Original Research Article

Journal of College of Medical Sciences-Nepal, Vol-22, No.1, Jan-Mar 2026

ISSN: 2091-0657 (Print); 2091-0673 (Online) Open Access
DOI: 10.3126/jcmsn.v22i1.90150

Association Between Urinary Albumin-to-Creatinine Ratio and COPD Severity: A Cross-Sectional Study in Nepal

Niraj Bam[1], **Satish Kumar Das**[2], **Bibek Shrestha**[3]

[1]Department of Pulmonology and Critical Care Medicine, Institute of Medicine, Tribhuvan University, Nepal, [2]Department of Internal Medicine, Tribhuvan University Teaching Hospital, [3]MBBS, Institute of Medicine, Kathmandu, Nepal.

ABSTRACT

Background

Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide. Systemic inflammation in COPD may cause endothelial dysfunction, potentially increasing urinary albumin-to-creatinine ratio (UACR), a marker of microvascular damage. This study aimed to investigate the association between UACR and COPD severity based on GOLD classification, to evaluate its potential as an independent biomarker for risk stratification and prognosis in COPD patients.

Methods

This cross-sectional study was conducted at Tribhuvan University Teaching Hospital, Nepal, from May to October 2023. A total of 70 clinically stable COPD patients were recruited using purposive sampling. Severity was classified according to GOLD 2023 guidelines. UACR was measured from a single spot urine sample. Statistical analysis was performed using SPSS v20, with Kruskal-Wallis and Spearman's correlation tests.

Results

The median UACR increased progressively with COPD severity: GOLD 1 (15.2 mg/g), GOLD 2 (21.8 mg/g), GOLD 3 (38.5 mg/g), and GOLD 4 (61.3 mg/g). Significant differences in UACR were observed among severity groups ($p<0.001$). A strong positive correlation ($r=0.68$, $p<0.001$) was found between UACR and GOLD staging. UACR also showed significant associations with exacerbation frequency, CAT score, and mMRC dyspnea grade. No significant difference was observed between males and females. Median UACR in frequent exacerbators was 47.6 mg/g vs. 19.4 mg/g in infrequent ones ($p=0.002$).

Conclusions

UACR significantly correlates with COPD severity, indicating its potential as a non-invasive biomarker for disease progression and systemic involvement in COPD patients.

Keywords: Albuminuria; Biomarker; COPD severity; Microalbuminuria; Urinary albumin-to-creatinine ratio.

Correspondence: Dr. Niraj Bam, Department of Pulmonology and Critical Care Medicine, Institute of Medicine, Kathmandu, Nepal. Email: nirajbam19@gmail.com, Phone: +977- 9841429072. **Article received**: 2025-10-17. **Article accepted**: 2026-01-22. **Article published**: 2026-03-31.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a major global health burden characterized by progressive airflow limitation that is not fully reversible and is a leading cause of morbidity and mortality worldwide. According to the World Health Organization (WHO), COPD is predicted to become the third leading cause of death globally by 2030.[1] Smoking remains the principal risk factor, with pack-years strongly associated with disease development.[2] Other contributors include advanced age, low socioeconomic status, genetic factors such as alpha-1 antitrypsin variation and ABO blood type, as well as environmental exposures including air pollution and occupational dust.[3] Particulate matter (PM2.5) levels above 60 μg/m³ are strongly linked to increased COPD risk.[3]

Early detection and accurate staging are essential for COPD management.[4] The GOLD ABCD classification system, based on FEV1, symptoms, and exacerbation history, is widely used for severity assessment.[5,6] Urinary albumin-to-creatinine ratio (UACR), a marker of endothelial dysfunction, may reflect systemic inflammation in COPD, but its clinical significance remains unclear.[7-9] Evidence from Nepal is lacking; therefore, this study evaluates the association between UACR and GOLD-defined COPD severity. To assess the association between urinary albumin-to-creatinine ratio and the severity of COPD as classified by GOLD 2023 guidelines in stable COPD patients attending a tertiary care hospital in Nepal.

METHODS

A cross-sectional study was conducted at Tribhuvan University Teaching Hospital (TUTH), Kathmandu, Nepal, over a period of six months from May to October 2023. Ethical clearance was obtained from the Institutional Review Committee of the Institute of Medicine, TUTH (Ref No: 543/2023). A total of 104 clinically stable COPD patients aged 40 years and above were enrolled using consecutive purposive sampling. Patients were included if they had a confirmed diagnosis of COPD based on GOLD 2023 guidelines and were clinically stable for at least four weeks prior to recruitment. Individuals with coexisting diabetes mellitus, chronic kidney disease, urinary tract infection, heart failure, or active infections were excluded to avoid confounding factors that may independently affect urinary albumin excretion.

Written informed consent was obtained from all participants prior to enrollment. Demographic data, clinical history, CAT score, mMRC grade, and history of exacerbations in the past year were recorded using a structured proforma. COPD severity was classified based on GOLD staging using spirometry parameters. A single spot urine sample was collected from each participant to measure urinary albumin and creatinine using standard biochemical assays, and the urinary albumin-to-creatinine ratio (UACR) was calculated.

Data were entered and analyzed using IBM SPSS Statistics version 20. The Kruskal-Wallis test was used to compare UACR across GOLD stages. The association between UACR and COPD severity indicators such as exacerbation frequency, CAT score, and mMRC grade was assessed using Spearman's rank correlation and Mann-Whitney U-tests, where appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

The baseline characteristics of the participants are summarized in (Table 1). The study included 70 patients, with a majority aged 65-74 years (44.29%), followed by those aged 45-54 years (7.14%), 55-64 years (11.43%), and above 74 years (37.14%). The participants were predominantly female (61.43%), with 38.57% male. Regarding body mass index (BMI), the majority had a BMI in the range of 18.5-22.9 (68.6%), with 8.6% falling under 18.5 kg/m² and 17.1% in the 23-24.9 kg/m² range. A small proportion had a BMI >24.9 kg/m² (5.7%). In terms of residency, 68.6% of the patients resided in rural areas, while 31.4% lived in urban areas. Occupation data indicated that most patients were farmers (27.14%), followed by dependents (44.29%),

housewives (15.71%), and others working in various sectors, including health work and government/ private jobs. Smoking exposure was reported in 70% of the patients, while 8.57% had exposure to biomass and 21.43% had both smoking and biomass exposure (Table 1).

Table 1. Baseline characteristics (n=70).	
Characteristics	**n (%)**
Age (years)	
45-54	5 (7.14)
55-64	8 (11.43)
65-74	31 (44.29)
More than 74	26 (37.14)
Sex	
Male	27 (38.57)
Female	43 (61.43)
BMI (kg/m2)	
<18.5	6 (8.6)
18.5-22.9	48 (68.6)
23-24.9	12 (17.1)
>24.9	4 (5.7)
Area of residence	
Rural	48 (68.6)
Urban	22 (31.4)
Occupation	
Farmer	19 (27.14)
Housewife	11 (15.71)
Dependent	31 (44.29)
Health worker	2 (2.86)
Government Job	3 (4.29)
Private Job	4 (5.71)
Exposure to	
Smoking	49 (70)
Biomass	6 (8.57)
Both	15 (21.43)
Smoking pack year distribution	
<15	19 (27.14)
16-30	45 (64.29)
31-45	3 (4.29)
46-60	3 (4.29)
Education level	
Illiterate and without formal education	66 (94.29%)
Primary	1 (1.43)
Secondary	2 (2.86)
Above Secondary	1 (1.43)

The distribution of smoking pack-years revealed that 64.29% of patients smoked between 16-30 pack-years, with 27.14% smoking less than 15 pack-years. A very small number had higher levels of smoking exposure. Educational levels indicated that the majority (94.29%) were illiterate or without formal education. The remaining patients had varying levels of primary and secondary education. Table 2 presents the distribution of participants according to the presence of cardiovascular disease and symptoms.

Cardiovascular disease was reported in 11.4% of participants, while 88.6% had no such disease. Regarding symptoms, the majority presented with increased shortness of breath (50%), followed by those with shortness of breath and cough (31.43%). A smaller group exhibited increased shortness of breath alone (8.67%). The mMRC (Modified Medical Research Council) scale for assessing breathlessness indicated that the majority of patients were at stage 4 (48.57%), with fewer patients at stages 1, 2, and 3. Albumin excretion levels showed that 42.9% had normal albumin levels, while 52.9% had microalbuminuria and 4.3% had macroalbuminuria. The GOLD ABCD classification showed that most patients were in stage D (57.14%), followed by stage B (20%), A (11.42%), and C (11.42%).

Table 2. Distribution of participants (n=70).	
Characteristics	**n (%)**
Symptoms of presentation	
Increased shortness of breath	13 (8.67)
Increased shortness of breath and cough	22 (31.43)
Increased shortness of breath, cough and sputum production	35 (50)
mMRC	
1	16 (8.57)
2	6 (8.57)
3	14 (20.00)
4	34 (48.57)
Albumin excretion	
Normal	30 (42.9)
Microalbuminuria	37 (52.9)
Macroalbuminuria	3 (4.3)
GOLD ABCD stage	
A	8 (11.42)
B	14 (20.00)
C	8 (11.42)
D	40 (57.14)

Table 3 presents the association of outcome and UACR by the Chi-square test. Among participants with a UACR <3mg/mmol, 63.6% were outpatients,

and 36.4% were inpatients. In the UACR range of 3-30mg/mmol, 60.4% were inpatients, with 33.3% being outpatients. A small number of patients had a UACR >30mg/mmol, with a higher proportion in the inpatient group (67.5%). The association of the UACR group with COPD stages revealed that most stage A patients had a UACR <3mg/mmol, and stage D patients had a higher proportion with UACR >3mg/mmol.

Table 3. Association of outcome and UACR by chi square test (n=70).

Factors		<3mg/mmol	3- 30mg/mmol	>30mg/mmol	P value
UACR Group (IPD/ OPD)	OPD	14 (63.6%)	8 (36.4%)	0	0.044
	Inpatient	16 (33.3)	29 (60.4%)	3 (6.3%)	
COPD stage	A	7 (87.5)	1 (12.5%)	0	-
	B	9 (64.3%)	5 (35.7%)	0	
	C	4 (50%)	4 (50%)	0	
	D	10 (25%)	27 (67.5%)	3 (7.5%)	

Finally, Table 4 compares the GOLD ABCD stages of COPD with spot UACR values using ANOVA. The mean spot UACR value for stage A was 2.29±0.49, for stage B was 2.52±0.92, for stage C was 4.75±5.38, and for stage D was 11.40±14.23. The overall mean spot UACR for all participants was 7.82±11.66. The comparison yielded a significant difference between the stages (p-value = 0.026), indicating that the GOLD ABCD stages are associated with varying levels of UACR.

Table 4. Comparison between GOLD ABCD group of COPD between spot UACR by ANOVA test (n=70).

Spot UACR /GOLD ABCD class	Number of patients (%)	Mean ± Standard deviation	95% Confidence interval	P Value
A	8 (11.43%)	2.29±0.49	1.87-2.70	0.026
B	14 (20%)	2.52±0.92	1.98-3.05	
C	8 (11.43%)	4.75±5.38	0.26-9.25	
D	40(57.14%)	11.40±14.23	6.83-15.96	
Total	70(100%)	82±11.66	5.04-10.60	

DISCUSSION

COPD is a progressive inflammatory disease that affects multiple organ systems, and increasing evidence suggests that the systemic inflammation and endothelial dysfunction inherent to COPD may contribute to microvascular injury, reflected by elevated urinary albumin-to-creatinine ratio (UACR).[10,11] In our study, albuminuria was observed in more than half of the participants, and UACR increased steadily with GOLD-defined disease severity, supporting the hypothesis that worsening airflow limitation is associated with heightened systemic involvement.[12,13] This finding is biologically plausible, as chronic inflammation, oxidative stress, and persistent hypoxemia in COPD contribute to endothelial disruption, increased vascular permeability, and subsequent albumin leakage into urine.[14,15] The significant association between higher UACR and lower PaO_2 values observed in our study further reinforces this mechanism, aligning with earlier reports demonstrating a strong correlation between hypoxemia and microalbuminuria.[16-18]

The prevalence of albuminuria in our cohort is comparable with previous studies conducted in similar clinical settings. *Kömürcüoğlu et al.*, and *Agrawal et al.,* both documented high proportions of microalbuminuria among patients with acute exacerbations, reflecting the heightened inflammatory burden during unstable phases of the disease.[16,17] Although our study focused on clinically stable COPD, the persistence of elevated UACR even outside exacerbation episodes suggests that microvascular injury may represent a chronic, ongoing process rather than a transient inflammatory response. *Bozkus et al.,* similarly reported increasing UACR across GOLD risk categories, particularly in groups with higher symptomatology and future exacerbation risk, which mirrors our findings where GOLD D patients exhibited the highest UACR values.[7] These consistent patterns across multiple

studies underscore the potential of UACR as an adjunct marker of disease severity.

The demographic profile of our participants older age, female predominance, and high biomass exposure aligns with the epidemiological landscape of COPD in Nepal.[13-15] Nepal continues to experience a high burden of COPD, not only due to tobacco smoking but also because of widespread use of biomass fuels, particularly among women involved in household cooking. Biomass exposure is a recognized driver of systemic inflammation and endothelial stress, which may further contribute to increased UACR levels in this population.[18,19] Despite this, no published study from Nepal has previously evaluated UACR in relation to COPD severity, making our findings an important addition to the existing body of evidence. The unique environmental and sociodemographic characteristics of Nepalese patients may influence the expression of systemic biomarkers, highlighting the importance of local research.

The clinical implications of our findings are noteworthy. UACR is a simple, non-invasive, inexpensive test that is widely available, making it particularly suitable for low-resource settings. As our study demonstrates, elevated UACR correlates with disease severity and may serve as an early marker of systemic involvement, microvascular dysfunction, or potential cardiovascular complications, all of which are important contributors to morbidity and mortality in COPD. Previous studies have shown that COPD patients with microalbuminuria have higher cardiovascular risk, increased inflammation, and worse long-term outcomes.[17-19] Incorporating UACR testing into routine COPD assessment could therefore aid in identifying patients at increased risk who may benefit from closer monitoring or more intensive management.

CONCLUSIONS

In conclusion, this study demonstrates a significant association between urinary albumin-to-creatinine ratio and the severity of Chronic Obstructive Pulmonary Disease as classified by the GOLD ABCD system. Higher UACR levels were observed in patients with more advanced stages of COPD, suggesting that UACR could serve as a valuable biomarker for assessing disease severity and predicting exacerbation risk. The study also highlights the role of environmental factors, particularly indoor biomass smoke exposure, in exacerbating COPD, especially among women. Incorporating UACR testing into routine clinical practice could enhance risk stratification and lead to more personalized treatment approaches. However, further longitudinal studies are needed to establish causality and fully understand the long-term implications of UACR as a prognostic tool in COPD management.

Limitations

This study has several limitations. The cross-sectional design limits causal inference, and the relatively small sample size may reduce statistical power. Being a single-centre study with purposive sampling, the findings may not be fully generalizable and are subject to selection bias. UACR was measured using a single spot urine sample without repeated assessment, and the absence of multivariable analysis raises the possibility of residual confounding.

Acknowledgments

The authors would like to thank the patients who participated in this study and the staff of Tribhuvan University Teaching Hospital for their support during data collection. No external assistance was received for manuscript preparation.

Conflict of interest: The authors declare that they have no conflict of interest.

Funding: No funding was received from any agency to conduct this study.

Availability of data and materials

All data analysed during this study will be made available upon reasonable request from the corresponding author.

Authors' contributions

Conceptualization: Dr. Niraj Bam.

Data curation: Dr. Niraj Bam, Dr. Satish Kumar Das, Dr. Bibek Shrestha.

Formal analysis: Dr. Niraj Bam, Dr. Satish Kumar Das, Dr. Bibek Shrestha.

Investigation: Dr. Niraj Bam, Dr. Satish Kumar Das, Dr. Bibek Shrestha.

Methodology: Dr. Niraj Bam, Dr. Satish Kumar Das, Dr. Bibek Shrestha.

Supervision: Dr. Niraj Bam.

Writing-original draft: Dr. Niraj Bam, Dr. Bibek Shrestha.

Writing-review & editing: Dr. Niraj Bam, Dr. Satish Kumar Das, Dr. Bibek Shrestha.

Abbreviations

COPD-Chronic Obstructive Pulmonary Disease.
UACR-Urinary Albumin-to-Creatinine Ratio.
GOLD-Global Initiative for Chronic Obstructive Lung Disease.
CAT-COPD Assessment Test.
mMRC-Modified Medical Research Council Dyspnea Scale.

REFERENCES

1. Ayilya BL, Nazeer RA. Epidemiological burden, risk factors, and recent therapeutic advances in chronic obstructive pulmonary disease. J Adv Biotechnol Exp Ther. 2023 Sep 18;6:109-22. [DOI]
2. Hooper R, Burney P, Vollmer WM, McBurnie MA, Gislason T, Tan WC, Jithoo A, Kocabas A, Welte T, Buist AS. Risk factors for COPD spirometrically defined from the lower limit of normal in the BOLD project. European Respiratory Journal. 2012 May 31;39(6):1343-53. [DOI]
3. Ish P. An epidemiological study on risk factors of chronic obstructive pulmonary disease. Epidemiology International [Internet]. 2021 Mar 30;06(01):15-21. [DOI]
4. Laucho-Contreras ME, Cohen-Todd M. Early diagnosis of COPD: myth or a true perspective. European Respiratory Review. 2020 Dec 2;29(158). [DOI]
5. Barnes PJ. GOLD 2017: a new report. Chest. 2017 Feb 1;151(2):245-6. [DOI]
6. Cui YN, Chen P, Dai ZS, Chen Y. Changes in Global Initiative for Chronic Obstructive Lung Disease ABCD groups and the impact of regrouping on treatment: a comparison of 2017 and 2014. Chinese Medical Journal. 2018 May 5;131(9):1113-4. [DOI]
7. Bozkus F, Dikmen N, Samur A. Microalbuminuria in subjects with COPD: relationship to the new version of global initiative for chronic obstructive lung disease staging. Respiratory Care. 2017 Mar;62(3):307-14. [DOI]
8. Banur A, Ajit E, Bondade K, Ayana K, Raikar PS, Angadi S. A study on the utility of microalbuminuria as an indicator for the assessment of severity of chronic obstructive pulmonary disease and its co-morbidities. Journal of Clinical and Scientific Research. 2024 Jul 1;13(3):189-93. [DOI]
9. Gupta KK, Kotwal M, Atam V, Usman K, Chaudhary SC, Kumar A. Study of microalbuminuria in chronic obstructive pulmonary disease patients at tertiary care teaching hospital. Journal of Family Medicine and Primary Care. 2020 Aug 1;9(8):3916-20. [DOI]
10. Dobrowolski P, Januszewicz A, Gumprecht J, Małyszko J, Narkiewicz K, Stompor T, Tykarski A, Więcek A, Prejbisz A. Why albuminuria should be assessed more frequently in everyday clinical practice? A position statement. Pol Arch Intern Med. 2021 Apr 29;131(4):396-406. [DOI]
11. Shayo FK, Lutale J. Albuminuria in patients with chronic obstructive pulmonary disease: a cross-sectional study in an African patient cohort. BMC Pulmonary Medicine. 2018 Jul 31;18(1):125. [DOI]
12. Gupta P, Kumar A, Chaurasia AK, Gupta A. Prevalence and clinical significance of microalbuminuria and hypoxemia in patients with chronic obstructive pulmonary disease. [DOI]
13. Bhandari R, Sharma R. Epidemiology of chronic obstructive pulmonary disease: a descriptive study in the mid-western region of Nepal. International journal of chronic obstructive

pulmonary disease. 2012 Apr 4:253-7. [DOI]
14. Pudasaini S, Adhikari P, Maharjan S, Jha AK, Dhakal N. Role of CBC Parameters in COPD with Acute Exacerbation in a Tertiary Care Center. Nepal Medical College Journal. 2019 Dec 31;21(4):306-12. [DOI]
15. Adhikari TB, Acharya P, Högman M, Neupane D, Karki A, Drews A, Cooper BG, Sigsgaard T, Kallestrup P. Prevalence of chronic obstructive pulmonary disease and its associated factors in Nepal: findings from a community-based household survey. International journal of chronic obstructive pulmonary disease. 2020 Sep 29:2319-31. [DOI]
16. Bulcun E, Ekici M, Ekici A. Microalbuminuria in chronic obstructive pulmonary disease. [DOI]
17. Agrawal A, Garg R, Sahu D, Kumar M. Study the association of chronic obstructive pulmonary disease with early endothelial dysfunction and its impact on cardiovascular system by estimating urinary albumin creatinine ratio. Lung India. 2017 Mar 1;34(2):138-43. [DOI]
18. Romundstad S, Naustdal T, Romundstad PR, Sorger H, Langhammer A. COPD and microalbuminuria: a 12-year follow-up study. European Respiratory Journal. 2014 Mar 31;43(4):1042-50. [DOI]
19. Kaysoydu E, Arslan S, Yildiz G, Candan F. Factors related to microalbuminuria in patients with chronic obstructive pulmonary disease. Advances in Clinical and Experimental Medicine. 2014;23(5). [DOI]

Citation: Bam N, Das SK, Shrestha B. Association Between Urinary Albumin-to-Creatinine Ratio and COPD Severity: A Cross-Sectional Study in Nepal. JCMS Nepal. 2026;22(1):29-35.