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A Prospective Observational Study to Estimate the Diagnostic Accuracy of Neutrophil Lymphocyte Ratio in Predicting in Hospital Outcomes of Chronic Obstructive Pulmonary Disease

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality worldwide. Acute exacerbations are associated with substantial in-hospital mortality and healthcare utilization. Simple, inexpensive biomarkers that predict short-term outcomes at admission may support early risk stratification and informed clinical decision-making. The neutrophil–lymphocyte ratio (NLR) has emerged as a potential inflammatory marker in this context.
Objectives: To estimate the diagnostic accuracy of NLR in predicting in-hospital outcomes among COPD patients and to compare its performance with other routinely available hematological and biochemical parameters.
Methods: This hospital-based prospective observational study was conducted in the Department of General Medicine, Trivandrum Medical College, and included 95 consecutively recruited adults admitted with spirometry-confirmed COPD (post-bronchodilator $FEV_1/FVC < 0.7$, GOLD 2023). Demographic, clinical, hematological (including NLR, TLC, platelets, ESR), CRP, and arterial blood gas parameters were recorded at admission. Outcomes assessed were in-hospital mortality, ICU admission, need for non-invasive and invasive ventilation, and length of stay. Receiver operating characteristic (ROC) analysis was performed to evaluate the diagnostic accuracy of NLR, TLC, CRP, platelets and hemoglobin; survivor versus non-survivor groups were compared using appropriate statistical tests.
Results: Ninety-four patients were analysed (mean age 63.16 ± 9.52 years; 67% male; 53.2% current smokers). The mean duration of COPD was 9.28 ± 3.88 years; 25.5% required ICU admission, 24.5% non-invasive ventilation, 13.8% invasive ventilation, and in-hospital mortality was 13.8%. Mean NLR was 8.46 ± 4.65. ROC analysis showed AUCs of 0.779 for TLC, 0.752 for CRP, and 0.677 for NLR, while hemoglobin and platelets had limited predictive value. Non-survivors had significantly different gas exchange parameters and oxygen requirement; NLR was statistically different between survivors and non-survivors, though with an inverse pattern in this cohort.
Conclusion: NLR demonstrated moderate diagnostic accuracy for adverse in-hospital outcomes in COPD and, together with TLC, CRP and ABG indices, may aid early prognostic assessment. However, its interpretation should be contextual and supported by clinical and biochemical correlates.

Keywords: *Chronic obstructive pulmonary disease; neutrophil–lymphocyte ratio; C-reactive protein; acute exacerbation; in-hospital mortality; systemic inflammation.*

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is one of the most prevalent chronic respiratory disorders worldwide and remains a major cause of morbidity, mortality, and healthcare utilization. It is characterized by persistent respiratory

symptoms and airflow limitation that result from abnormalities in the airways and alveoli, usually caused by significant exposure to noxious gases or particles. The condition is now recognized as a common chronic inflammatory airway disease, and its clinical manifestations progressively worsen over time, affecting quality of life and survival outcomes in affected individuals [1]. COPD has emerged as a major public health concern, particularly in low- and middle-income countries, where environmental exposures and delayed diagnosis contribute to worse outcomes.

Globally, COPD is currently ranked among the leading causes of death, and projections suggest that it may become the third most common cause of mortality in the coming decades. In India, the burden of COPD is particularly substantial, with epidemiological reports estimating a population prevalence of approximately 7.0% among adults over the age of thirty years [2]. This significant disease load contributes heavily to outpatient visits, emergency hospital admissions, and long-term disability. The public health impact is amplified by social determinants such as smoking, industrial pollution, biomass fuel exposure, and poor access to early therapeutic interventions.

A key clinical challenge in COPD management is the occurrence of acute exacerbations of COPD (AECOPD). These events are defined as acute and sustained worsening of the patient's respiratory symptoms beyond normal day-to-day variation, necessitating a change in regular medication or medical intervention [3]. The onset is typically rapid and is associated with increased airway inflammation, airflow restriction, and systemic inflammatory response. AECOPD episodes significantly alter disease trajectory and are widely recognized as critical events that negatively influence long-term prognosis.

Exacerbations play an independent and pivotal role in determining the clinical course of COPD. Evidence demonstrates that AECOPD episodes increase the likelihood of future exacerbations, accelerate the progressive decline in lung function, worsen functional capacity and health-related quality of life, and substantially increase the risk of death [4]. Additionally, AECOPD poses enormous economic challenges to both healthcare systems and families due to direct treatment costs, frequent hospital admissions, intensive care utilization, and indirect loss of productivity.

The frequency of exacerbations varies among patients. Approximately 22–40% of individuals with COPD experience at least one exacerbation annually, and 9–16% experience more than one episode per year [5]. Identifying patients at high risk and predicting adverse clinical outcomes at the time of hospital presentation are therefore vital for optimizing patient care. Early recognition not only facilitates timely clinical decisions and appropriate resource allocation but also enhances personalized treatment strategies aimed at reducing mortality and preventing future exacerbations.

Given the prognostic significance of acute exacerbations, clinicians and researchers have shown increasing interest in identifying simple, reliable, and cost-effective biomarkers capable of predicting disease severity, length of hospital stay, need for ventilatory support, and short-term outcomes. Biomarkers that can be measured immediately at admission hold particular value, as they may support rapid triage, risk stratification, and targeted clinical decision-making [6].

AECOPD is characterized by heightened systemic and airway inflammation, which contributes to clinical deterioration and may necessitate aggressive treatment, including oxygen therapy, antibiotics, bronchodilators, corticosteroids, and in severe cases, ventilatory support [7]. Numerous inflammatory biomarkers—such as C-reactive protein (CRP), fibrinogen, circulating cytokines, and leukocyte differentiation indices—have been evaluated for their potential role in predicting exacerbation severity and outcomes. However, several epidemiological studies indicate that these biomarkers do not provide significant incremental prognostic value beyond traditional clinical predictors such as exacerbation history and comorbidity burden [8].

Despite this limitation, emerging evidence supports the use of combined or composite inflammatory indicators to improve diagnostic and prognostic accuracy. For example, studies have shown that a combination of elevated CRP levels, increased neutrophil count, and clinical signs of labored breathing effectively differentiate patients with AECOPD from those with stable disease [9]. This highlights a growing trend toward incorporating inflammation-based hematological parameters into routine COPD assessment.

Among the various hematological indices being explored, ratios derived from complete blood count (CBC) parameters have gained substantial interest due to their simplicity, availability, and low cost. Two such commonly studied indices are the neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio (PLR). These ratios have demonstrated prognostic significance across diverse disease conditions, including solid organ malignancies, autoimmune disorders such as systemic lupus erythematosus, cardiovascular diseases including coronary artery disease, retinal vascular disorders, chronic kidney disease, and stable COPD [10].

The neutrophil-to-lymphocyte ratio (NLR) represents the balance between innate immune activation and adaptive immune suppression, thereby serving as an integrated indicator of systemic inflammatory response. Neutrophils are key mediators of acute inflammation and release pro-inflammatory cytokines, proteases, and reactive oxygen species that contribute to airway damage in COPD. In contrast, declining lymphocyte counts reflect physiological stress and impaired immune

regulation. An elevated NLR thus suggests pronounced inflammation and immune dysregulation, which are hallmarks of severe AECOPD [11]. Because NLR can be calculated from routine hematological investigations available at the time of patient admission, it offers a clinically meaningful and practical tool for early risk assessment.

Existing research has increasingly linked elevated NLR values with poor outcomes in conditions such as malignant tumors, acute myocardial infarction, pulmonary embolism, and community-acquired pneumonia. Its prognostic role is gaining recognition in respiratory diseases, particularly conditions characterized by heightened systemic inflammation. In the context of COPD, investigating NLR as a biomarker may support early prediction of disease trajectory during hospitalization, thereby enabling effective patient triage, determining the need for intensive monitoring, and improving short-term clinical outcomes. Further research exploring its diagnostic accuracy in predicting in-hospital outcomes among COPD patients may support its integration into risk-stratification models and routine clinical management.

MATERIALS AND METHODS

Study Design and Setting:

This was a hospital-based prospective observational study conducted in the Department of General Medicine at Trivandrum Medical College.

Study Population:

The study included adult patients admitted with a confirmed diagnosis of chronic obstructive pulmonary disease (COPD). Diagnosis was based on pulmonary function test criteria, with a post-bronchodilator FEV_1/FVC ratio of <0.7, as defined by the GOLD 2023 guidelines. Patients were classified into severity groups using the GOLD ABE assessment framework, which categorizes patients based on symptoms and exacerbation history. Group A included patients with low risk (0–1 exacerbations/year not requiring hospitalization) and fewer symptoms (mMRC 0-1 or CAT <10). Group B included those with low risk but higher symptoms (mMRC ≥2 or CAT ≥10). Group E included patients at high risk (≥2 exacerbations/year or ≥1 hospitalization), irrespective of symptom burden.

Eligibility Criteria:

Patients aged 18 years and above with documented COPD were eligible for inclusion. Individuals were excluded if COPD was not the primary reason for admission or if they were admitted due to other acute events such as acute pulmonary edema. Patients with alternative diagnoses, end-stage illnesses, or medical conditions known to elevate neutrophil-to-lymphocyte ratio (NLR)—including malignancy, recent trauma, post-surgical status, myocardial infarction, stroke, or atherosclerosis—were also excluded.

Sample Size and Sampling Technique: The sample size was calculated using the formula,

$$N = \frac{4Sp(1 - Sp)}{d^2(1 - P)}$$

Where specificity (Sp) of NLR for predicting mortality in COPD was 69.17% based on Yao CY et al., [12] mortality prevalence (P) was 12.21%, and allowable error (d) was 10%. The calculated sample size was 94.99; therefore, a minimum of 95 participants was recruited. Eligible patients were selected using consecutive sampling until the sample size requirement was met.

Study Tools and Variables:

Data were collected using an interviewer-administered semi-structured proforma. Independent variables included demographic characteristics (age, sex, smoking status in pack years, comorbidities), history of COPD (duration, GOLD staging, presenting symptoms, symptom duration), routine hematological markers (hemoglobin, TLC, platelet count, neutrophils, lymphocytes, ESR), serum CRP, vital signs, systemic examination findings, arterial blood gas values, and chest imaging.

Outcome variables included clinical endpoints such as in-hospital mortality, ICU admission, requirement of invasive or non-invasive ventilation, pulmonary hypertension, length of hospital stay, and prediction accuracy of hematological markers (TLC, neutrophils, NLR, ESR, CRP).

Study Procedure:

The study protocol received approval from the Institutional Ethics Committee before commencement. Eligible participants or their legal representatives were approached, and written informed consent was obtained after explaining study objectives and procedures. Clinical evaluation included detailed history taking, general physical examination, and systemic assessment. Laboratory investigations such as complete blood count, CRP, ECG, chest X-ray, and arterial blood gas analysis were performed at admission before initiation of antibiotic therapy. Pulmonary function test results and treatment details were recorded. Patients were managed according to standard clinical guidelines without any alteration to treatment due to study participation. Follow-up continued throughout hospitalization until a defined outcome was recorded.

Data Analysis:
Quantitative variables were summarized as mean ±standard deviation (SD) or median with interquartile range (IQR), while categorical variables were expressed as frequencies and percentages. Receiver operating characteristic (ROC) curve analysis was used to determine the diagnostic accuracy, sensitivity, and specificity of hematological markers (TLC, neutrophil count, NLR, ESR, and CRP) in predicting outcomes. The optimal cutoff value was determined from the ROC curve where sensitivity equaled specificity. Comparisons between survivor and non-survivor groups were made using the chi-square test for categorical variables and independent t-tests for continuous variables. Correlations between NLR/PLR and CRP were assessed using Pearson's correlation test. A p-value <0.05 was considered statistically significant. Data processing and statistical analysis were performed using Microsoft Excel 2019 and SPSS version 26 (IBM Corp., Armonk, NY, USA).

Ethical Considerations:
The study adhered to ethical principles, with no interference in routine clinical management. Confidentiality of participant information was strictly maintained, and collected data were used solely for research purposes.

RESULTS
A total of 94 patients with COPD were included in the analysis. Baseline demographic and smoking-related characteristics are summarized in Table 1. The cohort was predominantly older adults, with a higher proportion of males and a majority being current smokers.

Table 1. Baseline demographic and smoking characteristics of the study participants (n = 94)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	Mean ± SD: 63.16 ± 9.52		
Gender	Male	63	67.0
	Female	31	33.0
Smoking status	Smoker	50	53.2
	Ex-smoker	12	12.8
	Non-smoker	32	34.0

Clinical characteristics at admission and hospitalization outcomes are summarized in Table 2. Most patients were conscious at presentation, and a substantial proportion required supplemental oxygen. A quarter required ICU admission, and a minority required invasive mechanical ventilation. In-hospital mortality occurred in a subset of patients.

Table 2. Clinical and hospitalization characteristics of COPD patients (n = 94)

Variable	Category	Frequency (n)	Percentage (%)
Duration of COPD (years)	Mean ± SD: 9.28 ± 3.88		
Level of consciousness	Conscious	74	78.7
	Drowsy	20	21.3
Oxygen (O_2) requirement	Yes	63	67.0
	No	31	33.0
Days of O_2 required	Mean ± SD: 2.30 ± 1.98		
Length of hospital stay (days)	Mean ± SD: 4.05 ± 1.61		
ICU admission	Yes	24	25.5
	No	70	74.5
Non-invasive mechanical ventilation	Yes	23	24.5
	No	71	75.5
Invasive mechanical ventilation	Yes	13	13.8
	No	81	86.2
In-hospital mortality	Yes	13	13.8
	No	81	86.2

Baseline laboratory and arterial blood gas (ABG) parameters at admission are presented in Table 3. Overall, inflammatory markers were elevated, and ABG values reflected respiratory acidosis with variable degrees of compensation.

Table 3. Laboratory and arterial blood gas parameters at admission (n = 94)

Parameter	Mean	Standard deviation (SD)
Hemoglobin (g/dL)	12.85	1.16
Total leukocyte count (cells/mm³)	12,475	4,855.7
Platelet count (lakhs/µL)	2.96	0.95
C-reactive protein (mg/L)	45.66	38.16
ABG pH	7.21	0.14
PCO_2 (mmHg)	74.78	28.67
HCO_3^- (mEq/L)	34.84	8.13
Neutrophil–lymphocyte ratio (NLR)	8.46	4.65

Diagnostic Accuracy of Hematological Parameters

The diagnostic performance of hematological and inflammatory markers in predicting adverse hospital outcomes was evaluated using ROC analysis. The area under the curve (AUC), standard error, significance, and 95% confidence intervals are shown in Table 4. TLC and CRP showed good discriminatory ability, while NLR demonstrated moderate diagnostic accuracy. Hemoglobin and platelet count had limited predictive value.

Table 4. Diagnostic accuracy of laboratory parameters for adverse hospital outcomes (ROC analysis)

Parameter	AUC	Std. Error	p-value	95% CI – Lower	95% CI – Upper
Hemoglobin (Hb)	0.439	0.079	0.480	0.284	0.593
Total leukocyte count (TLC)	0.779	0.064	**0.001**	0.654	0.904
Platelets	0.637	0.090	0.115	0.460	0.814
C-reactive protein (CRP)	0.752	0.053	**0.004**	0.649	0.855
Neutrophil–lymphocyte ratio (NLR)	0.677	0.072	**0.041**	0.537	0.817

A comparison of clinical and laboratory parameters between survivors and non-survivors is presented in Table 5. Several parameters differed between groups, particularly gas exchange variables, oxygen requirement, and NLR.

Table 5. Comparison of laboratory and clinical parameters between survivors and non-survivors

Variable	Survivors (n = 81) Mean (SD)	Non-survivors (n = 13) Mean (SD)	p-value
TLC (cells/mm³)	12,178.39 (4,875.01)	14,323.07 (4,471.97)	1.00
Platelets (lakhs/µL)	2.97 (0.94)	2.92 (1.01)	0.660
Neutrophils (%)	80.11 (10.32)	83.84 (7.15)	0.158
Lymphocytes (%)	14.39 (9.13)	9.38 (5.67)	0.069
CRP (mg/L)	42.90 (38.02)	62.92 (35.72)	0.865
ABG pH	7.26 (0.13)	7.12 (0.12)	0.99
PCO_2 (mmHg)	65.68 (23.00)	92.30 (31.06)	0.20
HCO_3^- (mEq/L)	32.88 (7.21)	38.61 (8.75)	0.40
Oxygen requirement (0 = no, 1 = yes)	0.61 (0.48)	1.00 (0.00)	<0.0001
NLR	8.06 (4.65)	4.30 (1.49)	0.0001

The relationship between systemic inflammation (CRP) and NLR was explored using Pearson's correlation. CRP (mean 45.67, SD 38.17) and NLR (mean 8.47, SD 4.65

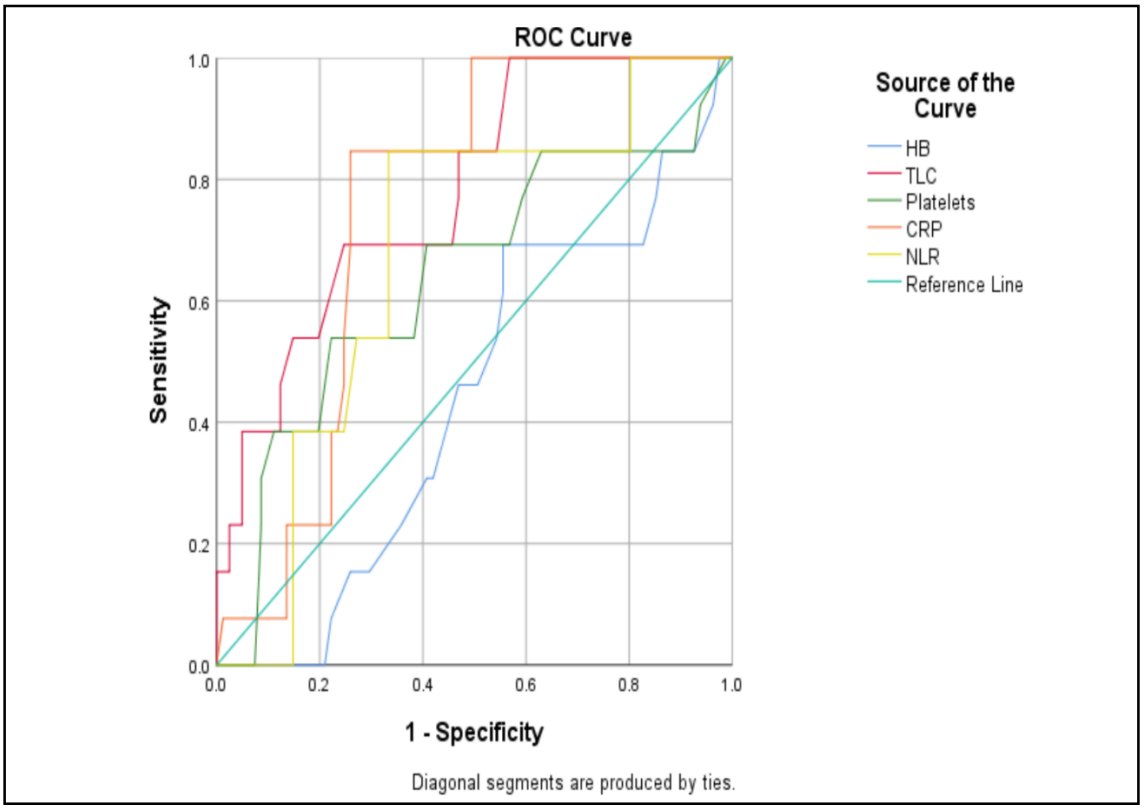

Figure 1- Receiver Operating Characteristic (ROC) Curves for Diagnostic Accuracy of Laboratory Parameters in Predicting Hospital Outcomes in COPD Patients

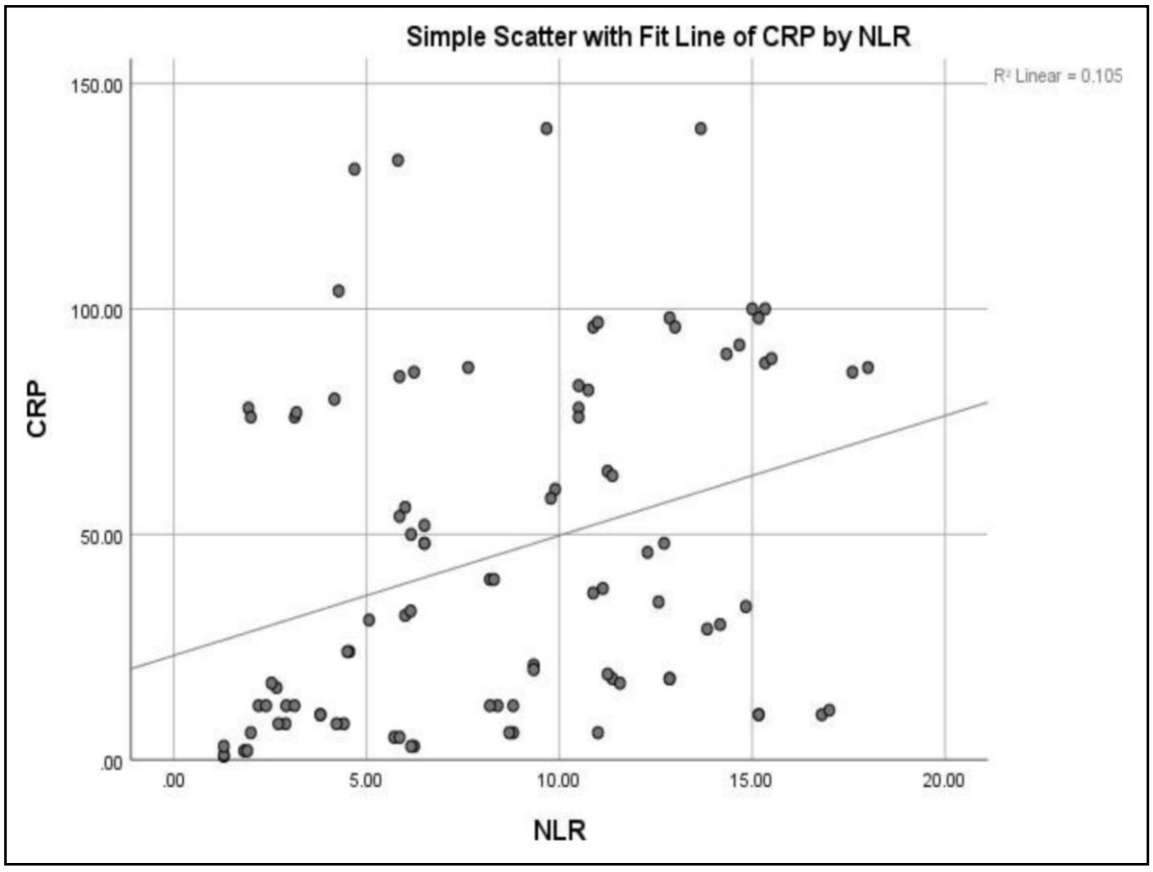

Figure 2- Correlation between CRP and NLR

showed a moderate positive correlation (r = 0.324, p = 0.001; n = 94), indicating that higher CRP values tended to be associated with higher NLR. (Figure 2)

DISCUSSION

The present study evaluated demographic patterns, clinical features, laboratory markers, and diagnostic predictive values among hospitalized COPD patients. The mean age of participants was 63.16 years, indicating that COPD predominantly affects older adults. This aligns with established evidence demonstrating greater disease burden in advancing age due to cumulative exposure to environmental and lifestyle risk factors such as smoking [1]. The higher representation of males (67%) also corresponds with prior epidemiological findings wherein historically higher smoking rates among men contributed to increased COPD prevalence; however, the gender gap has been narrowing in recent years with rising smoking trends among females [2].

More than half of the study population were current smokers, highlighting the persistent influence of smoking as a major risk factor for COPD development and exacerbation [3]. The presence of ex-smokers in the cohort reflects ongoing cessation efforts, which are known to slow disease progression and improve long-term respiratory outcomes [4]. A considerable proportion of non-smokers in the sample suggests possible contribution from alternative etiologies, including exposure to biomass fuels, occupational pollutants, or genetic predisposition, which have been documented in COPD pathogenesis particularly in LMIC settings [3].

The clinical profile of patients demonstrated the chronic and progressive nature of the disease, with an average disease duration of over nine years. Altered consciousness on admission was noted in approximately one-fifth of patients, which may indicate severe exacerbations, hypercapnia, or advanced respiratory compromise, consistent with previous reports associating acute exacerbations with altered sensorium and systemic complications [7].

Most patients required oxygen therapy during hospitalization, reflecting impaired gas exchange common in severe COPD exacerbations. The need for ICU admission in one-fourth of the participants emphasizes the clinical severity and high resource utilization associated with acute exacerbations, similar to findings reported in previous studies of hospitalized COPD populations [9]. Non-invasive ventilation was used in nearly one-fourth of cases, consistent with evidence supporting its effectiveness in reducing the need for invasive mechanical ventilation and associated complications [8]. Only a minority required intubation, reflecting successful stabilization with non-invasive modalities and appropriate clinical intervention strategies.

The mean duration of hospitalization was comparable to previously published data reporting variability based on severity and treatment response [5]. An in-hospital mortality rate of 13.8% underscores the life-threatening nature of severe exacerbations and highlights the importance of early recognition and risk stratification [8].

Laboratory evaluation showed elevated inflammatory markers, including total leukocyte count and CRP, reflecting active inflammation and frequent infectious triggers during acute COPD exacerbations [13,14]. Arterial blood gas analysis demonstrated respiratory acidosis with compensatory metabolic alkalosis, characteristic of advanced COPD with impaired ventilation and chronic hypercapnia [15,16]. The elevated NLR reflects systemic inflammation and stress response and corresponds with previous research supporting NLR as a marker of inflammatory burden in COPD [17].

When examining the diagnostic accuracy of laboratory variables, hemoglobin demonstrated poor predictive capability (AUC 0.439), suggesting limited role in prognostication despite its physiological relevance in oxygen delivery. Similar findings have been noted where hemoglobin did not correlate strongly with acute outcomes in COPD [11]. In contrast, total leukocyte count exhibited good diagnostic accuracy (AUC 0.779), reflecting its strong association with acute inflammatory exacerbations and infection-driven deterioration [14].

Platelet counts demonstrated moderate but statistically insignificant predictive power. Although platelets contribute to inflammatory pathways and vascular remodeling, their independent prognostic role in COPD remains inconclusive, consistent with previously reported findings [10]. CRP demonstrated good diagnostic accuracy (AUC 0.752), reinforcing its role as a clinically meaningful biomarker of systemic inflammation and exacerbation severity in COPD [13]. NLR displayed moderate yet statistically significant predictive value (AUC 0.677), supporting its utility as an accessible and inexpensive marker for outcome prediction and risk stratification in acute COPD hospital admissions [16,17].

Taken together, the findings indicate that inflammatory markers—particularly TLC, CRP, and NLR—may serve as clinically useful predictors of adverse outcomes in hospitalized COPD patients. The significance of gas exchange variables and oxygen requirement further supports the integration of clinical and laboratory indicators for comprehensive risk assessment.

CONCLUSION

This prospective observational study evaluated the diagnostic accuracy of the neutrophil–lymphocyte ratio (NLR) and other routine laboratory markers in predicting in-hospital outcomes among patients admitted with COPD. We observed substantial systemic inflammation and respiratory compromise in this cohort, with meaningful rates of ICU admission, ventilatory support, and in-hospital mortality. ROC analysis demonstrated good predictive ability for TLC and CRP and

moderate diagnostic accuracy for NLR. Although, paradoxically, lower NLR values were observed among non-survivors in this sample, NLR remained a statistically significant predictor, suggesting that inflammatory indices may behave differently across clinical contexts and severity spectra. Gas exchange parameters and oxygen requirement were also strongly associated with adverse outcomes. Overall, easily obtainable bedside variables such as NLR, TLC, CRP and ABG indices can contribute to early risk stratification in hospitalized COPD patients, but NLR should be interpreted in conjunction with clinical status and other biomarkers. Larger multicentre studies are warranted.

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