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Diagnostic Accuracy of C-Reactive Protein as a Predictor of Readmission for Acute Exacerbation of Chronic Obstructive Pulmonary Disease

Abdul Salam, Muhammad Younus, Muhammad Zaid Iqbal, Muhammad Usman, Ghulam Mohiuddin, Muhammad Sarfraz Munawar

ABSTRACT

Objective: This study aimed to assess the diagnostic accuracy of CRP as a predictor of readmission for acute exacerbations of chronic obstructive pulmonary disease (AECOPD), with 30-day readmission as the gold standard.

Study design and setting: A prospective observational study was carried out at the Institute of TB and Chest Medicine, Mayo Hospital, Lahore, over six months from April 2025 to September 2025.

Methods: A total of 201 patients aged 40–70 years with a confirmed diagnosis of COPD, as per GOLD 2025 guidelines, and presenting with AECOPD were enrolled using a non-probability consecutive sampling technique. Serum CRP levels were measured on admission using the Spectra 1000 analyzer. Participants were followed for 30 days post-discharge to record readmissions. Data were analyzed using SPSS version 27.0. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and diagnostic accuracy of CRP =15.8 mg/dl for predicting 30-day readmission were calculated.

Results: The mean age of participants was 53.80 ± 8.99 years, with 56.7% males. The mean CRP level was 16.37 ± 19.46 mg/dl. Higher CRP levels were significantly associated with advanced GOLD stages ($p < 0.001$). Overall, 30.8% experienced 30-day readmission. CRP = 15.8 mg/dl predicted readmission with sensitivity 70.9%, specificity 75.5%, and diagnostic accuracy 74.1% ($p < 0.001$).

Conclusion: Elevated CRP is significantly associated with COPD severity and early readmission, suggesting its role as a useful prognostic biomarker for AECOPD management. CRP could serve as a simple, inexpensive biomarker for identifying high-risk patients and guiding post-discharge management to reduce readmissions.

Keywords: Acute disease, Biomarker, C-reative protein (CRP), Chronic obstructive pulmonary disease (COPD)

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a prevalent and treatable condition characterized by a gradual restriction of airflow and damage to lung tissue. It is associated with alterations in lung structure resulting from persistent inflammation and is among the leading causes of morbidity and mortality worldwide.[1] COPD is the 4th leading cause of death worldwide, attributing to 3.5 million deaths in 2021.[2] The burden of disease is particularly higher in low- and middle-income countries (LMICs), where diagnostic limitations, poor air quality, and restricted access to healthcare exacerbate disease outcomes. The prevalence of COPD is reported to be 2.1 % in Pakistan.[3] Tobacco smoking is responsible for more than 70% of COPD cases in high-income nations. In low- and middle-income countries, tobacco smoking contributes to 30–40% of COPD cases, with biomass fuel exposure, household air pollution, occupational dust, and environmental pollutants serving as additional contributors. Continuous exposure to these irritants results in chronic airway inflammation, mucus hypersecretion, and bronchial wall remodeling, ultimately resulting in progressive airway obstruction and reduced

pulmonary compliance.[2]

Acute Exacerbation of COPD (AECOPD) is characterized by the acute worsening of COPD symptoms, such as shortness of breath and cough, beyond normal day-to-day variations, requiring additional management. The prevalence rate of AECOPD among all COPD patients ranges from 22 to 40% each year.[4, 5] AECOPD is associated with a gradual decline in pulmonary function and poor quality of life, and patients often experience more than one episode in a year, leading to frequent in-hospital readmissions. Approximately 63% of patients are readmitted at least once within the 12 months following admission due to an exacerbation, and nearly 79% present with acute hypercapnic respiratory failure.[6] Major risk factors for readmission in AECOPD patients are a history of prior exacerbations, bacterial colonization of the respiratory tract, reduced lung function, and lower levels of physical activity.[7] Early readmission in patients with AECOPD is associated with increased morbidity and adverse patient outcomes and also puts a considerable burden on healthcare resources.[8]

Chronic inflammation is fundamental to the pathogenesis of COPD, and inflammatory markers for their potential application as biomarkers of COPD have been explored.[9] Biomarkers are characteristic substances that are objectively assessed and analyzed to act as indicators of normal biological functions, disease processes, or responses to pharmacological treatments, enhancing the specificity and sensitivity of diagnosing and treating diseases. They had a significant role in the early detection of diseases, assessing disease severity, as well as evaluating treatment efficacy and prognosis.[10]

C-reactive protein (CRP) is an acute-phase protein synthesized by the liver that has been used as a marker of inflammation under a variety of clinical conditions. Its serum concentration rises rapidly following inflammation, infection, or tissue injury, making it a sensitive indicator of systemic inflammation. In COPD, persistently elevated CRP levels reflect ongoing airway and systemic inflammation and tend to increase during exacerbations, having an association with white blood cell count and active infection. Elevated CRP levels have also been linked to increased cardiovascular risk, metabolic dysfunction, and all-cause mortality, underscoring its potential as a prognostic biomarker.[11,12]

Given the low cost and easy availability of CRP represents a significant non-invasive biomarker for identifying patients at high risk of early readmission following an exacerbation. Early recognition of such patients may facilitate regular follow-up, optimization of management, and implementation of comprehensive discharge plans aimed at reducing recurrent hospitalizations. Despite its potential clinical utility, data from Pakistan and other LMICs regarding the diagnostic accuracy of CRP in predicting early readmissions among AECOPD patients remain limited. The rationale of this study is to bridge the gap in the literature and provide clinicians with an inexpensive marker to identify high-risk patients. This study aimed to assess the diagnostic accuracy of CRP as a predictor of readmission for AECOPD, taking 30-day readmission as the gold standard.

METHODOLOGY

A cross-sectional study was conducted at the Institute of TB and Chest Medicine, Mayo Hospital, Lahore, for a duration of 6 months from April 2025 to September 2025. Non-probability consecutive sampling technique was used for the inclusion of the participants presenting at the Department of Emergency and the Institute of TB and Chest Medicine, Mayo Hospital, Lahore.

A sample size of 201 was calculated by using parameters as a confidence interval of 95%, desired precision of 10%, prevalence of 30-day readmission of 42.4%, with specificity and sensitivity of elevated CRP as 74% and 67%, respectively.[13, 14]

Informed consent was acquired from the participants before inclusion in the study. The personal information of the participants, including their names, addresses, and contact details, was collected to maintain participant anonymity. Ethical approval for the study was obtained from the King Edward Medical University, Lahore, with reference number 228/RC/KEMU, dated 25/03/2025.

Participants aged 40 to 70 years, of either gender, with a confirmed diagnosis of COPD according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2025 guidelines, were eligible for inclusion. The GOLD classification categorizes the COPD based on the post-bronchodilator FEV_1 into four categories as GOLD 1 (Mild, FEV_1 = 80% predicted), GOLD 2 (Moderate, FEV1 50-79% predicted), GOLD 3 (Severe, FEV1 30-49% predicted), and GOLD 4 (Very Severe, FEV1 < 30% predicted). The diagnosis was confirmed based on a post-bronchodilator FEV1/FVC ratio < 0.7 and <12% improvement in FEV1 following administration of 400 µg salbutamol via a metered-dose inhaler. Only those presenting with AECOPD, defined as worsening dyspnea, cough, and/or sputum production within less than 14 days requiring intensified treatment, were enrolled. Patients with bronchial asthma, bronchiectasis, interstitial lung disease, active tuberculosis, lung abscess, or malignancy (lung cancer or bronchogenic carcinoma) were excluded to eliminate potential confounders. Pregnant or breastfeeding women were also excluded.[15, 16]

Demographic and clinical data were recorded using a structured proforma. Variables included age, gender, place of residence (urban/rural), smoking status, comorbidities such as diabetes mellitus and hypertension, and GOLD classification of COPD severity. All participants underwent standard clinical evaluation and management as per institutional protocols. At the time of admission, venous blood samples were collected under aseptic conditions for quantitative measurement of serum CRP levels. The analysis

was performed using the Spectra 1000 analyzer, which provides precise quantitative CRP measurement expressed in milligrams per deciliter (mg/dl). Quality control procedures were strictly adhered to, ensuring reliability and reproducibility of laboratory results. After initial treatment and stabilization, all patients were discharged according to institutional discharge criteria and were followed up for 30 days to evaluate hospital readmission due to recurrent exacerbation. Follow-up was conducted through contact and outpatient visits. Readmission was defined as any unplanned hospital admission for respiratory symptoms consistent with COPD exacerbation within 30 days of discharge from the index hospitalization. Patients who did not respond to follow-up calls or failed to attend scheduled visits were considered lost to follow-up and excluded from the outcome analysis.

All data were entered and analyzed using IBM SPSS Statistics version 27.0. Continuous variables such as age and CRP level were expressed as mean ± standard deviation (SD), while categorical variables such as gender, smoking status, comorbidities, GOLD stage, and readmission status were presented as frequencies and percentages. The diagnostic performance of CRP =15.8 mg/dl in predicting 30-day readmission was assessed by calculating sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall diagnostic accuracy. The $p < 0.05$ was set as statistically significant.

RESULTS

The total number of participants was 201, and the mean age was 53.80 ± 8.99 years. The study population consisted of 114 males (56.7%) and 87 females (43.3%). Regarding place of residence, 103 (51.3%) were urban residents, while 98 (48.7%) resided in rural areas. With respect to smoking status, 71 participants (35.3%) reported active or previous smoking, whereas 130 (64.7%) were non-smokers. The most prevalent comorbid conditions were hypertension (38.8%) and diabetes mellitus (34.3%). (Table 1). The mean serum CRP level among all participants was 16.37 ± 19.46 mg/dl. According to the GOLD classification, 75 (37.3%) of patients were in Stage I, 46 (22.9%) in Stage II, 32 (15.9%) in Stage III, and 48 (23.9%) in Stage IV. 62 (30.8%) of patients were readmitted within 30 days of discharge. (Table 2). Patients categorized under GOLD Stage I exhibited the lowest mean CRP concentration (9.18 ± 10.69 mg/dl), reflecting relatively mild inflammatory activity corresponding with early-stage disease and better-preserved lung function. In contrast, CRP levels increased progressively with disease severity, rising to 13.83 ± 16.05 mg/dl in Stage II, 18.76 ± 17.10 mg/dl in Stage III, and peaking at 28.43 ± 27.41 mg/dl in Stage IV, which represents the most advanced stage characterized by severe airflow limitation and frequent exacerbations. The observed difference in CRP levels among the GOLD categories was statistically significant ($p < 0.001$). (Table 3). Among patients readmitted in 30 days, 44 (71.0%) had elevated CRP levels, whereas only 34 (24.5%) of patients

Table 1: Demographic Characteristics of the participants

Variables	Frequency (Percentage)
Age (Years) (Mean ± S.D.)	53.80 ± 8.99
Gender	
Male	114 (56.7%)
Female	87 (43.3%)
Residence	
Rural	98 (48.7%)
Urban	103 (51.3%)
Smoking	
Yes	71 (35.3%)
No	130 (64.7%)
Comorbidities	
Diabetes mellitus	69 (34.3%)
Hypertension	78 (38.8%)

Table 2: Clinical Factors of Study Participants

Variables	Frequency (Percentage)
GOLD Classification	
Stage I	75 (37.3%)
Stage II	46 (22.9%)
Stage III	32 (15.9%)
Stage IV	48 (23.9%)
CRP level (Mean ± S.D.)	16.37 ± 19.46
30 Day Readmission	
Yes	62 (30.8%)
No	139 (69.2%)

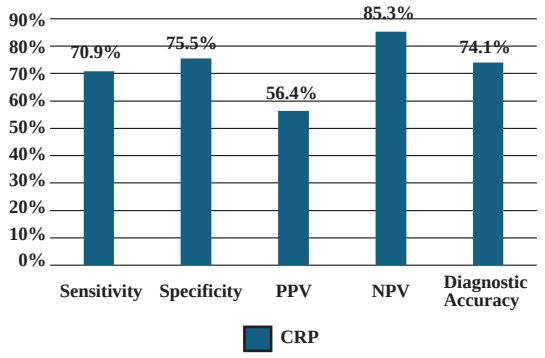
Table 3: Comparison of GOLD stage with CRP level

GOLD Classification	CRP level	*p*-value
Stage I	9.18 ± 10.69	<0.001
Stage II	13.83 ± 16.05	
Stage III	18.76 ± 17.10	
Stage IV	28.43 ± 27.41	

Table 4: Sensitivity, Specificity, PPV, NPV, and Diagnostic Accuracy of CRP

CRP level (mg/dl)	30 Day Readmission		Sensitivity	Specificity	PPV	NPV	Diagnostic Accuracy	*p*-value
	No	Yes						
<15.8	105 (75.5%)	18 (29.0%)	70.9%	75.5%	56.4%	85.3%	74.1%	<0.001
=15.8	34 (24.5%)	44 (71.0%)						

Figure 1: Sensitivity, Specificity, PPV, NPV, and Diagnostic Accuracy of CRP



with high CRP were not readmitted. Conversely, patients with CRP <15.8 mg/dl had a lower likelihood of readmission 18 (29.0%), indicating a strong association between high CRP and early hospital readmission ($p < 0.001$). The sensitivity of CRP =15.8 mg/dl for predicting 30-day readmission was 70.9%, while the specificity was 75.5%, demonstrating good discriminatory ability. The positive predictive value (PPV) was 56.4%, and the negative predictive value (NPV) was 85.3%, indicating that patients with normal CRP levels were significantly less likely to experience readmission. The overall diagnostic accuracy of CRP as a predictor of readmission was 74.1%. (Table 4)

DISCUSSION

This prospective observational study evaluated the role of C-reactive protein (CRP) as a predictor of 30-day hospital readmission among patients admitted with acute exacerbation of chronic obstructive pulmonary disease (AECOPD). The findings demonstrated a significant association between elevated CRP levels and both the severity of COPD, as classified by the GOLD staging system, and the likelihood of early readmission following discharge. Patients in advanced disease stages exhibited higher mean CRP concentrations, indicating greater systemic inflammation. Importantly, a CRP threshold of =15.8 mg/dl predicted 30-day readmission with a sensitivity of 70.9%, specificity of 75.5%, and an overall diagnostic accuracy of 74.1%, suggesting that CRP could serve as a simple, inexpensive biomarker for early risk stratification in clinical practice.

Patients with stage IV COPD had the highest mean CRP levels (28.43 ± 27.41 mg/dl), indicating a direct association between disease severity and systemic inflammation. Hassan and Jabbar had also found that advanced stages of COPD were associated with high levels of CRP.[17] Nuñez et.al reported that higher levels of CRP were present in COPD patients with recurrent episodes of exacerbations ($p < 0.01$) and GOLD group D ($p < 0.05$) than their counterparts.[18] COPD is characterized by chronic inflammation of the airways and lung parenchyma, which extends systemically, contributing to a state of low-grade chronic inflammation. CRP, an acute-phase reactant synthesized by hepatocytes under the influence of interleukin-6 (IL-6), serves as a key marker of this systemic inflammatory response. During an exacerbation, heightened airway inflammation caused by bacterial or viral infection leads to increased release of cytokines, which in turn stimulate hepatic CRP production. The elevated CRP levels observed in this study thus likely reflect the systemic inflammatory response accompanying acute exacerbations. The progressive increase in CRP across GOLD stages further supports the concept that systemic inflammation intensifies with advancing disease severity. Chronic inflammation contributes to airway remodeling, endothelial dysfunction, and skeletal muscle wasting, which collectively impair respiratory function and increase susceptibility to recurrent exacerbations. Consequently, persistently elevated CRP may represent not only acute disease activity but also the cumulative inflammatory burden contributing to disease progression.[19]

The ability of CRP = 15.8 mg/dl to predict 30-day readmission exhibited a sensitivity of 70.9% and a specificity of 75.5%, yielding a diagnostic accuracy of 74.1%. Yogesh et al reported the diagnostic accuracy of raised CRP in predicting 30-day readmission among patients diagnosed with AECOPD, and the raised CRP had 67% sensitivity and 74% specificity in predicting 30-day readmission, with an area under the curve value of 0.72.[14] Gandhi et al also reported that raised CRP had 62% sensitivity and 67% specificity in predicting repeated hospitalization with an area under the curve value of 0.69 in predicting 30-day readmission among patients diagnosed with AECOPD. High CRP level had a sensitivity and specificity of 62% and 67% for predicting prolonged hospital stay.[20] Ellingsen et. al found that after adjustment for clinically relevant confounders, levels of CRP = 5 mg/L, fibrinogen = 3.5 g/L, and WBC > 9 × 109 cells/L measured during stable-phase COPD were associated with a higher frequency of AECOPDs during a follow-up period.[21] Milenkovic et. al had discussed the role of CRP in predicting the in-hospital mortality. The cutoff value of CRP was 81 mg/L (Sn 60.7%, Sp 60%) for predicting in-hospital mortality, having statistical significance.[22] CRP levels correlate positively with the frequency of exacerbations and the need for hospital-based management, with higher CRP cut-offs predicting readmissions within 30 days of discharge. From a clinical standpoint, the measurement of CRP provides an objective, rapid, and cost-effective tool for identifying high-risk patients who may benefit from intensified monitoring, optimized pharmacotherapy, and structured post-discharge care. The negative predictive value of normal CRP levels is also clinically meaningful, as it can help reassure clinicians that patients with low systemic inflammation have a lower likelihood of short-term readmission. Furthermore, integrating CRP assessment with established clinical indices—such as GOLD classification, comorbidity profile, and functional parameters—can enhance the accuracy of readmission risk stratification. In resource-limited settings, where

comprehensive inflammatory profiling is often impractical, CRP serves as a practical and affordable biomarker that bridges laboratory simplicity with prognostic significance. Ultimately, incorporating CRP measurement into discharge planning protocols could enable early intervention strategies, reduce avoidable readmissions, and improve overall outcomes in patients with COPD.[6, 22]

In addition to CRP, several other inflammatory markers have been extensively studied for their potential to predict hospital readmission and adverse outcomes in patients with AECOPD. These biomarkers provide valuable insights into the underlying systemic inflammation, immune activation, and tissue injury that drive disease progression and recurrent exacerbations. Among these, fibrinogen, procalcitonin (PCT), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-á), and leukocyte counts have shown significant clinical relevance in assessing the risk of readmission and prognosis.[19] Fibrinogen is a plasma glycoprotein synthesized by the liver and an acute-phase reactant that increases during systemic inflammation. Elevated fibrinogen levels are associated with enhanced blood viscosity, endothelial dysfunction, and thrombotic risk, all of which contribute to disease exacerbation and cardiovascular comorbidity in COPD. PCT, a precursor of the hormone calcitonin, rises sharply in response to bacterial infections, distinguishing bacterial from non-bacterial exacerbations. Elevated PCT levels at admission have been linked to an increased likelihood of treatment failure and early relapse, leading to hospital readmission. PCT-guided antibiotic therapy has also been shown to reduce antibiotic overuse without compromising patient safety, underscoring its potential role in managing infectious exacerbations and preventing recurrence. Cytokines such as IL-6 and TNF-á play central roles in orchestrating systemic inflammation in COPD. Elevated IL-6 levels are associated with increased CRP synthesis, heightened inflammatory burden, and worse pulmonary function. Similarly, TNF-á contributes to muscle wasting, weight loss, and systemic inflammation, all of which exacerbate disease severity and increase readmission risk. Persistent elevation of these cytokines after discharge indicates incomplete resolution of inflammation and predicts poor recovery and early relapse.[16] Additionally, TLC and NLR have emerged as simple, cost-effective markers of systemic inflammation. Higher NLR values reflect immune dysregulation and have been independently associated with severe exacerbations and early readmission. Collectively, these inflammatory markers, when evaluated alongside CRP, offer a more comprehensive assessment of inflammatory status, enabling clinicians to better predict hospital readmission, personalize treatment, and improve long-term management strategies for AECOPD patients.[13]

CRP standalone is not a sufficient predictor of readmission, as evidenced by the positive predictive value of 56.4%. Consequently, its utilization should ideally be integrated with clinical variables, comorbidities, and functional parameters to enhance the accuracy of predictions. Advanced age, male gender, and elevated BMI are linked to an increased likelihood of readmission. Additionally, more frequent acute exacerbations and an extended duration of the disease have demonstrated strong associations with a heightened risk. Regarding the severity of COPD, a higher GOLD classification, the existence of cardiovascular comorbidities, and a reduced FEV1 are significantly correlated with an increased risk of readmission. (23) Predictive models of readmission for COPD patients had been introduced as ADO (age, dyspnoea, airflow obstruction), BODEX (BMI, airflow obstruction, dyspnoea, exacerbation), DOSE (dyspnoea, obstruction, smoking, exacerbation), and PEARL (previous admissions, extended Medical Research Council dyspnoea score, age, right-sided heart failure, left-sided heart failure) which could assist clinicians in developing treatment strategies specifically targeting those at high risk of hospitalization and readmissions.[24]

The strengths of this study include its prospective design, well-defined inclusion criteria, and uniform data collection using standardized procedures. The use of quantitative CRP measurement on a validated platform (Spectra 1000 analyzer) ensured reliable laboratory results. Moreover, the 30-day follow-up period was chosen to reflect clinically relevant readmission outcomes, consistent with international benchmarks for COPD management and hospital performance assessment.

Limitations: Several limitations should be acknowledged. First, the use of a non-probability consecutive sampling method may have introduced selection bias, potentially limiting generalizability to broader populations. Second, as a single-center study, external validity may be constrained by institutional protocols and patient demographics unique to the study setting. Third, CRP was measured only once at admission, without serial assessment during hospitalization or post-discharge, which could have provided additional insights into temporal trends and prognostic value. Moreover, potential confounding variables, such as medication adherence, environmental exposure, socioeconomic status, and severity of comorbidities, were not fully controlled. Lastly, the study did not compare CRP with other inflammatory biomarkers, such as fibrinogen or procalcitonin, which may complement its predictive capability.

CONCLUSION

CRP levels are significantly associated with greater disease severity and an increased likelihood of 30-day hospital readmission among patients admitted with AECOPD. The progressive rise in CRP concentrations across GOLD stages underscores the central role of systemic inflammation in COPD pathogenesis and its prognostic relevance in exacerbation outcomes. A CRP threshold of =15.8 mg/dl exhibited good diagnostic performance, with satisfactory

sensitivity, specificity, and overall accuracy, indicating its potential utility as a simple, inexpensive, and readily accessible biomarker for identifying patients at high risk of early readmission. Incorporating CRP measurement into clinical assessment at the time of admission could facilitate early risk stratification, guide post-discharge planning, and optimize the use of healthcare resources through targeted follow-up and intervention. However, CRP alone should not be considered a definitive predictor; rather, it should be integrated with established clinical indices and comorbidity profiles to enhance predictive accuracy. Future research involving larger, multicenter, and longitudinal studies is warranted to validate these findings, explore the prognostic value of serial CRP monitoring, and evaluate CRP-guided management strategies. Such approaches could strengthen clinical decision-making, improve patient outcomes, and reduce the healthcare burden associated with recurrent hospitalizations in COPD.

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Authors Contribution:
Abdul Salam: Conception and design of study, data collection, drafting of manuscript, final approval
Muhammad Younus: Study design, supervision, methodology refinement, critical review of manuscript
Muhammad Zaid Iqbal: Data acquisition, literature review, initial drafting of sections
Muhammad Usman: Methodology guidance, interpretation of results, critical revision, overall supervision
Ghulam Mohiuddin: Data entry, statistical analysis, preparation of tables/figures
Muhammad Sarfraz Munawar: Data interpretation, manuscript editing, final draft review

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