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Research Article

# Serum Uric Acid as an Adjunctive Biomarker of Disease Severity in Patients with Stable Chronic Obstructive Pulmonary Disease: A Hospital-Based Cross-Sectional Analytical Study

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## Abstract

**Background:** Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation and systemic manifestations resulting from chronic inflammation, oxidative stress, and hypoxemia. Serum uric acid, a by-product of purine metabolism, has emerged as a potential biomarker reflecting oxidative stress and tissue hypoxia. This study evaluated the association between serum uric acid levels and disease severity in patients with stable COPD.

**Methods:** A hospital-based cross-sectional analytical study was conducted among 100 clinically stable COPD patients attending the Department of General Medicine, Sardar Patel Medical College and P.B.M. Associated Group of Hospitals, Bikaner. COPD severity was classified according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria using post-bronchodilator spirometry. Serum uric acid was estimated by the enzymatic uricase method. Correlation analyses, one-way ANOVA, multivariable linear regression, and receiver operating characteristic (ROC) curve analysis were performed to determine the association between serum uric acid and disease severity.

**Results:** The mean serum uric acid level was 6.94 ±1.72 mg/dL and increased significantly with advancing GOLD stage ($p<0.001$). Serum uric acid demonstrated a significant negative correlation with $FEV_1$% predicted ($r = -0.681$, $p<0.001$) and oxygen saturation ($r = -0.506$, $p<0.001$), while positive correlations were observed with CAT score ($r = 0.592$), mMRC dyspnea grade ($r = 0.548$), smoking exposure ($r = 0.438$), and exacerbation frequency ($r = 0.472$) (all $p<0.001$). Multivariable regression identified serum uric acid as an independent predictor of reduced $FEV_1$, and ROC analysis demonstrated good diagnostic performance for identifying severe COPD (AUC = 0.824).

**Conclusion:** Serum uric acid levels were significantly associated with worsening airflow limitation, symptom burden, hypoxemia, and disease severity in patients with stable COPD. Owing to its low cost, widespread availability, and significant association with clinical severity, serum uric acid may serve as a useful adjunctive biomarker

for disease assessment and risk stratification in routine clinical practice.

**Keywords:** Chronic Obstructive Pulmonary Disease; Serum Uric Acid; Biomarkers; Disease Severity; GOLD Classification; Spirometry; Oxidative Stress

## Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable, and treatable respiratory disorder characterized by persistent respiratory symptoms and progressive airflow limitation resulting from airway and/or alveolar abnormalities caused by significant exposure to noxious particles or gases [1]. It is a major global public health problem owing to its high prevalence, progressive course, and substantial contribution to morbidity, mortality, and healthcare utilization. According to the World Health Organization, COPD is among the leading causes of death worldwide, with a disproportionately higher burden in low- and middle-income countries due to increasing tobacco consumption, biomass fuel exposure, environmental pollution, and occupational hazards [2,3]. India contributes significantly to the global COPD burden, with prevalence estimates ranging from 4% to 10%, particularly among smokers and populations exposed to biomass smoke [4,5].

The pathogenesis of COPD extends beyond persistent airflow limitation and encompasses chronic inflammation, oxidative stress, protease-antiprotease imbalance, and structural remodeling of the airways and lung parenchyma [6,7]. Increasing evidence indicates that COPD is a multisystem disease with extrapulmonary manifestations resulting from chronic hypoxemia and systemic inflammation. These systemic effects contribute to disease progression, impaired functional capacity, frequent exacerbations, cardiovascular comorbidities, and poor clinical outcomes [8]. Although spirometry remains the gold standard for diagnosis and assessment of disease severity, it does not adequately reflect the systemic alterations associated with COPD progression [1]. Consequently, there has been growing interest in identifying simple, reliable, and inexpensive biomarkers that complement spirometric assessment and provide additional prognostic information.

Serum uric acid, the final product of purine metabolism catalyzed by xanthine oxidase, has emerged as a promising biomarker in COPD. Under physiological conditions, uric acid functions as an important antioxidant by scavenging reactive oxygen species; however, chronic hypoxemia enhances adenosine triphosphate degradation and purine metabolism, resulting in increased serum uric acid production [9]. Elevated serum uric acid levels have been associated with oxidative stress, endothelial dysfunction, systemic inflammation, and impaired tissue oxygenation, all of which contribute to the progression of COPD [10].

Several clinical studies have demonstrated that higher serum uric acid levels are associated with worsening airflow limitation, reduced exercise capacity, increased frequency of exacerbations, prolonged hospitalization, cardiovascular comorbidities, and higher mortality among patients with COPD [11,12]. Owing to its low cost, widespread availability, and ease of estimation, serum uric acid has attracted considerable attention as a potential adjunctive biomarker for assessing disease severity and prognosis. However, evidence regarding its clinical utility, particularly among the Indian population, remains limited.

Therefore, the present study was undertaken to evaluate the association between serum uric acid levels and the severity of COPD as assessed by GOLD classification and clinical severity parameters. The findings may help establish serum uric acid as a practical adjunctive biomarker for evaluating disease severity and identifying patients at greater risk of adverse clinical outcomes.

## Materials and Methods

This hospital-based cross-sectional analytical study was conducted in the Department of General Medicine, Sardar Patel Medical College

and P.B.M. Associated Group of Hospitals, Bikaner, Rajasthan, over a period of six months after obtaining approval from the Institutional Ethics Committee. The study included clinically stable patients aged ≥40 years with a confirmed diagnosis of Chronic Obstructive Pulmonary Disease (COPD) according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria, defined by a post-bronchodilator forced expiratory volume in one second to forced vital capacity ($FEV_1/FVC$) ratio of <0.70. Patients with acute exacerbation within the preceding four weeks, significant cardiac failure (NYHA class III–IV), chronic liver disease, advanced chronic kidney disease (eGFR <30 mL/min/1.73 $m^2$), active malignancy, systemic inflammatory disorders, recent major surgery or trauma, pregnancy, or those receiving medications known to significantly influence serum uric acid levels were excluded.

The sample size was calculated using Fisher's Z transformation formula for estimating a correlation coefficient, assuming an expected correlation coefficient (r) of 0.30, 95% confidence level, and 80% power, yielding a minimum required sample size of 85 participants. To compensate for potential dropouts and improve statistical precision, a total of 100 consecutive eligible patients fulfilling the inclusion and exclusion criteria were enrolled after obtaining written informed consent.

A detailed clinical evaluation was performed for all participants, including demographic characteristics, smoking status and smoking exposure (pack-years), duration of COPD, history of exacerbations during the previous 12 months, associated comorbidities, and current treatment. Body mass index (BMI) was calculated using standard anthropometric measurements. Symptom burden was assessed using the Modified Medical Research Council (mMRC) dyspnea scale and the COPD Assessment Test (CAT). Spirometry was performed before and after bronchodilator administration according to standard recommendations, and post-bronchodilator $FEV_1$ percentage predicted values were used to classify disease severity into GOLD stages I–IV.

Venous blood samples were collected under aseptic precautions, preferably in the fasting state, and serum uric acid levels were estimated using the standard enzymatic uricase method in the institutional central laboratory following standard operating procedures. Routine laboratory investigations were performed as clinically indicated.

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0. Continuous variables were expressed as mean ±standard deviation, while categorical variables were presented as frequencies and percentages. Data normality was assessed using the Shapiro–Wilk test. Comparison of serum uric acid levels across GOLD stages was performed using one-way analysis of variance (ANOVA). Correlation between serum uric acid levels and clinical severity parameters, including $FEV_1$% predicted, CAT score, mMRC dyspnea grade, oxygen saturation, smoking exposure, and exacerbation frequency, was evaluated using Pearson's correlation coefficient. Multivariable linear regression analysis was performed to identify independent predictors of reduced $FEV_1$% predicted after adjusting for relevant covariates. Receiver operating characteristic (ROC) curve analysis was carried out to assess the diagnostic performance of serum uric acid in predicting severe COPD (GOLD stage III–IV). A p-value <0.05 was considered statistically significant.

The study was approved by the Institutional Ethics Committee of Sardar Patel Medical College, Bikaner. Written informed consent was obtained from all participants before enrollment, and confidentiality and anonymity of participant information were maintained throughout the study.

## Results

A total of 100 clinically stable patients with COPD were included in the study. The baseline demographic and clinical characteristics of the study population are summarized in Table 1. The majority of participants were males (74%) with a mean age of 63.48 ± 9.12 years. Most patients belonged to the GOLD II (39%) and GOLD III

(34%) categories. Smoking was common, with 82% of participants being current or former smokers. The mean smoking exposure was 13.05 ± 7.95 pack-years. The mean body mass index was 21.84 ± 3.76 kg/m², while the mean post-bronchodilator $FEV_1$ was 48.62 ± 18.94% predicted.

**Table 1.** Baseline Demographic and Clinical Characteristics of the Study Participants (n = 100)

| Variable | Value |
|---|---|
| Age (years), Mean ± SD | 63.48 ± 9.12 |
| Male sex, n (%) | 74 (74.0) |
| Rural residence, n (%) | 78 (78.0) |
| Current/Former smokers, n (%) | 82 (82.0) |
| Smoking exposure (pack-years), Mean ± SD | 13.05 ± 7.95 |
| BMI (kg/m²), Mean ± SD | 21.84 ± 3.76 |
| $SpO_2$ on room air (%), Mean ± SD | 91.62 ± 4.18 |
| Duration of COPD (years), Mean ± SD | 8.36 ± 4.72 |
| CAT score, Mean ± SD | 18.94 ± 6.48 |
| mMRC dyspnea grade, Mean ± SD | 2.71 ± 0.91 |
| $FEV_1$ (% predicted), Mean ± SD | 48.62 ± 18.94 |
| GOLD I, n (%) | 12 (12.0) |
| GOLD II, n (%) | 39 (39.0) |
| GOLD III, n (%) | 34 (34.0) |
| GOLD IV, n (%) | 15 (15.0) |

Serum uric acid levels increased progressively with worsening COPD severity. A statistically significant difference in mean serum uric acid levels was observed across the GOLD stages (Table 2).

**Table 2.** Comparison of Serum Uric Acid Levels Across GOLD Stages

| GOLD Stage | n | Serum Uric Acid (mg/dL), Mean ± SD |
|---|---|---|
| GOLD I | 12 | 5.02 ± 0.84 |
| GOLD II | 39 | 6.08 ± 1.12 |
| GOLD III | 34 | 7.64 ± 1.26 |
| GOLD IV | 15 | 9.02 ± 1.48 |

**Statistical analysis:** One-way ANOVA; $F = 34.86$; $p < 0.001$

Serum uric acid demonstrated significant correlations with spirometric, clinical, and functional parameters of COPD severity (**Table 3**).

**Table 3.** Correlation Between Serum Uric Acid and Clinical Severity Parameters

| Parameter | Correlation coefficient (r) | p-value |
|---|---|---|
| $FEV_1$ (% predicted) | -0.681 | <0.001 |
| CAT score | 0.592 | <0.001 |
| mMRC dyspnea grade | 0.548 | <0.001 |
| $SpO_2$ (%) | -0.506 | <0.001 |
| Exacerbation frequency | 0.472 | <0.001 |
| Smoking pack-years | 0.438 | <0.001 |

Multivariable linear regression analysis identified serum uric acid as an independent predictor of reduced $FEV_1$ after adjustment for age, body mass index, and smoking exposure (Table 4).

**Table 4.** Multivariable Linear Regression Analysis for Predictors of Reduced $FEV_1$ (% Predicted)

| Variable | β Coefficient | Standard Error | p-value |
|---|---|---|---|
| Serum uric acid | -2.14 | 0.72 | 0.004 |
| Age | -0.38 | 0.14 | 0.008 |
| BMI | 0.72 | 0.29 | 0.015 |
| Smoking pack-years | -0.46 | 0.11 | <0.001 |

**Model performance:** $R^2 = 0.684$; Adjusted $R^2 = 0.661$; Overall model $p < 0.001$

Receiver operating characteristic analysis demonstrated that serum uric acid had good discriminatory ability for identifying severe COPD (GOLD III–IV), with an area under the curve of 0.824 (Table 5).

**Table 5.** Diagnostic Performance of Serum Uric Acid for Predicting Severe COPD

| Cut-off (mg/dL) | AUC (95% CI) | Sensitivity (%) | Specificity (%) | p-value |
|---|---|---|---|---|
| >7.1 | 0.824 (0.742–0.905) | 79.6 | 76.5 | <0.001 |

## DISCUSSION

The present hospital-based cross-sectional analytical study evaluated the association between serum uric acid levels and disease severity among patients with stable Chronic Obstructive Pulmonary Disease (COPD). The findings demonstrated that serum uric acid levels increased progressively with advancing GOLD stage and showed significant correlations with airflow limitation, symptom burden, hypoxemia, smoking exposure, and exacerbation frequency. Furthermore, serum uric acid emerged as an independent predictor of reduced lung function and demonstrated good diagnostic accuracy for identifying severe COPD. These observations support the growing evidence that serum uric acid is a useful surrogate marker of chronic hypoxia, oxidative stress, and disease severity in COPD.

The mean serum uric acid level in the present study was 6.94 ±1.72 mg/dL, Serum uric acid increased significantly from 5.02 ±0.84 mg/dL in GOLD I patients to 9.02 ±1.48 mg/dL in GOLD IV patients, indicating a strong relationship between biomarker levels and worsening disease severity. Similar findings have been reported by Wattanachayakul et al. [13], who demonstrated significantly higher serum uric acid levels among COPD patients compared with healthy individuals. Kocak et al. [14] also reported that serum uric acid concentrations increased progressively with disease severity and were significantly associated with recurrent exacerbations. Likewise, Rumora et al. [15] observed that elevated serum uric acid and uric acid-to-creatinine ratio were associated with worsening pulmonary function, greater symptom burden, and poorer quality of life. These findings suggest that increasing serum uric acid reflects the cumulative effects of chronic hypoxemia and oxidative stress accompanying disease progression.

A significant inverse correlation was observed between serum uric acid levels and $FEV_1$% predicted ($r = –0.681$, $p < 0.001$), indicating that higher serum uric acid concentrations were associated with greater airflow limitation. Similar observations have been reported in the COSYCONET study, where Kahnert et al. [12] demonstrated significant associations between elevated serum uric acid levels and reduced lung function, impaired exercise capacity, and cardiovascular comorbidities. Garcia-Pachon et al. [16] also reported an inverse relationship between serum uric acid and pulmonary function parameters in patients with COPD. These findings support the concept that serum uric acid

reflects the severity of chronic hypoxia and progressive deterioration of pulmonary function.

Serum uric acid also demonstrated significant positive correlations with CAT score, mMRC dyspnea grade, smoking exposure, and exacerbation frequency, while showing a significant negative correlation with oxygen saturation. These observations indicate that elevated serum uric acid is associated not only with physiological impairment but also with increased symptom burden and clinical instability. Bartziokas et al. [11] reported that elevated serum uric acid independently predicted future exacerbations and mortality among patients with COPD. Similarly, Zhao and Lv [17] demonstrated positive correlations between blood uric acid levels and disease severity in patients with COPD, further supporting its clinical utility as a marker of adverse outcomes.

Multivariable linear regression analysis identified serum uric acid as an independent predictor of reduced $FEV_1$ after adjustment for age, body mass index, and smoking exposure. In addition, receiver operating characteristic analysis demonstrated good diagnostic performance of serum uric acid for predicting severe COPD, with an area under the curve of 0.824. These findings suggest that serum uric acid may provide valuable adjunctive information beyond conventional spirometric assessment. As serum uric acid estimation is inexpensive, widely available, and routinely performed in clinical laboratories, its incorporation into the clinical evaluation of COPD patients may facilitate early identification of individuals at higher risk of severe disease and adverse outcomes.

The present study has certain limitations. Being a single-center cross-sectional study, causal relationships between serum uric acid and disease progression cannot be established. The relatively modest sample size may also limit the generalizability of the findings. Nevertheless, the study provides important evidence supporting the association of serum uric acid with COPD severity in an Indian population. Further large-scale prospective multicentric studies are warranted to validate these findings and determine the prognostic role of serum uric acid in predicting long-term clinical outcomes among patients with COPD.

## CONCLUSION

The present study demonstrated that serum uric acid levels increased significantly with advancing severity of Chronic Obstructive Pulmonary Disease and were strongly associated with reduced lung function, greater symptom burden, hypoxemia, smoking exposure, and frequent exacerbations. Serum uric acid showed a significant inverse correlation with $FEV_1$% predicted and emerged as an independent predictor of airflow limitation after adjustment for relevant clinical variables. Additionally, it demonstrated good diagnostic accuracy for identifying patients with severe COPD. These findings suggest that elevated serum uric acid reflects the underlying oxidative stress and chronic hypoxic state associated with disease progression. Considering its simplicity, affordability, and widespread availability, serum uric acid may serve as a valuable adjunctive biomarker alongside spirometry for assessing disease severity and identifying patients at increased risk of adverse clinical outcomes. Further large-scale prospective multicentric studies are recommended to validate its prognostic utility and establish its role in routine clinical practice.

## DECLARATIONS

**Funding:** Nil.

**Conflicts of Interest:** The authors declare no conflicts of interest.

**Ethical Approval:** The study was approved by the Institutional Ethics Committee of Sardar Patel Medical College, Bikaner, Rajasthan.

**Informed Consent:** Written informed consent was obtained from all participants prior to enrollment.

**Author Contributions:** All authors contributed to the study conception, data collection, analysis, manuscript preparation, and approved the final manuscript.

**Data Availability:** The datasets generated and/or analyzed during the current study are available

from the corresponding author on reasonable request.

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**Use of Artificial Intelligence**

The authors declare that no artificial intelligence tools were used in the conduct, analysis, or preparation of this research.

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