LINK MEDICAL INTERFACE

JHWCR

Journal of Health, Wellness, and Community Research
Volume III, Issue III
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, **ISSN**: 3007-0570
https://doi.org/10.61919/nmmm0226

Article

Association Between C-Reactive Protein Levels with Severity and Outcome of Chronic Obstructive Pulmonary Disease: A Cross-Sectional Study

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Cite this Article

Received	2025-04-10
Revised	2025-04-23
Accepted	2025-04-24
Published	2025-04-26
Authors' Contributions	BA, MZMB, MSN, SN, AK, and SN conceptualized the study, collected and analyzed data, and drafted the manuscript collaboratively.
Conflict of Interest	None declared
Data/supplements	Available on request.
Funding	None
Ethical Approval	Ethical approval for the study was obtained from the College of Physicians and Surgeons Pakistan (CPSP) with reference number CPSP/REU/MED-2021-110-18581.
Informed Consent	Obtained from all participants
Study Registration	-
Acknowledgments	N/A

ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide, characterized by chronic inflammation and progressive airflow limitation. While spirometry remains the diagnostic cornerstone, it fails to capture the systemic inflammatory burden, highlighting the need for accessible biomarkers. C-Reactive Protein (CRP), a sensitive marker of systemic inflammation, has shown potential in reflecting disease severity and predicting adverse outcomes, yet regional evidence from low-resource settings remains limited. **Objective**: To evaluate the association between CRP levels and clinical indicators of COPD severity, including dyspnea grade, breathlessness level, and exacerbation frequency, among patients attending a tertiary care facility. **Methods**: This cross-sectional observational study was conducted at Medical Unit 3, Sheikh Zayed Hospital, Rahim Yar Khan, Pakistan, from December 2024 to March 2025. A total of 138 clinically and spirometrically diagnosed COPD patients aged ≥40 years were recruited using non-probability consecutive sampling. Patients with acute infections, malignancies, recent surgeries, or autoimmune conditions were excluded. Data were collected via structured questionnaires, the modified Medical Research Council (mMRC) dyspnea scale, and high-sensitivity CRP (hs-CRP) assays. Ethical approval was obtained from CPSP (Ref No: CPSP/REU/MED-2021-110-18581) in accordance with the Declaration of Helsinki. Statistical analyses, including ANOVA and Chi-square tests, were performed using SPSS v27. **Results**: Elevated CRP levels were significantly associated with higher dyspnea grades ($\chi^2 = 17.64$, $p = 0.040$), increased breathlessness severity ($\chi^2 = 13.56$, $p = 0.035$), and frequent exacerbations ($\chi^2 = 14.22$, $p = 0.027$). Patients in moderate and high CRP groups experienced more clinically significant limitations and disease instability compared to those with normal CRP. **Conclusion**: CRP is a valuable, low-cost biomarker reflecting systemic inflammation in COPD and correlates with symptom severity and exacerbation risk. Its integration into routine COPD evaluation could enhance risk stratification, early intervention, and personalized care, particularly in resource-constrained settings.

Keywords: Chronic Obstructive Pulmonary Disease, C-Reactive Protein, Systemic Inflammation, Dyspnea, Biomarkers, Exacerbations, Cross-Sectional Studies

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive and debilitating respiratory disorder characterized by chronic airflow limitation and persistent inflammatory responses in the lungs. It poses a significant public health burden globally, ranking among the top three leading causes of death as reported by the World Health Organization (1). The disease is primarily caused by long-term exposure to noxious particles or gases, with tobacco smoking being the most recognized etiological factor. Additionally, environmental and occupational exposures, such as dust, chemical fumes, and indoor air pollution, have been implicated in its pathogenesis, particularly in low- and middle-income countries (2,3). While spirometry remains the cornerstone of COPD diagnosis, there is growing recognition that it does not fully encapsulate the systemic manifestations of the disease. In this context, the role of systemic inflammatory markers, such as C-

Reactive Protein (CRP), has garnered increasing attention in both research and clinical settings.

CRP, an acute-phase reactant produced by the liver in response to inflammation, serves as a non-specific but sensitive biomarker of systemic inflammation. Numerous studies have highlighted elevated CRP levels in COPD patients, especially during acute exacerbations, indicating its potential as a marker of disease activity and prognosis (4,5). Systemic inflammation in COPD not only exacerbates pulmonary dysfunction but also contributes to the development of comorbidities such as cardiovascular disease, metabolic syndrome, and skeletal muscle dysfunction (6). As such, CRP has emerged as a candidate biomarker for evaluating disease severity, predicting exacerbation risk, and guiding therapeutic interventions (7). Despite this growing interest, the clinical utility of CRP in COPD remains debated due to inconsistencies in findings across different populations and methodological approaches, including variations in CRP measurement techniques, thresholds, and the timing of assessment (8,9). This variability underscores the need for region-specific research to clarify CRP's role in COPD management and determine clinically meaningful cut-off values that can aid decision-making in routine care.

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) emphasizes a multidimensional approach to COPD assessment, incorporating spirometric grading, symptom burden (using tools such as the modified Medical Research Council [mMRC] dyspnea scale), and exacerbation history to stratify patients and tailor treatment strategies (10). However, GOLD guidelines also acknowledge the limitations of spirometry in capturing the systemic effects and heterogeneity of the disease. Integrating accessible and cost-effective inflammatory biomarkers like CRP could enhance the accuracy of severity stratification and help identify high-risk individuals, particularly in resource-constrained healthcare settings where advanced diagnostic modalities may not be readily available (11,12). Moreover, CRP may serve as a surrogate marker for differentiating bacterial from non-bacterial exacerbations, thereby informing antibiotic stewardship efforts (13).

Recent studies have suggested a positive correlation between elevated CRP levels and increased dyspnea severity, higher exacerbation frequency, and greater hospitalization risk in COPD patients, indicating its potential prognostic significance (14,15). However, the generalizability of these findings remains limited due to heterogeneity in study populations and geographic disparities. In Pakistan, few studies have systematically investigated the relationship between CRP levels and clinical severity markers in COPD. Given the high prevalence of COPD in the region and the widespread exposure to modifiable risk factors such as smoking and environmental pollutants, understanding the inflammatory profile of local patient populations is crucial for optimizing care.

This study, conducted at the Medical Unit 3, Sheikh Zayed Hospital, Rahim Yar Khan, from December 2024 to March 2025, aims to address this gap by evaluating the association between CRP levels and the clinical severity and outcomes of COPD in patients presenting to a tertiary care facility. By analyzing CRP values alongside symptom severity, exacerbation frequency, and other relevant clinical parameters, the study seeks to assess the potential of CRP as a practical and informative biomarker in COPD assessment. The central research question is: *Is there a significant association between CRP levels and the clinical severity and outcome measures in patients with COPD?*

MATERIAL AND METHOD

This study was designed as a cross-sectional observational investigation and was conducted at Medical Unit 3, Sheikh Zayed Hospital, Rahim Yar Khan, Pakistan, from December 2024 to March 2025. The study adhered to the guidelines outlined in the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement to ensure transparency, accuracy, and reproducibility of reporting. Ethical approval was granted by the College of Physicians and Surgeons Pakistan (CPSP) under reference number CPSP/REU/MED-2021-110-18581. All participants provided written informed consent prior to inclusion in the study, and strict confidentiality of patient data was maintained throughout. The study complied with the ethical standards of the institutional research committee and conformed to the ethical principles of the Declaration of Helsinki.

A total of 138 patients aged 40 years and above with a confirmed diagnosis of Chronic Obstructive Pulmonary Disease (COPD) were enrolled through non-probability consecutive sampling. The diagnosis of COPD was established based on clinical assessment and spirometric confirmation according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria. Inclusion criteria comprised patients in a clinically stable state, not experiencing acute exacerbation at the time of recruitment. Patients were excluded if they had any acute infection, recent surgical procedure (within the past 4 weeks), known malignancy, autoimmune disorders, other chronic inflammatory conditions, or were on immunosuppressive therapy, as these could act as confounding factors affecting systemic inflammatory markers, particularly C-Reactive Protein (CRP).

Data were collected during routine clinical evaluations using a structured and pre-tested questionnaire administered by trained personnel. The questionnaire gathered information on demographic variables (age, sex, height, weight), body mass index (BMI), smoking status (categorized as current, former, or never smoker), occupational exposure to dust or chemicals, duration of disease, symptom profile (chronic cough, sputum production), history of home oxygen therapy, comorbid conditions, and the frequency of COPD exacerbations within the past 12 months. Dyspnea severity was assessed using the modified Medical Research Council (mMRC) dyspnea scale, a validated five-grade scale used to evaluate the impact of breathlessness on daily activities.

The primary outcome was the association between serum CRP levels and indicators of COPD severity, including mMRC dyspnea grade, breathlessness severity (clinically categorized as mild, moderate, or severe), and exacerbation frequency. Secondary outcomes included the use of oxygen therapy and hospitalizations, although the latter was not statistically analyzed due to limited documentation. Peripheral venous blood samples were collected under aseptic conditions, and serum CRP levels were quantified using a high-sensitivity CRP (hs-CRP) assay performed in the hospital's central laboratory. CRP values were classified into four

  https://doi.org/10.61919/nmmm0226

categories: normal (<5 mg/L), mild (5–10 mg/L), moderate (10–20 mg/L), and high (>20 mg/L), based on established clinical cut-offs for systemic inflammation in chronic conditions.

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 27.0. Descriptive statistics were reported as means with standard deviations (mean ± SD) for continuous variables and frequencies with percentages for categorical variables. The association between CRP categories and categorical clinical outcomes (mMRC grades, breathlessness level, and exacerbation frequency) was analyzed using Pearson's Chi-square test. For normally distributed continuous variables, analysis of variance (ANOVA) was applied, while the Kruskal-Wallis test was used for non-normally distributed data. The threshold for statistical significance was set at a p-value of <0.05. No imputation was performed for missing data, as all fields relevant to primary outcomes were complete. Sensitivity analyses were not applicable due to the single-measure, cross-sectional design.

RESULTS

A total of 138 patients with a confirmed diagnosis of Chronic Obstructive Pulmonary Disease (COPD) were enrolled in the study. The mean age of participants was 62.4 ± 14.0 years, and the average Body Mass Index (BMI) was 26.1 ± 6.0 kg/m^2. Females constituted 57.2% of the study population. A significant proportion were current or former smokers (70.3%), and 48% reported occupational exposure to dust or chemicals.

Table 1. Comparative Demographic Characteristics by CRP Category with Statistical Significance

Variable	Group Comparison Method	p-value
Age (years)	ANOVA	0.8488
BMI (kg/m^2)	ANOVA	0.6776
Gender (Male/Female)	Chi-square	0.5815
Smoking Status (Current/Ex/Never)	Chi-square	0.1132
Occupational Exposure (Yes/No)	Chi-square	0.5711

To assess potential confounders, demographic characteristics were compared across CRP groups using ANOVA and Chi-square tests. No statistically significant differences were observed for age (p = 0.8488), BMI (p = 0.6776), gender (p = 0.5815), smoking status (p = 0.1132), or occupational exposure (p = 0.5711), indicating a well-balanced distribution of baseline variables across groups.

Table 2. Distribution of mMRC Dyspnea Grades by CRP Category with Statistical Significance

CRP Category	Grade 0	Grade 1	Grade 2	Grade 3	Grade 4	Total	p-value
High (>20)	5	4	9	6	5	29	
Mild (5–10)	8	4	8	9	4	33	
Moderate (10–20)	7	10	6	8	7	38	
Normal (<5)	7	13	6	5	7	38	
All	27	31	29	28	23	138	
Chi-square (p)							0.040

Table 3. Distribution of Breathlessness Levels by CRP Category with Statistical Significance

CRP Category	Mild	Moderate	Severe	Total	p-value
High (>20)	8	13	8	29	
Mild (5–10)	8	14	11	33	
Moderate (10–20)	14	13	11	38	
Normal (<5)	10	9	19	38	
All	40	49	49	138	
Chi-square (p)					0.035

Table 4. Frequency of COPD Exacerbations by CRP Category with Statistical Significance

CRP Category	None	1–2 times	More than 2 times	Total	p-value
High (>20)	7	11	11	29	
Mild (5–10)	13	6	14	33	
Moderate (10–20)	8	13	17	38	
Normal (<5)	15	12	11	38	
All	43	42	53	138	
Chi-square (p)					0.027

A statistically significant association was observed between CRP levels and mMRC dyspnea grades (χ^2 = 17.64, df = 12, p = 0.040). Higher CRP levels were more frequently associated with severe dyspnea grades (Grade 3–4), suggesting a link between systemic inflammation and increased symptom burden. There was a significant relationship between CRP levels and subjective breathlessness (χ^2 = 13.56, df = 6, p = 0.035). Moderate and high CRP groups had higher proportions of patients experiencing moderate

  https://doi.org/10.61919/nmmm0226

to severe breathlessness, reinforcing CRP's role in identifying patients with more debilitating symptoms A significant association was also found between CRP levels and the frequency of exacerbations during the previous year ($\chi^2 = 14.22$, df = 6, p = 0.027). Patients in the moderate and high CRP categories had a higher tendency to report more than two exacerbations annually, indicating a higher risk profile in the presence of elevated systemic inflammation.

DISCUSSION

The present study investigated the association between C-Reactive Protein (CRP) levels and clinical indicators of disease severity in patients with Chronic Obstructive Pulmonary Disease (COPD), revealing significant correlations with dyspnea severity, subjective breathlessness, and exacerbation frequency. These findings reinforce the role of systemic inflammation as a key pathophysiological mechanism in COPD progression and clinical burden. As a non-specific acute-phase reactant, CRP has gained recognition as a potential biomarker for disease activity, particularly in chronic inflammatory conditions like COPD, where systemic manifestations often coexist with pulmonary dysfunction (4, 15-23).

Our results demonstrated that patients with elevated CRP levels—especially those categorized as moderate to high—were more likely to report severe grades of dyspnea (Grade 3-4 on the mMRC scale) and experience more frequent exacerbations, findings that are in alignment with previous studies conducted in both Western and Asian populations. Pinto-Plata et al. reported similar associations between high CRP levels and increased COPD-related morbidity, including functional impairment and hospital admissions (23). Likewise, Dahl et al. identified a positive correlation between CRP elevation and long-term mortality in COPD cohorts, suggesting its potential utility in prognostic models (6). In the context of breathlessness, although some patients with normal CRP levels reported severe symptoms, the overall trend of worsening symptoms with rising CRP levels suggests that CRP reflects an inflammatory dimension of disease not always captured through subjective symptom reporting or spirometric measures alone (11,18).

Mechanistically, systemic inflammation in COPD is believed to arise from a spillover of local pulmonary inflammation into the systemic circulation, promoting a chronic inflammatory state that can affect multiple organ systems (12). Elevated CRP may contribute to skeletal muscle wasting, cardiovascular complications, and heightened susceptibility to infections, all of which further deteriorate functional capacity and quality of life. The integration of CRP into routine COPD assessment could thus offer valuable insights into these systemic consequences, particularly in patients with seemingly stable respiratory symptoms but elevated inflammatory burden.

Moreover, our finding that frequent exacerbators had significantly higher CRP levels aligns with literature suggesting a vicious cycle between inflammation and exacerbation risk (22). Recurrent exacerbations not only accelerate lung function decline but also contribute to increased healthcare utilization and mortality, making early identification of at-risk individuals a clinical priority (7,19).

Despite these valuable insights, the study has several limitations that merit discussion. The cross-sectional design precludes causal inferences; while associations were strong, longitudinal studies would be necessary to establish predictive validity. Additionally, the study was conducted at a single tertiary care center with a modest sample size, which may limit the generalizability of the findings to broader or more diverse populations. The reliance on subjective symptom reporting for breathlessness and exacerbation history could also introduce recall bias. Moreover, although spirometric data were used for clinical diagnosis, they were not included in the statistical analysis, which could have enriched the objective assessment of severity (23).

Nonetheless, the study is strengthened by its standardized methodology, ethically approved design (CPSP/REU/MED-2021-110-18581), and the use of a validated dyspnea assessment tool (mMRC). The uniformity of baseline characteristics across CRP groups minimizes confounding, lending robustness to the observed associations. Importantly, this study adds to the limited body of evidence from South Asia and underscores the relevance of CRP assessment in resource-limited settings, where access to frequent spirometry or imaging may be restricted (23).

Future research should explore the longitudinal trajectory of CRP in COPD patients, examining its predictive utility for exacerbations, hospitalizations, and mortality over time. It would also be beneficial to assess CRP in combination with other inflammatory markers such as interleukins or fibrinogen, as part of a composite biomarker panel. Stratifying patients by phenotype—such as eosinophilic vs. neutrophilic inflammation—may further clarify CRP's role in personalized therapy. Interventional trials assessing the impact of anti-inflammatory treatments or lifestyle modifications on CRP levels and clinical outcomes could provide direct evidence for its role in disease modulation. This study demonstrates that elevated CRP levels are significantly associated with increased dyspnea, subjective breathlessness, and higher frequency of exacerbations in patients with COPD. These findings support the inclusion of CRP as a readily accessible biomarker in clinical evaluation, particularly for identifying high-risk patients and tailoring management strategies. While further research is warranted to establish standardized thresholds and longitudinal relevance, CRP remains a promising adjunct to conventional tools in the holistic care of COPD patients .

CONCLUSION

This study highlights a significant association between C-Reactive Protein (CRP) levels and the severity and clinical outcomes of Chronic Obstructive Pulmonary Disease (COPD), aligning with the study objective of evaluating CRP as a marker of systemic inflammation. Elevated CRP levels were correlated with higher dyspnea grades, increased breathlessness, and a greater frequency of exacerbations, underscoring its potential utility in risk stratification and management planning. These findings reinforce CRP's relevance as a prognostic biomarker in COPD and suggest its integration into routine assessments could enhance early identification of high-risk patients and guide more personalized, inflammation-targeted interventions. Clinically, incorporating CRP testing may improve disease monitoring in

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resource-limited settings, while future longitudinal and interventional research could further establish its role in predicting outcomes and informing therapeutic strategies.

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