DOI: https://doi.org/10.53350/pjmhs20231711355

ORIGINAL ARTICLE

Correlation Between High-Sensitivity C-Reactive Protein (HS-CRP) and Severity of Coronary Artery Disease in Diabetic Patients

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ABSTRACT

Background: Coronary artery disease (CAD) is a major cause of morbidity and mortality in patients with type 2 diabetes mellitus (T2DM). Systemic inflammation, as measured by high-sensitivity C-reactive protein (hs-CRP), has been implicated in the pathogenesis of atherosclerosis, yet its correlation with angiographic CAD severity in diabetic patients remains underexplored.

Objective: To assess the relationship between serum hs-CRP levels and the angiographic severity of CAD in patients with T2DM and evaluate the associated clinical and biochemical risk factors.

Methods: This cross-sectional study included 100 patients with type 2 diabetes mellitus (T2DM), aged between 40 and 75 years, who underwent coronary angiography for suspected coronary artery disease (CAD). Based on angiographic findings, patients were categorized into three groups according to disease severity: mild, moderate, and severe CAD. Clinical data, including age, gender, duration of diabetes, hypertension status, and body mass index (BMI), were systematically recorded. Laboratory investigations comprised high-sensitivity C-reactive protein (hs-CRP), fasting blood glucose, glycated hemoglobin (HbA1c), complete lipid profile, and serum creatinine. The association between hs-CRP levels and CAD severity was evaluated using IBM SPSS version 25.0, with a p-value of <0.05 considered statistically significant.

Results: Serum hs-CRP levels increased significantly with CAD severity (2.4 ± 0.9 mg/L in mild, 4.6 ± 1.1 mg/L in moderate, and 6.8 ± 1.5 mg/L in severe CAD; $p < 0.001$). Patients with severe CAD also had significantly higher fasting blood glucose, HbA1c, total cholesterol, LDL, triglycerides, and serum creatinine, and lower HDL levels compared to those with mild disease (all $p < 0.05$). Male predominance, older age, longer diabetes duration, higher BMI, and hypertension were more common in patients with advanced CAD.

Conclusion: Elevated hs-CRP levels show a strong and independent association with angiographic severity of CAD in patients with T2DM, supporting the role of systemic inflammation in diabetic atherogenesis. hs-CRP may serve as a valuable adjunctive biomarker for cardiovascular risk stratification in diabetic individuals undergoing CAD evaluation.

Keywords: Type 2 diabetes mellitus, Coronary artery disease, hs-CRP, Inflammatory biomarkers, Atherosclerosis, Angiography, Cardiovascular risk, Glycemic control

INTRODUCTION

Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide, with diabetic patients exhibiting a markedly increased risk for its development and progression. Diabetes mellitus (DM), particularly type 2, accelerates atherosclerotic changes in the coronary vasculature due to chronic hyperglycemia, insulin resistance, oxidative stress, and a pro-inflammatory state [1]. Among the various biomarkers of systemic inflammation, high-sensitivity C-reactive protein (hs-CRP) has emerged as a significant predictor and mediator of cardiovascular risk. hs-CRP is a hepatic acute-phase reactant synthesized in response to interleukin-6 and other pro-inflammatory cytokines, and its elevated levels have been independently associated with endothelial dysfunction, plaque instability, and adverse cardiac events[2].

In diabetic individuals, the interplay between persistent low-grade inflammation and vascular damage exacerbates the pathogenesis of CAD, even in the absence of overt clinical symptoms [3]. Several studies have suggested that hs-CRP may reflect the burden of subclinical inflammation and serve as a valuable marker for assessing atherosclerotic disease severity. This becomes particularly important in diabetic patients, in whom traditional risk stratification tools often underestimate the extent and severity of coronary artery involvement 19. Given the growing interest in inflammatory markers as tools for cardiovascular risk prediction, evaluating the correlation between hs-CRP levels and the angiographic severity of CAD in diabetic patients could provide deeper insights into the inflammatory underpinnings of diabetic heart disease [4]. This study aims to explore this relationship and assess whether hs-CRP can serve as a non-invasive marker for identifying diabetic individuals at higher risk of severe coronary artery involvement.

Understanding the relationship between hs-CRP and the severity of coronary artery disease in diabetic patients is crucial not only for risk stratification but also for guiding therapeutic decisions and monitoring treatment efficacy [5]. As inflammation plays a pivotal role in both the initiation and progression of atherosclerosis, hs-CRP offers a dynamic reflection of the underlying pathophysiological processes [6]. Elevated hs-CRP levels have been associated with multi-vessel disease, greater plaque burden, and poor clinical outcomes following percutaneous coronary interventions [20]. Furthermore, hs-CRP may help in identifying high-risk diabetic individuals who could benefit from aggressive lifestyle modification, anti-inflammatory therapies, and stricter glycemic control [7]. Therefore, investigating the clinical utility of hs-CRP as a biomarker for CAD severity in the diabetic population may provide a cost-effective and accessible tool for improving cardiovascular prognosis in this vulnerable group.

MATERIALS AND METHODS

This cross-sectional analytical study was conducted on 100 patients with type 2 diabetes mellitus (T2DM) who presented with symptoms suggestive of coronary artery disease (CAD) and underwent diagnostic coronary angiography at the cardiology departments of Aziz Bhatti Shaheed Teaching Hospital, Gujrat, and Chaudhary Pervaiz Elahi Institute of Cardiology, Wazirabad, Pakistan. The study was conducted over a defined period from June 2022 till May 2023, and participants were enrolled consecutively using a non-probability sampling technique. This approach allowed for the inclusion of all eligible diabetic patients presenting during the study window, facilitating the collection of

Received on 05-06-2023
Accepted on 10-10-2023

representative clinical data while maintaining feasibility within a hospital-based setting.

Both male and female patients aged between 40 and 75 years were included. The deliberate inclusion of both genders was intended to capture potential sex-related differences in inflammatory responses and CAD progression. Recognizing the emerging evidence that gender influences cardiac physiology, hormonal patterns, and inflammatory biomarker expression, a balanced gender representation enabled a more comprehensive and generalizable assessment of hs-CRP trends in the diabetic population. All included patients had a confirmed diagnosis of type 2 diabetes mellitus for a minimum of five years, providing a relatively homogeneous cohort at high risk for subclinical or established atherosclerosis.

Exclusion criteria comprised a history of acute infection, chronic inflammatory or autoimmune disease, malignancy, recent trauma or surgery within the past three months, or current use of immunosuppressive or anti-inflammatory medications. These conditions were excluded to avoid confounding elevations in hs-CRP and to ensure the inflammatory marker truly reflected cardiovascular risk. Ethical approval for the study was obtained from the institutional review boards of the participating hospitals, and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

Demographic and clinical information was collected using a structured proforma, which included age, gender, body mass index (BMI), duration of diabetes, smoking status, hypertension, and family history of CAD. BMI was calculated using the standard formula (weight in kilograms divided by the square of height in meters), and patients were categorized as overweight or obese based on WHO guidelines. Particular emphasis was placed on the duration of diabetes, given its established role in accelerating endothelial dysfunction and vascular inflammation. These variables were analyzed to explore their potential association with both CAD severity and inflammatory marker levels.

Venous blood samples were obtained from all patients after an overnight fast of 10 to 12 hours, using strict aseptic technique. The primary inflammatory marker measured was high-sensitivity C-reactive protein (hs-CRP), analyzed using a high-sensitivity immunoturbidimetric assay. Additional biochemical parameters included fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) to assess glycemic control, as well as a comprehensive lipid profile comprising total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides. Renal function was assessed using serum creatinine and urea levels; patients with evidence of chronic kidney disease were excluded to minimize confounding effects on hs-CRP levels.

All enrolled patients underwent standard coronary angiography via either femoral or radial access. The procedures were performed by experienced interventional cardiologists who were blinded to the patients' biochemical results, thereby minimizing observer bias. The angiographic severity of CAD was assessed by quantifying luminal stenosis and the number of involved vessels. Based on these findings, patients were categorized into three groups: mild CAD (less than 50% luminal narrowing in a single vessel), moderate CAD (50–70% narrowing in one or more vessels), and severe CAD (greater than 70% narrowing in multiple vessels or left main disease). This classification enabled correlation of hs-CRP levels with the anatomical burden of disease.

Data were analyzed using IBM SPSS version 25.0. Continuous variables such as age, BMI, hs-CRP, FBG, HbA1c, and lipid values were presented as mean ± standard deviation (SD), while categorical variables including gender, smoking status, and CAD severity were expressed as frequencies and percentages. Group comparisons were performed using independent-samples t-tests and one-way analysis of variance (ANOVA), and associations between categorical variables were tested using the Chi-square test. A p-value < 0.05 was considered statistically significant for all analyses.

RESULTS

The study enrolled a total of 100 patients with type 2 diabetes mellitus (T2DM) who underwent coronary angiography to evaluate the association between serum high-sensitivity C-reactive protein (hs-CRP) levels and the severity of coronary artery disease (CAD). Based on angiographic findings, patients were categorized into three groups: mild CAD (n=28), moderate CAD (n=34), and severe CAD (n=38). Among the total participants, 62 were male (62%) and 38 were female (38%), with a progressive increase in male proportion noted across the CAD severity spectrum. Specifically, the mild CAD group comprised 15 males (53.6%) and 13 females (46.4%), the moderate CAD group included 21 males (61.8%) and 13 females (38.2%), while the severe CAD group had 26 males (68.4%) and 12 females (31.6%). Although male predominance was observed with increasing CAD severity, the difference was not statistically significant (p = 0.271), indicating no gender-based bias in the distribution of disease severity.

A significant difference in age was observed among the three groups, with mean age increasing from 54.2 ± 6.3 years in the mild CAD group to 58.5 ± 7.4 years in moderate CAD, and 62.1 ± 6.9 years in the severe CAD group (p < 0.001). This suggests that advancing age may be independently associated with more extensive coronary artery involvement. The mean duration of diabetes also differed significantly among the groups, rising from 6.2 ± 2.5 years in patients with mild CAD to 7.9 ± 3.1 years in those with moderate disease and 9.4 ± 2.8 years in the severe CAD group (p < 0.001), reinforcing the role of chronic hyperglycemia in atherosclerotic progression. Hypertension was present in 11 patients (39.3%) in the mild CAD group, 19 patients (55.9%) in the moderate group, and 27 patients (71.0%) in the severe group, showing a statistically significant association between hypertensive status and CAD severity (p = 0.014). The body mass index (BMI) also increased progressively with disease severity, with a mean BMI of 27.3 ± 2.8 kg/m² in the mild group, 28.1 ± 3.2 kg/m² in moderate CAD, and 29.5 ± 3.1 kg/m² in severe CAD (p = 0.036), highlighting the role of obesity in CAD pathogenesis as shown in table 1.

Table 1: Demographic and Clinical Characteristics of the Study Population (with Gender Distribution)

Parameter	Mild CAD (n=28)	Moderate CAD (n=34)	Severe CAD (n=38)	p-value
Mean Age (years)	54.2 ± 6.3	58.5 ± 7.4	62.1 ± 6.9	<0.001
Male Gender (%)	15 (53.6%)	21 (61.8%)	26 (68.4%)	0.271
Female Gender (%)	13 (46.4%)	13 (38.2%)	12 (31.6%)	—
Duration of Diabetes (yrs)	6.2 ± 2.5	7.9 ± 3.1	9.4 ± 2.8	<0.001
Hypertension (%)	11 (39.3%)	19 (55.9%)	27 (71.0%)	0.014
BMI (kg/m²)	27.3 ± 2.8	28.1 ± 3.2	29.5 ± 3.1	0.036

In addition to clinical characteristics, significant differences were observed in biochemical and inflammatory markers among the three groups. High-sensitivity C-reactive protein (hs-CRP) levels showed a robust and statistically significant elevation with increasing CAD severity. The mean hs-CRP concentration was 2.4 ± 0.9 mg/L in the mild CAD group, rising to 4.6 ± 1.1 mg/L in moderate CAD and peaking at 6.8 ± 1.5 mg/L in the severe CAD group (p < 0.001), indicating a strong correlation between systemic inflammation and atherosclerotic burden. Similarly, fasting blood glucose levels increased progressively, from 132 ± 18 mg/dL in mild CAD to 148 ± 20 mg/dL in moderate and 165 ± 24 mg/dL in

severe CAD ($p < 0.001$), suggesting poorer glycemic control in patients with more severe coronary disease.

Glycated hemoglobin (HbA1c), a marker of long-term glycemic control, was also significantly higher in patients with more extensive CAD. Mean HbA1c values were 7.1 ± 0.6% in mild CAD, 7.9 ± 0.8% in moderate CAD, and 8.5 ± 0.7% in the severe CAD group ($p < 0.001$). Lipid profile analysis revealed significant dyslipidemia correlating with CAD severity. Total cholesterol levels rose from 182 ± 25 mg/dL in the mild group to 195 ± 32 mg/dL and 208 ± 28 mg/dL in the moderate and severe groups respectively ($p = 0.002$). Low-density lipoprotein (LDL) levels increased from 106 ± 19 mg/dL in mild CAD to 134 ± 23 mg/dL in severe CAD ($p < 0.001$), while high-density lipoprotein (HDL) levels declined significantly from 43 ± 6 mg/dL in mild CAD to 35 ± 5 mg/dL in severe disease ($p = 0.001$), indicating a pro-atherogenic lipid shift with worsening disease.

Triglycerides were markedly elevated in the severe CAD group (211 ± 36 mg/dL) compared to 185 ± 32 mg/dL in moderate and 160 ± 28 mg/dL in the mild CAD group ($p < 0.001$). Furthermore, serum creatinine levels, although within normal physiological limits, showed a subtle yet statistically significant increase, rising from 0.9 ± 0.2 mg/dL in mild CAD to 1.1 ± 0.3 mg/dL in severe CAD ($p = 0.031$), suggesting early renal stress associated with advanced cardiovascular pathology as shown in table 2.

Table 2: Biochemical and Inflammatory Biomarkers Across CAD Severity Groups

Biomarker	Mild CAD (n=28)	Moderate CAD (n=34)	Severe CAD (n=38)	p-value
hs-CRP (mg/L)	2.4 ± 0.9	4.6 ± 1.1	6.8 ± 1.5	<0.001
Fasting Blood Glucose	132 ± 18	148 ± 20	165 ± 24	<0.001
HbA1c (%)	7.1 ± 0.6	7.9 ± 0.8	8.5 ± 0.7	<0.001
Total Cholesterol	182 ± 25	195 ± 32	208 ± 28	0.002
LDL (mg/dL)	106 ± 19	118 ± 21	134 ± 23	<0.001
HDL (mg/dL)	43 ± 6	39 ± 7	35 ± 5	0.001
Triglycerides (mg/dL)	160 ± 28	185 ± 32	211 ± 36	<0.001
Serum Creatinine (mg/dL)	0.9 ± 0.2	1.0 ± 0.2	1.1 ± 0.3	0.031

Overall, the results indicate a clear and progressive increase in systemic inflammatory burden, glycemic dysregulation, dyslipidemia, and renal function compromise with increasing angiographic severity of coronary artery disease in diabetic patients. Importantly, both male and female patients were adequately represented across all CAD categories, and no statistically significant gender disparity was observed in disease severity, confirming the generalizability of these findings to both sexes. These observations strongly support the utility of hs-CRP and associated metabolic markers as valuable predictors of CAD progression in the diabetic population.

DISCUSSION

This study evaluated the correlation between high-sensitivity C-reactive protein (hs-CRP) levels and the angiographic severity of coronary artery disease (CAD) in patients with type 2 diabetes mellitus (T2DM), revealing a strong and statistically significant association across all disease categories[8]. The data demonstrated that hs-CRP levels increased progressively from patients with mild CAD to those with severe CAD, indicating that systemic inflammation plays a critical role in the pathophysiology and progression of coronary atherosclerosis in diabetic individuals[9]. These findings are in agreement with prior studies suggesting that hs-CRP functions not only as a sensitive marker of systemic inflammation but also as a direct contributor to atherogenesis through mechanisms involving endothelial dysfunction, plaque destabilization, and thrombotic activity[10].

In the present study, patients with severe CAD exhibited significantly higher fasting blood glucose and glycated hemoglobin (HbA1c) levels, reflecting inadequate long-term glycemic control. Chronic hyperglycemia is well documented to enhance oxidative stress, activate pro-inflammatory signaling pathways, and promote endothelial injury, all of which accelerate atherosclerotic progression[11]. The observed co-elevation of hs-CRP with poor glycemic indices further supports the hypothesis that systemic inflammation serves as a mechanistic link between metabolic disturbance and vascular injury in diabetes mellitus. This relationship reinforces earlier findings that hyperglycemia and inflammation act synergistically in the development of diabetic macrovascular complications[12].

Additionally, the study found a consistent trend in dyslipidemia across CAD severity groups. Patients with more advanced disease had significantly higher levels of total cholesterol, low-density lipoprotein (LDL), and triglycerides, along with reduced high-density lipoprotein (HDL) levels. These abnormalities are established contributors to atherogenesis and are particularly hazardous when accompanied by elevated inflammatory markers such as hs-CRP[13]. Dyslipidemia and systemic inflammation not only contribute independently to endothelial dysfunction and plaque formation but may also potentiate each other, resulting in accelerated vascular injury in patients with T2DM[14].

Interestingly, although serum creatinine levels remained within the normal reference range, patients with severe CAD demonstrated a mild yet statistically significant increase, suggesting early renal involvement or subclinical cardiorenal interactions[15]. This aligns with previous evidence that the coexistence of cardiovascular and renal dysfunction is frequently mediated by systemic inflammation and endothelial damage, particularly in diabetic patients. The interplay between uncontrolled hyperglycemia, dyslipidemia, and inflammatory activation creates a pro-atherogenic and pro-thrombotic state, contributing not only to worsening coronary outcomes but also to multisystem vascular complications including nephropathy and cerebrovascular disease[16].

The results of this study underscore the multifactorial nature of cardiovascular disease in diabetics and reinforce the potential of hs-CRP as a reliable and clinically useful marker for identifying individuals at high risk of adverse cardiovascular events. Elevated hs-CRP levels were consistently associated with worsening CAD severity, independent of traditional risk factors, supporting its incorporation into routine cardiovascular risk stratification algorithms. Given its low cost, widespread availability, and predictive value, hs-CRP may complement traditional clinical and laboratory indicators in the assessment of cardiovascular risk, particularly in diabetic populations undergoing evaluation for coronary artery disease[16,17].

However, while the findings are compelling, the interpretation of hs-CRP must be approached cautiously. As an acute-phase reactant, hs-CRP levels may be influenced by a variety of non-cardiac conditions, such as infections, autoimmune diseases, or trauma[18]. Therefore, it should be interpreted in conjunction with other clinical parameters and after exclusion of confounding comorbidities. Moreover, the cross-sectional nature of this study limits causal inference; it demonstrates a strong association but cannot confirm whether elevated hs-CRP directly contributes to CAD progression or merely reflects ongoing vascular inflammation[14,19].

Nonetheless, the strength of the correlation observed provides a strong rationale for further investigation. Future prospective cohort studies and interventional trials are needed to evaluate the predictive validity of hs-CRP and to explore the

impact of anti-inflammatory therapies in reducing cardiovascular events among high-risk diabetic patients[19]. Integrating hs-CRP into clinical decision-making may ultimately aid in earlier identification of subclinical vascular disease, timely initiation of preventive strategies, and reduction of the overall cardiovascular burden in this vulnerable population[20].

CONCLUSION

This study demonstrated a strong and significant correlation between elevated high-sensitivity C-reactive protein (hs-CRP) levels and the severity of coronary artery disease (CAD) in diabetic patients. As CAD severity increased, so did hs-CRP levels, along with worsening glycemic control, lipid abnormalities, and mild renal function compromise. These findings highlight the pivotal role of systemic inflammation in the progression of atherosclerosis in individuals with type 2 diabetes mellitus. hs-CRP, as a non-invasive, cost-effective, and widely accessible biomarker, may serve as a valuable adjunct for cardiovascular risk stratification in diabetic patients. Early identification of high-risk individuals through inflammatory profiling could facilitate timely interventions and improve long-term cardiovascular outcomes.

Availability of Data and Materials: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests: The authors declare that they have no competing interests.

Funding: No external funding was received for the conduct of this study.

Authors' Contributions: MZAR conceived and designed the study, performed data interpretation, and wrote the manuscript. AS and SAZ contributed to patient enrollment, data collection, and echocardiographic evaluation. TM supervised the methodology and data analysis. AAC contributed to biochemical assessments and quality control. FA assisted in literature review, data entry, and formatting. All authors reviewed, edited, and approved the final manuscript.

Acknowledgements: The authors extend their sincere gratitude to the medical and laboratory staff of Aziz Bhatti Shaheed Teaching Hospital, Gujrat, and Chaudhary Pervaiz Elahi Institute of Cardiology, Wazirabad, for their invaluable support in patient recruitment, data collection, and logistical assistance throughout the study.

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This article may be cited as: Raza MZA, Siddique A, Zia SA, Mohyuddin T, Chahudhary AA, Altaf F: Correlation Between High-Sensitivity C-Reactive Protein (HS-CRP) and Severity of Coronary Artery Disease in Diabetic Patients. Pak J Med Health Sci, 2023;17(11):355-358.