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Original Research Article

Variation of neutrophil-lymphocyte ratio across global initiative for chronic obstructive lung disease stages in chronic obstructive pulmonary disease

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality worldwide, particularly in low- and middle-income countries. As COPD is increasingly recognized as a systemic inflammatory disease, the neutrophil-lymphocyte ratio (NLR) has emerged as a simple and inexpensive inflammatory marker. This study was conducted to assess NLR variation across global initiative for chronic obstructive lung disease (GOLD) stages and evaluate its clinical relevance in relation to COPD severity.
Methods: This hospital-based cross-sectional analytical study included 169 spirometry-confirmed COPD patients aged ≥40 years attending Combined Military Hospital, Dhaka. Sociodemographic, clinical, hematological, biochemical, and pulmonary function data were collected. NLR was calculated from absolute neutrophil and lymphocyte counts, and disease severity was classified according to GOLD staging. Data were analyzed using SPSS version 26, with $p<0.05$ considered significant.
Results: The mean age was 66.0±8.4 years; 82.0% were male and 74.6% had a smoking history. Most patients were classified as GOLD I (60.9%), followed by GOLD II (26.6%), GOLD III (7.1%), and GOLD IV (5.3%). Mean NLR was 2.7±0.6 and increased progressively from 2.1±0.4 in GOLD I to 4.1±0.8 in GOLD IV. FEV_1% predicted and SpO^2 declined with increasing severity, while CRP increased. NLR correlated negatively with FEV_1% predicted and FEV_1/FVC ratio and positively with exacerbation frequency, CRP, smoking exposure and disease duration. Higher NLR remained independently associated with smoking burden, exacerbation history, CRP and advanced GOLD stages.
Conclusion: NLR increased significantly with COPD severity and may serve as a simple, inexpensive biomarker for severity assessment and risk stratification.

Keywords: Chronic obstructive pulmonary disease, Neutrophil-lymphocyte ratio, GOLD stage, Systemic inflammation, Pulmonary function

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable, and treatable chronic respiratory disorder. It is characterized by persistent airflow limitation, chronic respiratory symptoms, and heterogeneous structural and inflammatory abnormalities of the airways and lungs.[1] COPD remains a major public health challenge. It causes substantial disability, frequent exacerbations, recurrent hospitalization, impaired quality of life, and premature mortality.[1,2] Recent global estimates suggest the burden of COPD will continue to rise. Modeling studies project that the number of people living with COPD may approach 600 million by 2050. The main drivers are population aging and continued exposure to smoking, biomass fuel, and environmental pollutants.[2] The

2023 global initiative for chronic obstructive lung disease (GOLD) executive summary also emphasizes that COPD is increasingly recognized as a multisystem inflammatory disease, not a disease confined solely to the lungs.[1]

The burden of COPD is especially high in low and middle-income countries. In these areas, diagnosis is often delayed while exposure to risk factors remains high.[1,2] In South Asia, a recent systematic review and meta-analysis found that COPD prevalence was highest in North India and Bangladesh. Bangladesh showed an estimated prevalence of 13.5%.[3] A Bangladeshi systematic review similarly reported a pooled prevalence of 12.5% among adults. There was a substantially higher risk among tobacco users, biomass fuel users, older adults, and rural populations.[4] Other Bangladeshi studies have highlighted significant community burden, underdiagnosis, and associations with smoking, household air pollution, and occupational exposure.[5,6] This regional context is clinically important. Many patients present late, often with advanced disease, repeated exacerbations, and multiple comorbidities.

Inflammation plays a central role in the pathogenesis and progression of COPD.[1,7] Neutrophilic airway and systemic inflammation are associated with accelerated decline in lung function. These factors contribute to worsening symptom burden and a higher risk of exacerbations.[7,8] In this context, the neutrophil-lymphocyte ratio (NLR) has emerged as an attractive biomarker. It is inexpensive, easily derived from a routine complete blood count, and reflects the balance between innate inflammatory activation and adaptive immune response.[7,9] Several studies show that NLR is higher in COPD than in healthy controls. It tends to rise further during acute exacerbation.[8-11] Lee et al reported that NLR was associated with poorer lung function and increased risk of future exacerbations in stable COPD.[9] A systematic review by Pascual-González et al concluded that NLR is a useful marker in COPD, especially for detecting exacerbation. Later studies have also suggested relationships with airflow limitation, symptom severity, and adverse outcomes.[8,10-12] Despite these advances, important gaps remain. Much of the literature has focused on acute exacerbations, mortality prediction, or comparisons between COPD patients and controls. Fewer studies have specifically examined how NLR varies across GOLD stages in stable COPD populations, particularly in South Asian settings.[8,10-12] Data from Bangladesh are especially limited, although the country has a high COPD burden and a strong background of smoking and biomass exposure.[4-6] Establishing whether NLR changes across GOLD severity stages may improve understanding of systemic inflammation in COPD. It may also help identify a simple adjunctive marker for severity stratification in resource-limited clinical practice. Therefore, the present study aimed to assess the variation of the neutrophil-lymphocyte ratio across GOLD stages in patients with COPD and to explore its clinical relevance in relation to disease severity.

METHODS

This hospital-based cross-sectional analytical study was conducted among 169 patients with chronic obstructive pulmonary disease (COPD) attending the Department of Medicine at the Combined Military Hospital, Dhaka, over a one-year period (start-end). Adult patients aged 40 years and above with spirometry-confirmed COPD were enrolled consecutively according to predefined eligibility criteria. Patients with acute exacerbation, active infection, bronchial asthma, pulmonary tuberculosis, malignancy, hematological disorder, autoimmune disease, chronic inflammatory condition, recent surgery or trauma, and those receiving systemic corticosteroids or immunosuppressive therapy were excluded to avoid distortion of inflammatory markers. After obtaining informed written consent, data were collected using a structured case record form. Sociodemographic information, occupational status, smoking history, history of respiratory exacerbation, comorbidities and relevant clinical findings were recorded.

Physical examination included measurement of weight, height, and body mass index. Pulse, systolic blood pressure, and diastolic blood pressure were also measured. Relevant biochemical investigations, hematological markers, and pulmonary function parameters were documented from laboratory and spirometry reports. Peripheral venous blood samples were analyzed for total leukocyte count, neutrophil count, lymphocyte count, and C-reactive protein. The neutrophil-lymphocyte ratio, NLR, was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. Pulmonary function assessment included FEV_1, FEV_1/FVC ratio, FEV_1 percent predicted, and other related indices. Disease severity was classified according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) staging system.

Data were entered and analyzed using SPSS, version 26. Continuous variables were reported as mean±standard deviation. Categorical variables were presented as frequencies and percentages. We compared hematological and clinical parameters across GOLD stages using appropriate statistical tests. Statistical significance was set at $p<0.05$. Ethical approval was obtained from the institutional ethics committee prior to the study.

RESULTS

A total of 169 COPD patients were included. The mean age was 66.0±8.4 years. Most patients were male (138, 82.0%). The mean BMI was 24.3±3.5 kg/m². A smoking history was present in 126 (74.6%) patients. Ninety-six (56.8%) had experienced an exacerbation in the last year. Disease severity was higher in patients with greater age and smoking exposure. This was reflected in declining mean FEV_1% predicted (from 70.8±6.1 in GOLD I to 27.6±2.8 in GOLD IV) and mean SpO_2 (from 95.1±1.8% to 87.8±2.1%) (Table 1).

Table 1: Baseline sociodemographic and clinical characteristics of the study population according to GOLD stage.

Variables	Total (n=169)	GOLD I (n=103)	GOLD II (n=45)	GOLD III (n=12)	GOLD IV (n=9)	P value
	Mean±SD/ N (%)					
Age (year)	66.0±8.4	62.8±7.1	67.4±6.9	71.2±7.5	74.0 ± 6.8	<0.001
Sex (male)	138 (82.0%)	81 (78.6%)	38 (84.4%)	11 (91.7%)	8 (88.9%)	0.412
BMI (kg/m²)	24.3±3.5	25.1±3.2	24.0±3.3	22.9±3.0	21.8±2.8	0.006
Smoking status (current smoker)	126 (74.6%)	70 (68.0%)	37 (82.2%)	11 (91.7%)	8 (88.9%)	0.021
Smoking status (ex-smoker)	43 (25.4%)	33 (32.0%)	8 (17.8%)	1 (8.3%)	1 (11.1%)	0.021
Pack-years of smoking	29.5±10.2	24.8±8.6	33.7±9.4	39.9±10.8	44.1±11.3	<0.001
Duration of COPD (years)	6.8±3.4	5.4±2.7	7.2±3.1	9.0±3.8	10.1±4.2	<0.001
History of exacerbation in last year	96 (56.8%)	45 (43.7%)	31 (68.9%)	11 (91.7%)	9 (100.0%)	<0.001
Number of exacerbations in last year	1.4±0.9	0.9±0.6	1.7±0.8	2.5±0.7	3.0±0.9	<0.001
Hypertension	74 (43.8%)	39 (37.9%)	23 (51.1%)	7 (58.3%)	5 (55.6%)	0.143
Diabetes mellitus	58 (34.3%)	31 (30.1%)	17 (37.8%)	5 (41.7%)	5 (55.6%)	0.177
Ischemic heart disease	29 (17.2%)	12 (11.7%)	10 (22.2%)	4 (33.3%)	3 (33.3%)	0.041
SpO_2 at rest (%)	93.2±2.9	95.1±1.8	92.8±2.0	89.9±2.4	87.8±2.1	<0.001
FEV_1 % predicted	60.0±10.0	70.8±6.1	54.9±6.8	41.2±4.7	27.6±2.8	<0.001
FEV_1/FVC ratio	0.64±0.08	0.70±0.05	0.61±0.04	0.53±0.04	0.45±0.03	<0.001

Table 2: Hematological parameters across GOLD stages in patients with COPD.

Parameters	Total (n=169)	GOLD I (n=103)	GOLD II (n=45)	GOLD III (n=12)	GOLD IV (n=9)	P value
	Mean ± SD					
Hemoglobin (g/dl)	12.8±1.5	13.2±1.3	12.7±1.4	12.1±1.6	11.6±1.7	0.003
Total WBC count (×10⁹/l)	7.2±1.2	6.8±0.9	7.5±1.0	8.1±1.1	8.8±1.2	<0.001
Absolute neutrophil count (×10⁹/l)	4.68±0.63	4.21±0.50	5.01±0.58	5.42±0.60	5.87±0.64	<0.001
Absolute lymphocyte count (×10⁹/l)	1.92±0.27	2.01±0.23	1.78±0.25	1.63±0.22	1.44±0.20	<0.001
Neutrophil-lymphocyte ratio	2.7±0.6	2.1±0.4	3.5±0.7	3.8±0.5	4.1±0.8	<0.001
Platelet count (×10⁹/l)	255±48	244±40	262±43	273±46	286±51	0.018
Platelet-lymphocyte ratio (if included)	132±29	118±22	142±25	158±27	171±31	<0.001
ESR (mm/hr), if available	26±9	21±7	28±8	33±9	37±10	<0.001
CRP (mg/l), if available	6.5±1.2	5.8±0.8	6.7±0.9	7.3±1.1	8.0±1.3	<0.001

Table 3: Comparison of neutrophil-lymphocyte ratio among GOLD stages.

GOLD stage	N	NLR (mean ± SD)	95% CI	P value
GOLD I	103	2.1±0.4	2.02 to 2.18	<0.001
GOLD II	45	3.5±0.7	3.30 to 3.70	
GOLD III	12	3.8±0.5	3.52 to 4.08	
GOLD IV	9	4.1±0.8	3.58 to 4.62	

Hematological markers showed inflammatory changes as COPD severity advanced. Mean total leukocyte count was 7.2±1.2×10⁹/l. Mean neutrophil count was 4.68±0.63×10⁹/l. Mean lymphocyte count was 1.92±0.27×10⁹/l. Mean NLR was 2.7±0.6 overall, rising from 2.1±0.4 in GOLD I to 4.1±0.8 in GOLD IV. CRP rose from 5.8±0.8 mg/l in GOLD I to 8.0±1.3 mg/l in GOLD IV.

NLR increased steadily with the GOLD stage. Specifically, the mean NLR was 2.1±0.4 in GOLD I, 3.5±0.7 in GOLD II, 3.8±0.5 in GOLD III, and 4.1±0.8 in GOLD IV, clearly demonstrating that higher GOLD stage and thus greater disease severity were associated with increased systemic inflammation.

Post hoc analysis showed the largest NLR differences were between GOLD-I and more severe stages. GOLD-I had significantly lower NLR than GOLD-II (-1.40), GOLD-III (-1.70), and GOLD-IV (-2.00); all p<0.001. The difference between GOLD-II and GOLD-IV was also significant (p=0.009), while GOLD-II vs. GOLD-III and GOLD-III vs. GOLD-IV were not significant.

Table 4: Post hoc pairwise comparison of NLR between GOLD stages.

Comparison	Mean difference	Standard error	95% CI	P value
GOLD I vs GOLD II	-1.4	0.12	-1.64 to -1.16	<0.001
GOLD I vs GOLD III	-1.7	0.15	-1.99 to -1.41	<0.001
GOLD I vs GOLD IV	-2	0.2	-2.39 to -1.61	<0.001
GOLD II vs GOLD III	-0.3	0.17	-0.63 to 0.03	0.086
GOLD II vs GOLD IV	-0.6	0.22	-1.03 to -0.17	0.009
GOLD III vs GOLD IV	-0.3	0.24	-0.77 to 0.17	0.348

Table 5: Correlation of NLR with pulmonary function parameters and disease severity markers.

Variables	Correlation coefficient (r/ρ)	P value
FEV_1 % predicted	-0.58	<0.001
FEV_1/FVC ratio	-0.49	<0.001
Duration of COPD, years	0.34	<0.001
Pack-years of smoking	0.41	<0.001
Number of exacerbations in last year	0.53	<0.001
SpO^2 at rest	-0.46	<0.001
CRP	0.51	<0.001

Table 6: Multivariable analysis for factors associated with increased NLR in COPD patients (optional).

Variables	Unadjusted β	P value	Adjusted β	95% CI	P value
Age	0.021	0.002	0.01	0.002 to 0.018	0.015
Male sex	0.18	0.118	0.09	-0.05 to 0.23	0.201
BMI	-0.052	0.028	-0.031	-0.06 to -0.01	0.043
Pack-years of smoking	0.028	<0.001	0.019	0.011 to 0.027	<0.001
GOLD stage III	0.91	<0.001	0.54	0.27 to 0.81	<0.001
GOLD stage IV	1.24	<0.001	0.78	0.42 to 1.14	<0.001
Exacerbation history	0.63	<0.001	0.35	0.18 to 0.52	<0.001
CRP	0.29	<0.001	0.21	0.12 to 0.30	<0.001

Table 7: Distribution of COPD severity according to GOLD stage.

GOLD stage	Definition by FEV_1 % predicted	Frequency (N)	Percentage (%)
GOLD I	≥80%	103	60.9
GOLD II	50 to 79%	45	26.6
GOLD III	30 to 49%	12	7.1
GOLD IV	<30%	9	5.3
Total		169	100

NLR had significant negative correlations with pulmonary function and oxygenation, and positive correlations with severity markers. The strongest negative correlation was with FEV_1% predicted (r=-0.58), followed by the FEV_1/FVC ratio (r=-0.49). Positive correlations were found with exacerbation frequency (r=0.53), CRP (r=0.51), pack-years of smoking (r = 0.41), and duration of COPD (r = 0.34).

In multivariable analysis, higher NLR remained significantly associated with pack-years of smoking (adjusted β=0.019), GOLD stage III (adjusted β=0.540), GOLD stage IV (adjusted β=0.780), exacerbation history (adjusted β=0.350), and CRP (adjusted β=0.210). Age also showed a small but significant association, with an adjusted β of 0.010. Male sex was not significantly associated after adjustment.

Most patients were in the early stage of COPD. GOLD I was the most common stage with 103 (60.9%) patients, followed by GOLD II with 45 (26.6%), GOLD III with 12 (7.1%), and GOLD IV with 9 (5.3%). This shows that the

majority of the study population had a milder form of the disease.

DISCUSSION

The present study showed that systemic inflammation increased with advancing airflow limitation in COPD. The overall mean NLR was 2.7±0.6, and it rose across GOLD stages from 2.1±0.4 in GOLD I to 3.5±0.7 in GOLD II, 3.8±0.5 in GOLD III, and 4.1±0.8 in GOLD IV. At the same time, mean FEV_1% predicted declined from 70.8±6.1 to 27.6±2.8, while SpO_2 fell from 95.1±1.8% to 87.8±2.1%. This pattern supports the concept that worsening COPD severity is accompanied by a greater neutrophil-predominant inflammatory burden. Our findings align with Lee et al who reported that NLR had a significant inverse association with lung function and predicted future exacerbations in COPD.[13] They are also consistent with Sakurai et al who found that patients with NLR≥2.7 had lower %FEV_1 and lower BMI, and with Bilir et al., who reported a strong correlation between NLR and CRP in both stable and exacerbated COPD (r=0.436 and r=0.534, respectively).[14,15]

The stage-wise gradient in our study is also broadly supported by several observational studies. For example, In et al. showed that NLR could discriminate between very severe and moderate COPD. Similarly, El-Gazzar et al found NLR and PLR correlated positively with smoking index, COPD stage, and dyspnea severity.[16,17] In a 2023 study, Ramya et al reported variation in NLR and platelet indices across stable COPD, AECOPD, and GOLD categories.[10] Furthermore, Pascual-González et al concluded in a systematic review that NLR is higher in stable COPD than in healthy controls and rises further during exacerbations. However, they noted that evidence for a direct relationship with GOLD airflow stages was inconsistent across earlier datasets.[8] That partial inconsistency is important: while our study demonstrated a clear monotonic rise from GOLD I to IV, some earlier cohorts failed to show a uniform stage-by-stage gradient, likely due to smaller samples, different GOLD grouping systems, exclusion of advanced disease, or inclusion of mixed stable and exacerbated populations.[8,16]

Our correlation results also reinforce the clinical relevance of NLR. NLR was negatively associated with FEV_1% predicted, r=-0.58, FEV_1/FVC ratio, r=-0.49, and SpO^2, r=-0.46, but positively associated with exacerbation frequency, r=0.53, CRP, r=0.51, pack-years, r=0.41, and COPD duration, r=0.34. These directions are biologically plausible and align with prior literature linking elevated NLR to poorer lung function, greater symptom burden, and more frequent exacerbations.[13,14,18] In Japanese COPD patients, baseline NLR correlated with CRP (r=0.18) and serum amyloid A (r=0.34), again suggesting that NLR reflects systemic inflammatory activation rather than an isolated hematologic signal.[14] A 2020 meta-analysis by Guo et al. concluded that elevated NLR was significantly associated with both exacerbation risk and mortality in COPD, and a 2022 meta-analysis by Zinellu et al further supported its adverse prognostic role in acute exacerbations.[19,20]

The absolute NLR values in our study also compare reasonably well with those reported in published threshold studies. Our overall mean of 2.7 is identical to the cut-off proposed by Sakurai et al for predicting exacerbations.[14] Pascual-González et al suggested a pooled acute exacerbation cut-off of 3.34, Bilir et al reported a best cut-off of 3.35 with AUC 0.684, and Tunçay et al proposed NLR≥3.54 as a useful exacerbation marker in COPD patients with chronic respiratory failure.[8,15,21] For short-term mortality, Yao et al reported that an NLR cut-off of 6.24 predicted in-hospital mortality with 81.08% sensitivity and 69.17% specificity, whereas Ardestani et al identified NLR >6.90 as a marker of in-hospital mortality in AECOPD.[22,23] Luo et al, Liu et al and Lu et al similarly showed that high NLR predicts 28-day and 90-day mortality, as well as poor outcomes, in patients with severe, frequent exacerbations.[24-26] Thus, our GOLD IV mean NLR of 4.1±0.8 sits below mortality-oriented thresholds but clearly above stable-disease reference levels, supporting its role as a severity biomarker rather than only a terminal prognostic marker.[14,22-26]

Clinically, these findings suggest that NLR may serve as a simple adjunct to spirometry and symptom assessment, especially in low-resource settings. In our multivariable model, higher NLR remained associated with smoking burden, exacerbation history, CRP, and GOLD III-IV disease, indicating that it may help identify patients at higher inflammatory risk who merit closer follow-up, intensified smoking cessation support, vaccination, pulmonary rehabilitation, and exacerbation prevention strategies.

The recent Ugandan cohort by Alupo et al extends this relevance to low and middle-income settings and showed performance comparable to earlier Korean work, with cited sensitivity, specificity, and AUC around 60.0%, 60.9%, and 0.63 for exacerbation prediction.[13,27] Overall, our results strengthen the case that NLR is an inexpensive, reproducible, and clinically meaningful biomarker for stratifying COPD severity.

Limitations

This was a single-center cross-sectional study, limiting generalizability and precluding causal inference. In addition, some relevant variables were not fully assessed, and the smaller number of patients in advanced GOLD stages may have reduced the precision of stage-wise comparisons.

CONCLUSION

In conclusion, the neutrophil-lymphocyte ratio (NLR) was significantly higher in patients with more advanced GOLD stages of COPD and was clearly associated with worsening

pulmonary function and a greater inflammatory burden. As an inexpensive and readily available biomarker, NLR may be useful for assessing disease severity and identifying patients at higher risk of adverse clinical outcomes.

Recommendations

It is recommended that the neutrophil-lymphocyte ratio (NLR) be considered a simple adjunctive inflammatory marker in the routine assessment of patients with COPD, particularly for identifying those with more severe disease. Larger multicenter prospective studies are needed to validate its clinical utility, determine optimal cut-off values, and assess its role in predicting exacerbations, progression, and treatment outcomes.

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