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Inflammation and Cardiovascular Risk: Investigating the Link between CRP and Coronary Artery Disease

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ABSTRACT

Objective: To evaluate the association between C-reactive protein levels and the severity of coronary artery disease in a population of patients. **Methodology:** This was a retrospective observational study involving 220 patients, divided into two groups based on their hs-CRP levels: Group A (<3 mg/L) and Group B (≥3 mg/L). Data was collected from hospital records, including patient demographics, CRP levels, and coronary angiography results. Chi-square and T-tests were used for statistical analysis, with $p < 0.05$ considered significant. **Results:** Of the 220 patients, Group B (hs-CRP ≥3 mg/L) was significantly older (55.1 ± 9.4 years vs. 50.2 ± 11.1 years, $p = 0.0004$). Chi-square test showed a significant correlation between CRP levels and CAD severity ($\chi^2 = 15.21$, $p = 0.0016$), with more patients in Group B presenting with multi-vessel disease (TVD/DVD). T-test for age comparison between CRP groups revealed a significant age difference ($t = -3.59$, $p = 0.0004$). **Conclusion:** Elevated CRP levels are strongly associated with increased CAD severity and older age in patients, supporting CRP as an effective inflammatory marker for predicting cardiovascular risk. These findings highlight the importance of incorporating CRP testing into routine cardiovascular screening.

INTRODUCTION

Cardiovascular Disease (CVD) remains the leading cause of morbidity and mortality worldwide, particularly due to the increasing burden of Coronary Artery Disease (CAD) in both developed and developing countries. While traditional risk factors such as hypertension, diabetes, dyslipidaemia, and smoking are well-established, recent research has shed light on the pivotal role of inflammation in the development and progression of CAD. Among the inflammatory markers, C-Reactive Protein (CRP), especially high-sensitivity CRP (hs-CRP), has emerged as a significant biomarker in assessing cardiovascular risk and guiding therapeutic decisions.[1]

CRP is an acute-phase protein synthesized by the liver in response to interleukin-6 (IL-6) and other cytokines. Its elevated levels are closely associated with systemic inflammation and have been independently linked to adverse cardiovascular outcomes.[2] Studies show that CRP not only reflects the underlying inflammatory state but may also play a direct role in promoting atherosclerosis by affecting endothelial function, plaque stability, and thrombosis.[3]

The relationship between inflammation and CAD has gained considerable attention due to its implications for both prognosis and treatment. In a large-scale analysis, elevated hs-CRP was significantly associated with an increased risk of adverse cardiovascular events, including myocardial infarction and stroke.[4] This finding underscores the potential of CRP as a predictive tool for long-term cardiac outcomes, especially among patients undergoing Percutaneous Coronary Interventions (PCI).

In Pakistan, cardiovascular diseases are a growing public health challenge. A prospective study conducted at Nishtar Medical University, Multan, highlighted the significant predictive value of hs-CRP among women with cardiovascular risk factors.[5] The study emphasized the importance of incorporating hs-CRP into regular cardiovascular screening protocols, especially in resource-limited settings like ours.

Similarly, a study further confirmed CRP's role in cardiovascular pathology, not just as a marker, but also as a potential mediator of disease progression.[6] The findings support the growing hypothesis that CRP may actively contribute to atherosclerotic plaque destabilization, leading to acute coronary syndromes.

High-sensitivity CRP assays are particularly valuable for detecting low-grade inflammation that might be missed by conventional CRP tests. These assays have been shown to correlate well with clinical outcomes and are now recommended as part of risk stratification strategies in cardiovascular medicine.[7]

The significance of hs-CRP in predicting the severity of CAD was further demonstrated in a retrospective study analysing angiographic findings in NSTEMI patients, where higher CRP levels were linked to more complex CAD.[8]

The underlying mechanism connecting CRP and cardiovascular pathology has also been explored at a molecular level. CRP, especially in its monomeric form, may trigger local pro-inflammatory responses and amplify vascular damage, independent of systemic inflammation.[9]

In addition, a Pakistani study explored CRP's prognostic value in chronic heart failure patients and found a strong correlation between elevated CRP and disease severity, emphasizing its utility in risk stratification and management.[10]

Emerging genetic studies have further solidified the causal role of CRP in cardiovascular diseases. A Mendelian randomization study showed a significant association between genetically elevated CRP levels and increased risk of hypertensive heart disease, although the link with other forms of CAD remains under investigation.[11]

The burden of cardiovascular risk due to inflammation is even more significant among individuals with comorbidities such as diabetes and chronic kidney disease. The study found that high CRP levels in patients with CKD and stable CAD were predictive of adverse outcomes after PCI.[12]

A review study emphasized that CRP, particularly hs-CRP, should be a central component of personalized cardiovascular risk management due to its predictive accuracy and potential as a therapeutic target.[13]

The predictive power of CRP extends across different demographic groups. A study demonstrated that CRP is a strong independent risk marker in young female populations, pointing to its universal applicability across age and gender.[14]

At Hayatabad Medical Complex in Peshawar, there is increasing interest in implementing hs-CRP as a routine part of cardiovascular assessment protocols. Considering the local disease burden and resource constraints, integrating cost-effective and reliable inflammatory markers like CRP could enhance early diagnosis and preventive strategies, especially in high-risk populations.

The rationale for this study is rooted in the need to enhance cardiovascular risk stratification through inflammatory biomarkers in our local clinical setting. While global data is robust, there remains a significant knowledge gap in the application of CRP screening for CAD within Pakistani populations. This study, conducted in the Department of Cardiology, Hayatabad Medical Complex, Peshawar, aims to contribute to the local understanding and utility of CRP in cardiovascular risk prediction.

The objective of this study was to evaluate the association between C-reactive protein (CRP) levels and the severity of CAD among patients presenting at Hayatabad Medical Complex, Peshawar.

MATERIALS AND METHODS

Study Design and Setting

This retrospective study was conducted in the Department of Cardiology at Hayatabad Medical Complex, Peshawar, over a one-year period from Jan 2023 to Dec 2023. The study was designed to evaluate the relationship between serum C-reactive protein (CRP) levels and the severity of CAD among patients who were admitted for coronary evaluation, including angiography.

Study Type

The research adopted a retrospective observational approach, based on a review of patient records and laboratory data archived during the specified time period. The study population consisted of adult patients presenting with symptoms suggestive of ischemic heart disease who underwent coronary angiography for diagnostic purposes.

Sample Size Estimation

The sample size was calculated using the WHO sample size calculator. Referring to a prior study conducted by Shahid (2022) at Nishtar Medical University, which reported a prevalence of elevated hs-CRP in 58% of patients with cardiovascular disease, the sample size was estimated at a confidence level of 95%, absolute precision of 7%, and a power of 80%, resulting in a calculated minimum of 190 patients.[5] To improve study strength and account for incomplete data, a total of 220 patients were included in the analysis. These patients were divided into two groups based on CRP levels: Group A included patients with hs-CRP <3 mg/L (n = 110), and Group B included patients with hs-CRP ≥3 mg/L (n = 110).

Inclusion and Exclusion Criteria

Inclusion criteria consisted of adult patients aged 18 years or older of both sexes who were admitted with chest pain, underwent coronary angiography, and had hs-CRP levels measured within 24 hours of admission.

Patients with documented infections, recent trauma or surgery within the past 4 weeks, autoimmune diseases, chronic inflammatory conditions (such as rheumatoid arthritis or lupus), malignancies, and those already on long-term steroid or immunosuppressive therapy were excluded. Additionally, cases with incomplete records or missing hs-CRP data were omitted from the analysis.

Randomization and Blinding

Due to the retrospective nature of the study, randomization and blinding were not applicable.

Data Collection Procedure

Data were collected from electronic hospital records, including demographic details, clinical history, laboratory reports, angiographic findings, and outcomes. hs-CRP values were obtained from hospital laboratory records, measured using immunoturbidimetric assay methods. CAD severity was classified based on angiographic findings as: no disease, single-vessel disease (SVD), double-vessel disease (DVD), or triple-vessel disease (TVD).

Definitions and Variable Assessment

The primary variables included hs-CRP level and angiographic severity of CAD. hs-CRP was defined as low if <3 mg/L and high if ≥3 mg/L, in accordance with American Heart Association guidelines. CAD was assessed through coronary angiography and interpreted by experienced cardiologists. Severity classification was based on the number of major epicardial coronary arteries with ≥50% luminal stenosis.

Statistical Analysis

Data were entered into SPSS version 25 for analysis. Descriptive statistics were used to report means and standard deviations for continuous variables, and frequencies and percentages for categorical variables. Chi-square test was applied to compare categorical variables such as CRP groups and vessel involvement. Independent sample t-tests were used to compare means between groups. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

All ethical considerations were strictly followed. The study received approval from the Ethical and Research Committee of Hayatabad Medical Complex, Peshawar. Since this was a retrospective study based on hospital records, patient anonymity and data confidentiality were ensured by de-identifying patient information prior to analysis. For those patients whose records were reviewed and still undergoing treatment or follow-up, informed consent was obtained as per institutional policy. No animal subjects were involved in this study, and all procedures involving human participants complied with the ethical standards of the responsible committee and the 1964 Helsinki Declaration and its later amendments.

RESULTS

Overview and Patient Count

A total of 220 patients were included in this retrospective study conducted in the Department of Cardiology, Hayatabad Medical Complex, Peshawar, between Jan 2023 and Dec 2023. The study aimed to investigate the association between high-sensitivity C-reactive protein (hs-CRP) levels and the severity of CAD. Patients were divided into two equal groups: Group A (hs-CRP <3 mg/L, n = 110) and Group B (hs-CRP ≥3 mg/L, n = 110), based on CRP stratification criteria defined in the methodology. The overall mean age of participants was 52.8 ± 10.4 years. Males constituted 58.2% (n=128) and females 41.8% (n=92). Table 1 provides a detailed summary of demographic characteristics across CRP groups.

Description of Patient Characteristics

Table 1 shows the demographic and clinical profile of patients. Group B (hs-CRP ≥3 mg/L) had a significantly higher mean age (55.1 ± 9.4 years) compared to Group A (50.2 ± 11.1 years), with a p-value of 0.0004 (Independent t-test). A slight male predominance was observed in both groups. Notably, comorbidities such as diabetes, hypertension, and hyperlipidemia were more frequent in Group B.

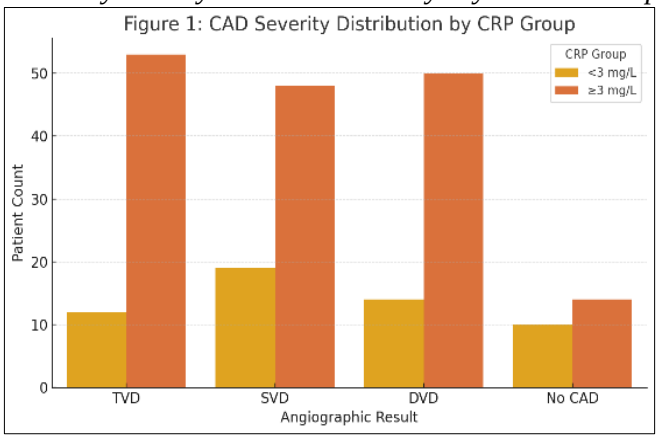
Table 1

Demographic and Clinical Characteristics of Patients (n = 220)

Variable	Group A (CRP <3)	Group B (CRP ≥3)	p-value
Sample Size (n)	110	110	-
Mean Age (years)	50.2 ± 11.1	55.1 ± 9.4	0.0004
Male (%)	59 (53.6%)	69 (62.7%)	0.18
Diabetes Mellitus (%)	39 (35.4%)	58 (52.7%)	0.01
Hypertension (%)	45 (40.9%)	65 (59.1%)	0.005
Hyperlipidaemia (%)	32 (29.1%)	45 (40.9%)	0.07

Figure 1

Coronary Artery Disease Severity by CRP Group



CRP Levels and CAD Severity

A statistically significant association was observed between hs-CRP levels and CAD severity, as assessed by coronary angiography. Chi-square test revealed a strong correlation ($\chi^2 = 15.21$, $p = 0.0016$), with more

patients in Group B presenting with double (DVD) and triple vessel disease (TVD) compared to Group A, which had more cases of single-vessel disease (SVD) and no CAD. Figure 1 illustrates the distribution of angiographic findings across CRP groups, with Group B showing a significantly higher burden of multi-vessel CAD.

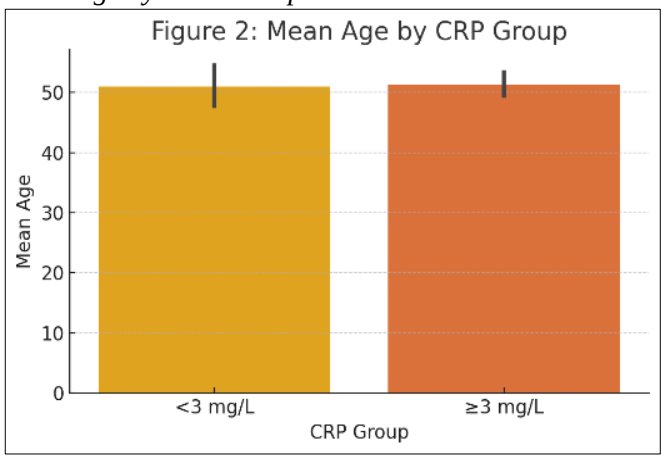
Table 2

CAD Severity by CRP Group

Angiographic Finding	CRP <3 mg/L (n=110)	CRP ≥3 mg/L (n=110)	p-value
No CAD	10 (9.1%)	14 (12.7%)	-
Single Vessel (SVD)	19 (17.3%)	48 (43.6%)	
Double Vessel (DVD)	14 (12.7%)	50 (45.4%)	
Triple Vessel (TVD)	12 (10.9%)	53 (48.2%)	0.0016

Figure 2

Mean Age by CRP Group



Statistical Tests Summary

The statistical analyses revealed significant associations between CRP levels and various clinical outcomes. The Chi-square test showed a strong correlation between elevated CRP levels and more severe CAD ($\chi^2 = 15.21$, $p = 0.0016$). Additionally, the T-test for age comparison between CRP groups demonstrated that patients with higher CRP levels were significantly older ($t = -3.59$, $p = 0.0004$). Further Chi-square tests indicated a significant association between CRP levels and the presence of comorbidities such as diabetes ($\chi^2 = 6.52$, $p = 0.01$) and hypertension ($\chi^2 = 7.81$, $p = 0.005$), supporting CRP as a key inflammatory marker linked to both CAD severity and cardiovascular risk factors.

Table 3

Summary of Key Statistical Tests

Test	Value	p-value	Significance
Chi-square (CRP vs CAD severity)	$\chi^2 = 15.21$	0.0016	Yes
T-test (Age between CRP groups)	t = -3.59	0.0004	Yes
Chi-square (CRP vs Diabetes)	$\chi^2 = 6.52$	0.01	Yes
Chi-square (CRP vs Hypertension)	$\chi^2 = 7.81$	0.005	Yes

DISCUSSION

The operative treatment of thoracolumbar fractures requires a choice by the treating physician as to the optimal approach and means of fixation. Alvine et al and Esses et al both demonstrated good clinical and radiologic outcomes following the open instrumentation of thoracolumbar fractures[11,12]. Advocates of the percutaneous technique cite decreased operative time, decreased blood loss, and decreased disruption of the already traumatized soft tissues. Conversely, opponents of the MIS technique cite the long surgeon learning curve and the possibility of inadequate restoration of VBH and local kyphosis.

In this study, we investigated the effects of MISS for thoracolumbar trauma on patient out- comes. Our patients had an average age of 54.4±14.2 years. Most of our patients were men, and motor vehicle collisions and accidental falls made up 80% of cases. Our patient population was therefore similar to that of Wang et al.'s[13] epidemiological study on traumatic spinal fractures

A systematic review of minimally invasive versus open surgery for the treatment of types B and C thoracolumbar injuries was carried out and found that the MISS can significantly reduce the blood loss, length of hospital stay, and complications; however, the fusion rate and operative time was similar[14]. Another meta-analysis found that the MISS not only reduced the blood loss more than open surgery but also had significantly lower VAS of back pain and ODI scores[15].

In our study, less intra-operative blood loss and shorter operative time has been found in cohort treated with MISS group as compared to the cohort treated with open surgery, however, no significant difference was observed in duration of hospital stay and ODI score.

In 2016, Zhang et al[16] published the results of a prospective randomized clinical trial comparing 29 patients randomly assigned to MISS (type A: 7, type B2: 5, and type C: 17) and 30 who underwent open surgery (type A: 8, type B2: 5, and type C: 17). Follow-up was 12 months. Operative time was longer in the MISS group (218 vs 190 minutes), but this difference was nonsignificant ($P = .165$). Blood loss (302 vs 536 mL, $P = .011$) and length of hospital stay (18.6 ± 10.3 vs 27.5 ± 15 days, $P = .011$) were significantly less in the MISS group.

Grossbach et al.[17] prospectively compared patients with flexion-distraction injuries between May 2003 and March 2013. A total of 38 patients with type B fractures (11 MISS and 27 open surgery) were followed for an average of 13 months (9–18 months). Patients who had undergone MISS had a shorter operative time (195 vs 257 minutes, P = .07) and less blood loss (93 vs 498 mL, P = .003). The other measured parameters (kyphotic angulation correction, length of hospital stay, and neurologic recovery) were no different in the 2 groups.

The studies conducted in the past have analyzed the

retrospective series of the patients with thoracolumbar traumatic spinal injury. Therefore, there may be a significant chance of biasness. We therefore conducted a prospective study to compare the outcomes of open versus minimally invasive spinal surgery for the treatment of thoracolumbar traumatic spinal injury.

Limitations of the study include use of non-probability consecutive sampling technique was used and this may not allow generalization of results to the population. Furthermore, though, the sample size used for study was evident base but still, it was small to establish the minimally invasive spinal surgery choice of technique for the treatment of thoracolumbar traumatic spinal injury, more studies with larger sample size should be conducted.

Furthermore, shorter follow up period and single centered study may question the generalizability of the results. This study explored the role of C-reactive protein (CRP) as an inflammatory marker in predicting CAD severity. Our results indicate a significant association between elevated CRP levels and increased CAD severity, with the high CRP group (≥3 mg/L) showing a predominance of multi-vessel disease, specifically double and triple vessel disease. Moreover, patients with higher CRP levels were found to be older, aligning with the hypothesis that inflammation plays a crucial role in the pathogenesis of atherosclerosis, especially as patients age.

This study adds valuable data to the growing body of evidence on CRP and cardiovascular risk. Previous studies globally have confirmed CRP as a reliable marker for assessing CAD severity, but few have specifically focused on its role in a Pakistani population. Research in this area is still in its early stages within Pakistan, despite international studies recognizing the inflammatory nature of CAD. This is particularly important given the unique genetic and lifestyle factors that may influence CRP levels in Pakistani patients.

Our results align with studies conducted in Europe and the US, where elevated CRP levels are widely acknowledged as independent predictors of CAD severity.[13] Studies have shown that high CRP levels not only reflect inflammation but also actively contribute to atherosclerosis and plaque rupture.[1]

International studies, also demonstrate that CRP is an inflammatory marker that correlates with CAD severity.[15] Additionally, Rosuvastatin has been shown to significantly reduce CRP levels, indicating its potential role in cardiovascular risk management.[16]

While global research, especially in Western countries, has extensively validated CRP's utility in CAD prediction, similar large-scale studies specifically targeting the Pakistani population are sparse. The Pakistani healthcare system faces challenges such as under-reporting of cardiovascular diseases, and thus, this research provides new insights into how CRP could be effectively utilized in local clinical settings for better CAD risk stratification.

In Pakistan, a few studies have examined the relationship between CRP and CAD risk, such as the study by Shahid (2022), which investigated CRP as a cardiovascular risk predictor in women.[5] Other research, highlighted the prevalence of inflammatory markers in patients with heart disease.[17] However, most of these studies focus on general risk factors and do not provide a comprehensive assessment of CRP's role in CAD progression.

CRP is becoming increasingly recognized in Pakistan as a useful marker of systemic inflammation, with studies establishing its relevance in predicting cardiovascular risk.[5,18] However, comprehensive studies linking CRP to CAD severity in the Pakistani context are still limited.

In contrast to our findings, studies in Western populations have consistently shown that hs-CRP levels can predict both short-term and long-term outcomes in patients with acute coronary syndrome (ACS) and stable CAD. A study highlighted how CRP and other inflammatory markers are integrated into predictive risk models for CAD, further enhancing treatment personalization.[1] The JUPITER trial (2008) demonstrated that statins, which reduce CRP, significantly lower the risk of cardiovascular events in patients with elevated CRP but without traditional cardiovascular risk factors.[19]

Our study complements these findings but emphasizes the need for tailored cardiovascular management strategies in South Asian populations, particularly in Pakistan, where lifestyle diseases such as diabetes and hypertension are highly prevalent.

The elevated CRP levels observed in this study serve as an indicator of systemic inflammation, which is linked to advanced atherosclerosis and CAD progression. The higher frequency of multi-vessel CAD in patients with elevated CRP levels supports the idea that inflammation exacerbates vascular injury and may contribute to plaque instability, as suggested by previous studies.[13] The mean age difference between CRP groups further supports the hypothesis that aging and systemic inflammation contribute synergistically to CAD development.

Study Limitations and Future Directions

One of the limitations of this study is its cross-sectional design, which prevents the establishment of causal relationships between CRP and CAD severity. A longitudinal study following patients over time would provide more robust evidence regarding the predictive value of CRP in CAD progression. Additionally, comorbid conditions such as chronic kidney disease and diabetes were self-reported, and their influence on CRP levels could be further explored with more precise biomarkers. Future studies should also stratify patients based on specific ethnic groups and regional factors to better understand the role of inflammation in CAD risk.

CONCLUSION

This study successfully met its objective of exploring the association between C-reactive protein (CRP) levels and CAD severity. The results clearly demonstrate that elevated CRP levels are significantly associated with increased CAD severity, with patients in the high CRP group showing more frequent multi-vessel disease. Additionally, the study highlighted the influence of age and comorbidities such as diabetes and hypertension on CRP levels and CAD progression.

The findings support the conclusion that CRP is a valuable inflammatory marker for predicting CAD risk, particularly in Pakistani populations where this research is underexplored. The study's results underscore the need for inflammatory markers to be integrated into risk stratification and early intervention protocols for CAD management in clinical practice.

Future Recommendations

Further research is recommended, particularly longitudinal studies, to establish causal relationships between CRP and CAD progression. Future work should focus on ethnic-specific risk factors and explore the role of CRP in personalized treatment for CAD patients. Additionally, larger, multi-centre studies can validate these findings and enhance the integration of CRP testing in routine cardiovascular screening.

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