Exclusion Criteria

- Acute or chronic infections
- Autoimmune diseases
- Chronic kidney disease
- Chronic liver disease
- Malignancy
- Pregnant women
- Patients receiving anti-inflammatory drugs or statins

Data Collection

Detailed clinical history, anthropometric measurements, and blood pressure recordings were obtained.

Laboratory Investigations

- Fasting blood glucose
- Serum triglycerides
- HDL cholesterol
- hs-CRP estimation by immunoturbidimetric method

Statistical Analysis

Data were analyzed using SPSS version 26.0.

- Continuous variables expressed as mean $\pm$ standard deviation
- Student's t-test used for comparison
- Pearson correlation coefficient used for association
- Multivariate regression analysis performed
- p-value <0.05 considered statistically significant

RESULTS

A total of 180 participants were included in the present study, comprising 90 patients diagnosed with metabolic syndrome (MetS) and 90 healthy controls. The demographic, anthropometric, clinical, and biochemical parameters were analyzed and compared between the two groups.

Demographic and Anthropometric Characteristics

The mean age of participants in the MetS group was 48.3 ± 9.4 years, while that of the control group was 46.7 ± 8.8 years. The difference was statistically insignificant ($p=0.241$). Male participants constituted 62.2% of cases and 57.8% of controls.

Body mass index (BMI), waist circumference, and systolic

blood pressure were significantly higher among patients with metabolic syndrome compared to controls ($p < 0.001$). These findings indicate the presence of significant central obesity and hypertension among MetS patients, as shown in Table 1.

Biochemical Parameters

The mean fasting blood glucose among MetS cases was significantly higher compared to controls (132.6 ± 28.5 mg/dL vs 89.2 ± 11.4 mg/dL; $p < 0.001$). Serum triglyceride levels were markedly elevated among MetS patients, whereas HDL cholesterol levels were significantly reduced.

The mean hs-CRP level among cases was 5.92 ± 2.14 mg/L compared to 1.48 ± 0.76 mg/L among controls, and this difference was highly statistically significant ($p < 0.001$). These findings are summarized in Table 2.

Figure 1 demonstrates significantly elevated hs-CRP levels among patients with metabolic syndrome compared to healthy controls.

Distribution of hs-CRP Levels Among Study Participants

Among metabolic syndrome patients, 72.2% had hs-CRP levels > 3 mg/L, indicating high cardiovascular risk, whereas only 8.9% of controls showed elevated hs-CRP levels. The distribution of hs-CRP categories is presented in Table 3.

Correlation of hs-CRP with Metabolic Parameters

Pearson correlation analysis revealed a strong positive correlation between hs-CRP and waist circumference ($r = 0.61$, $p < 0.001$), BMI ($r = 0.58$, $p < 0.001$), fasting blood glucose ($r = 0.52$, $p < 0.001$), triglycerides ($r = 0.49$, $p < 0.001$), and systolic blood pressure ($r = 0.46$, $p < 0.001$).

These findings suggest that increasing obesity and worsening metabolic abnormalities are associated with

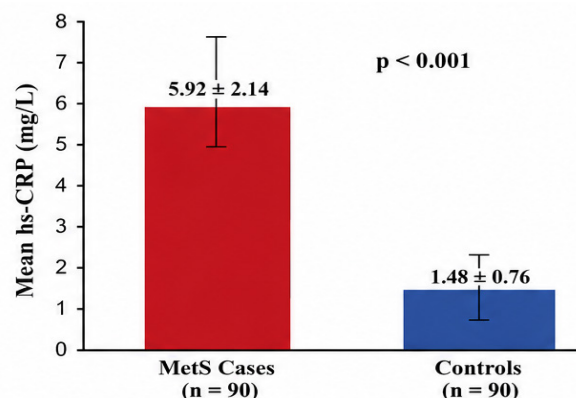


Figure 1: Comparison of Mean hs-CRP Levels Between MetS Cases and Controls

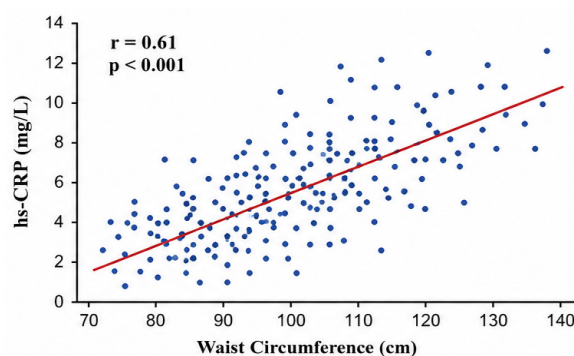


Figure 2: Correlation Between Waist Circumference and hs-CRP

greater degrees of systemic inflammation. The correlation analysis is presented in Table 4.

Figure 2 demonstrates a significant positive linear correlation between waist circumference and hs-CRP levels among metabolic syndrome patients.

Table 1: Demographic and Anthropometric Characteristics of Study Population

Variable	MetS Cases (n=90)	Controls (n=90)	p-value
Age (years)	48.3 ± 9.4	46.7 ± 8.8	0.241
Male (%)	56 (62.2%)	52 (57.8%)	0.563
Female (%)	34 (37.8%)	38 (42.2%)	0.563
BMI (kg/m ²)	31.6 ± 4.2	23.9 ± 2.8	<0.001
Waist Circumference (cm)	103.4 ± 9.8	82.6 ± 7.2	<0.001
Systolic BP (mmHg)	146.2 ± 14.6	118.5 ± 10.4	<0.001
Diastolic BP (mmHg)	92.4 ± 8.6	76.8 ± 7.2	<0.001

Table 2: Comparison of Biochemical Parameters Between Cases and Controls

Parameter	MetS Cases (n=90)	Controls (n=90)	p-value
Fasting Blood Glucose (mg/dL)	132.6 ± 28.5	89.2 ± 11.4	<0.001
Triglycerides (mg/dL)	218.4 ± 46.2	118.5 ± 24.3	<0.001
HDL Cholesterol (mg/dL)	37.2 ± 6.4	48.8 ± 7.2	<0.001
Total Cholesterol (mg/dL)	224.5 ± 38.7	176.8 ± 26.4	<0.001
LDL Cholesterol (mg/dL)	142.7 ± 31.5	98.4 ± 20.6	<0.001
hs-CRP (mg/L)	5.92 ± 2.14	1.48 ± 0.76	<0.001

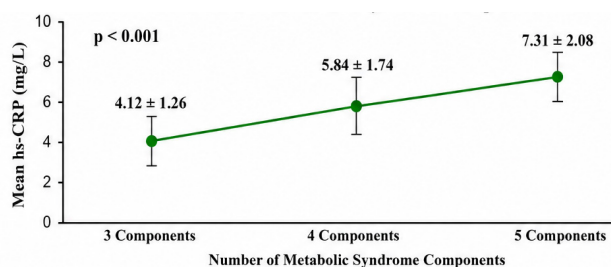


Figure 3: Progressive Increase in hs-CRP Levels with Increasing Metabolic Syndrome Components

hs-CRP Levels According to Number of Metabolic Syndrome Components

The mean hs-CRP levels increased progressively with increasing number of metabolic syndrome components. Patients with five components demonstrated the highest hs-CRP levels (7.31 ± 2.08 mg/L), whereas patients with three components showed comparatively lower hs-CRP levels (4.12 ± 1.26 mg/L). This trend was statistically significant (p<0.001), as shown in Table 5.

Figure 3 demonstrates a progressive increase in hs-CRP levels with increasing number of metabolic syndrome components.

Multivariate Regression Analysis

Multivariate regression analysis was performed to identify independent predictors of elevated hs-CRP levels. Waist circumference and fasting blood glucose emerged as independent predictors of increased hs-CRP levels after adjustment for confounding variables.

The regression analysis findings are summarized in Table 6.

The regression model demonstrated that abdominal obesity and hyperglycemia were the strongest predictors of subclinical inflammation among metabolic syndrome patients.

Table 3: Distribution of hs-CRP Levels Among Cases and Controls

hs-CRP Level (mg/L)	MetS Cases (n=90)	Controls (n=90)	p-value
<1 mg/L	6 (6.7%)	42 (46.7%)	
1–3 mg/L	19 (21.1%)	40 (44.4%)	
>3 mg/L	65 (72.2%)	8 (8.9%)	<0.001

Table 4: Correlation of hs-CRP with Metabolic Parameters

Parameter	Correlation coefficient (r)	p-value
BMI	0.58	<0.001
Waist Circumference	0.61	<0.001
Fasting Blood Glucose	0.52	<0.001
Triglycerides	0.49	<0.001
Systolic Blood Pressure	0.46	<0.001
LDL Cholesterol	0.38	<0.001
HDL Cholesterol	-0.41	<0.001

Table 5: hs-CRP Levels According to Number of Metabolic Syndrome Components

Number of components	Number of patients	Mean hs-CRP (mg/L)	p-value
3 Components	28	4.12 ± 1.26	
4 Components	37	5.84 ± 1.74	
5 Components	25	7.31 ± 2.08	<0.001

DISCUSSION

The present study demonstrated significantly elevated hs-CRP levels among patients with metabolic syndrome compared to healthy controls, indicating the presence of chronic low-grade inflammation in metabolic syndrome. [15] These findings support the concept that inflammation plays a central role in the pathogenesis of cardiometabolic disorders.[16]

Table 6: Multivariate Regression Analysis for Predictors of Elevated hs-CRP

Variable	Beta coefficient (β)	Standard error	p-value
Waist circumference	0.42	0.08	<0.001
BMI	0.28	0.11	0.012
Fasting blood glucose	0.36	0.09	<0.001
Triglycerides	0.21	0.10	0.028
Systolic blood pressure	0.17	0.07	0.041

The mean hs-CRP level among cases in the present study was 5.92 ± 2.14 mg/L, which was significantly higher than controls. Similar findings were reported by Ridker et al., who observed elevated hs-CRP levels in individuals at increased cardiovascular risk.[17] Jialal et al. also reported significant association between hs-CRP and metabolic syndrome components.[18]

A strong positive correlation was observed between hs-CRP and waist circumference. Visceral adiposity contributes significantly to inflammatory activation through release of IL-6 and TNF- α from adipocytes and macrophages present within adipose tissue.[19] Increased abdominal obesity therefore leads to enhanced hepatic CRP synthesis.[20]

The present study also demonstrated significant positive correlation between hs-CRP and fasting blood glucose. Hyperglycemia induces oxidative stress and endothelial dysfunction, thereby promoting inflammatory responses.[21] Similar associations between hs-CRP and insulin resistance have been reported in previous studies.[22]

Triglyceride levels showed significant positive correlation with hs-CRP in the current study. Dyslipidemia contributes to vascular inflammation and atherogenesis through endothelial injury and oxidative modification of lipoproteins.[23] Reduced HDL cholesterol among metabolic syndrome patients may further aggravate inflammation due to loss of antioxidant and anti-inflammatory properties of HDL particles.[24]

An important finding of the present study was the progressive increase in hs-CRP levels with increasing number of metabolic syndrome components. This observation suggests that cumulative metabolic abnormalities intensify systemic inflammation and cardiovascular risk.[25]

The present study emphasizes the potential role of hs-CRP as an early biomarker of subclinical inflammation in metabolic syndrome. Measurement of hs-CRP may therefore help in early identification of high-risk individuals and implementation of preventive interventions aimed at reducing cardiovascular morbidity and mortality.

However, the study had certain limitations. The cross-sectional design does not establish causality. Furthermore, long-term follow-up studies are required to determine the prognostic significance of hs-CRP among Indian populations with metabolic syndrome.

CONCLUSION

The present study demonstrated significantly elevated hs-CRP levels among patients with metabolic syndrome compared to healthy controls. hs-CRP showed strong positive correlation with obesity indices, fasting blood glucose, triglycerides, and blood pressure. The findings support the utility of hs-CRP as an early biomarker of subclinical inflammation and cardiovascular risk in metabolic syndrome.

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