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Hyperuricemia and its Role in Assessing Severity of COPD: Evidence from a Hospital-Based Study

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ABSTRACT: ***Background:*** Chronic Obstructive Pulmonary Disease (COPD) is a chronic debilitating disease. Oxidative stress plays a crucial role in COPD pathogenesis, and serum uric acid (SUA), a marker of oxidative stress, may serve as an indicator of disease severity. ***Aim:*** This study aims to evaluate the relationship between serum uric acid levels and COPD severity among patients at Rajshahi Medical College Hospital. ***Methods:*** A cross-sectional study was conducted among 162 COPD patients diagnosed with GOLD guidelines. Spirometry was performed to classify disease severity into mild, moderate, severe, and very severe groups. Serum uric acid levels were measured using the Enzymatic-Colorimetric Trinder Endpoint method. Statistical analyses were conducted using SPSS 26, and associations between SUA levels and COPD severity were determined using the chi-square test, with $p < 0.05$ considered statistically significant. ***Results:*** The mean age of participants was 64.1 ± 9.9 years, with a 97.5% male predominance. A significant proportion of patients had moderate (38.3%) and severe (29.6%) COPD, with only 16.7% having mild and 15.4% having very severe disease. Mean serum uric acid levels were significantly higher in patients with more severe COPD (5.2 ± 1.2 mg/dL in mild vs. 8.03 ± 1.5 mg/dL in very severe cases, $p < 0.001$). Additionally, serum uric acid levels correlated positively with disease duration, with the highest levels observed in patients with >10 years of COPD (7.9 mg/dL, $p < 0.05$). Among patients with a history of acute exacerbations, 94.1% had elevated SUA levels ($p = 0.033$). ***Conclusion:*** Serum uric acid levels correlate positively with COPD severity and disease duration, indicating its potential as a biomarker for disease progression. Monitoring SUA levels may aid in risk stratification and management of COPD, particularly in resource-limited settings.

Keywords: COPD, Serum Uric Acid, Oxidative Stress, Spirometry, Disease Severity, Biomarker.

Article at a glance:

Study Purpose*: To assess the relationship between serum uric acid levels and the severity of COPD in hospitalized patients.*
Key findings: *Higher serum uric acid levels were significantly associated with increased COPD severity, disease duration, and exacerbation frequency.*
Newer findings: *SUA may serve as a cost-effective biomarker for COPD severity assessment and progression monitoring in clinical settings.*
Abbreviations: *COPD – Chronic Obstructive Pulmonary Disease, SUA – Serum Uric Acid, GOLD – Global Initiative for Chronic Obstructive Lung Disease*

INRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by irreversible airway obstruction, leading to a persistent decline in lung function. This obstruction results from an abnormal inflammatory response of the lungs to prolonged exposure to noxious particles, including cigarette smoke, biomass fuel, and industrial pollutants.[1] COPD is a leading cause of morbidity and mortality worldwide, with a significant and growing disease burden. The global prevalence of COPD was estimated to be 10.6%, accounting for approximately 480 million cases in 2020.[2] This prevalence is projected to increase by 112

million by 2050, reflecting a 23.3% relative increase 2. Nearly 90% of COPD-related deaths occur in individuals aged 70 years and above, with the burden of mortality rising at a much faster rate in low- and middle-income countries compared to the West.[3, 4] In Bangladesh, approximately six million people suffer from COPD, and mortality is nearly four times higher than in Western populations.[5] The pathophysiology of COPD is complex and involves both local and systemic inflammation, oxidative stress, and structural damage to the lungs 6,7. Inflammation in COPD is driven by an influx of neutrophils, macrophages, and cytotoxic T lymphocytes into the airways.[8, 9] These immune cells generate excessive levels of reactive oxygen species (ROS) and reactive nitrogen species (RNS), which further contribute to oxidative stress, apoptosis, and tissue destruction.[10-12] Several pro-inflammatory cytokines, including IL-1, IL-3, IL-6, TNF-α, and TGF-β, play critical roles in disease progression. [13, 14] While TNF-α, IL-1β, and IL-6 amplify the inflammatory response, TGF-β induces fibrosis of the small airways, leading to airflow limitation and structural remodeling.[15-17] The combination of chronic airway inflammation and oxidative stress results in progressive alveolar destruction, impaired lung elastic recoil, and increased susceptibility to exacerbations. Among the various markers of oxidative stress, serum uric acid (SUA) has emerged as a potential biomarker for COPD severity. Uric acid is a key extracellular antioxidant in the airway epithelium, with a protective role against oxidative damage when maintained at physiological levels (≤5 mg/dL)18 which at higher concentrations, its antioxidant properties diminish, and it becomes pro-oxidant, contributing to systemic inflammation and oxidative damage. Hypoxia, a hallmark of COPD, induces purine metabolism, leading to the degradation of adenosine to hypoxanthine and xanthine, which are subsequently converted into uric acid by xanthine oxidase.[19] This process results in elevated serum uric acid levels in COPD patients, correlating with disease progression and severity.[20]

Hyperuricemia in COPD has been linked to both protective and detrimental effects.[20] Recent evidence suggests that serum uric acid levels correlate with disease severity, making it a potential biomarker for assessing COPD progression.[21-24] This is particularly relevant in resource-constrained settings where spirometry is not always readily available. The ability to utilize serum uric acid as a surrogate marker could aid in the early identification and stratification of COPD patients, guiding appropriate disease management strategies.

Aims and Objectives

This study aims to assess the association between serum uric acid levels and the severity of COPD in patients attending Rajshahi Medical College Hospital

MATERIALS AND METHODS

This descriptive type of cross-sectional study was conducted at the Medicine Outpatient Department (OPD) and the Department of Medicine, Rajshahi Medical College Hospital, Rajshahi, Bangladesh, over a period of two years from July 2021 to June 2023. The study included patients diagnosed with COPD attending the OPD and those on follow-up after hospitalization for acute exacerbation. Patients were enrolled based on specific eligibility criteria. Inclusion criteria consisted of clinically and Spiro metrically confirmed COPD as per GOLD guidelines and age above 40 years. Patients with asthma-COPD overlap (ACO), acute exacerbation of COPD, left heart failure, chronic kidney disease, decompensated liver disease, malignancy, a history of gout, or those taking thiazide diuretics were excluded. The sample size was calculated using the single proportion estimate formula based on an anticipated COPD prevalence of 12.5%, yielding a required minimum of 162 participants at a 95% confidence level with a 5% margin of error. Patients were selected using a purposive sampling technique.[25]

The diagnosis of COPD was based on clinical signs and symptoms, smoking history, laboratory tests, and spirometry parameters. After obtaining written informed consent, clinical data including age, gender, symptoms, and smoking habits were recorded. Serum uric acid levels were measured using the Enzymatic-Colorimetric Trinder Endpoint method, with the upper limit defined as 7 mg/dL for males and 6 mg/dL for females. Spirometry was performed following standard guidelines, where bronchodilator medications (400 µg Salbutamol via a metered-dose inhaler) were administered, and FEV_1 was measured after 15–20 minutes. The highest value from at least three FVC maneuvers was recorded, ensuring acceptability and repeatability criteria, and interpreted by a pulmonologist. Airflow limitation was classified as mild (FEV_1 ≥80% predicted),

moderate (FEV_1 50%–79% predicted), severe (FEV_1 30%–49% predicted), and very severe (FEV_1 <30% predicted). Data collection was carried out using structured questionnaires, spirometry, and biochemical assessments. Statistical analysis was performed using SPSS 26, with continuous variables expressed as mean ± standard deviation (SD) and categorical variables as frequency (percentage). Associations between variables were determined using the Chi-square test, with a p-value of <0.05 considered statistically significant. Ethical approval for the study was obtained from the Ethical Committee of Rajshahi Medical College. All participants were provided with detailed information about the study objectives, procedures, potential risks, and benefits in their local language before obtaining written informed consent. Confidentiality of all patient data was strictly maintained, and ethical research guidelines were followed throughout the study.

RESULTS

A total of 162 COPD patients were included in the study, with a mean age of 64.1 ± 9.9 years. The majority of participants were in the 61-70 years age group (42.6%), followed by 51-60 years (24.7%) and 71-80 years (19.1%). Only 4.3% of patients were above 80 years, and 9.3% were aged ≤50 years. The study population was predominantly male (97.5%). Regarding smoking habits, 69.1% of patients had a smoking history of 10-19 pack-years, followed by 26.5% with <10 pack-years. Only 0.6% had a smoking history of 20-29 pack-years, while 3.7% were non-smokers. The duration of COPD varied, with 37% of patients having COPD for more than 10 years, 34% for 6-10 years, and 29% for 1-5 years.

Table 1: Distribution of the Participants According to Demographic Characteristics (n=162)

Variables	Frequency	Percentage
≤ 50 years	15	9.30%
51-60 years	40	24.70%
61-70 years	69	42.60%
71-80 years	31	19.10%
> 80 years	7	4.30%
mean±SD	**64.13±9.9**	
Gender		
Male	158	97.5
Smoking habit		
Nonsmoker	6	3.7
<10 pack year	43	26.54
10-19 pack year	112	69.14
20-29 pack year	1	0.62
Duration of COPD		
1-5 years	47	29
6-10 years	55	34
>10 years	60	37

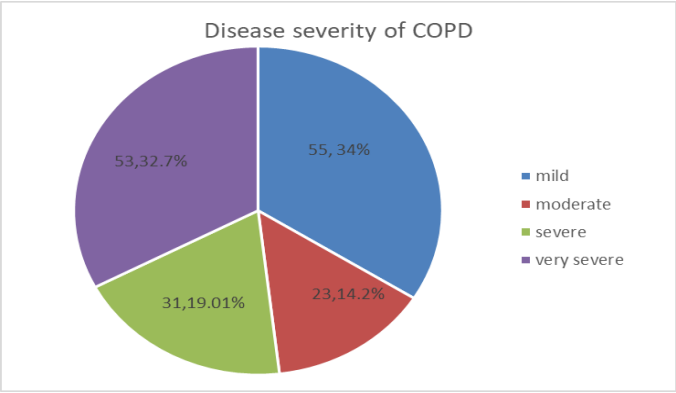

Figure 1: Distribution Of the Participants According to Disease Severity of COPD (N=162)

COPD Severity Distribution

Based on spirometric classification, 16.7% of patients had mild COPD, 38.3% had moderate COPD, 29.6% had severe COPD, and 15.4% had very severe COPD (Figure 1). This distribution suggests that a significant proportion of the study population had advanced disease, emphasizing the need for better management strategies.

Table 2: Difference of Means of Serum Uric Acid Level Among Severity Class of COPD

Serum uric acid	Disease severity				P value
	Mild	Moderate	Severe	Very severe	
mean±SD	5.2±1.2	7.03±1.3	7.6±1.0	8.03±1.5	**<0.001**[a]

a= one way ANOVA

Serum Uric Acid and COPD Severity

The mean serum uric acid level was significantly higher in patients with more severe COPD. Patients with mild COPD had a mean uric acid level of 5.2 ± 1.2 mg/dL, while those with moderate, severe, and very severe COPD had levels of 7.03 ± 1.3 mg/dL, 7.6 ± 1.0 mg/dL, and 8.03 ± 1.5 mg/dL, respectively. The difference in serum uric acid levels across disease severity classes was statistically significant ($p < 0.001$) (Table 2).

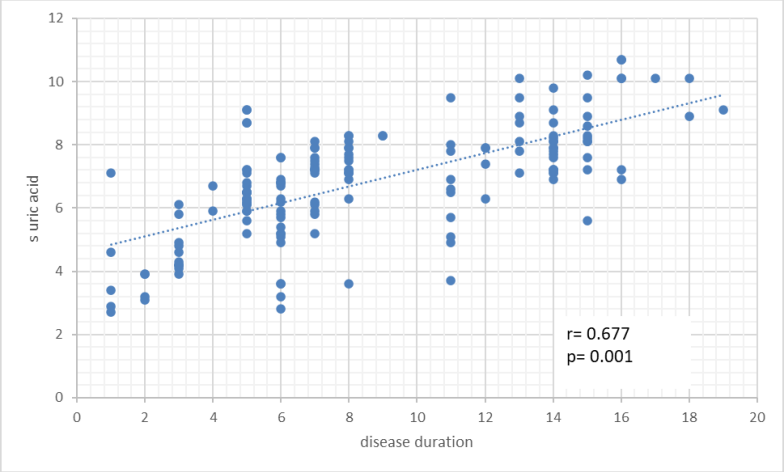

Figure 2: Correlation Among Disease Duration and Serum Uric Acid Level (N=162)

Correlation Between Serum Uric Acid and Disease Duration

A positive correlation was observed between disease duration and serum uric acid levels (Figure 2). Among patients with a disease duration of 1-5 years (29%), the mean serum uric acid level was 5.9 mg/dL, while those with 6-10 years of COPD (34%) had a mean uric acid level of 7.1 mg/dL. Patients with more than 10 years of disease duration (37%) had the highest mean uric acid level (7.9 mg/dL).

Table 3: Association Between Serum Uric Acid Level and History of Exacerbation of COPD In Last 1 Year (N=162)

H/O of COPD exacerbation	Uric acid level		P value
	Normal (≤5 mg/dL)	Increased (>5mg/dL)	
Yes	3 (6.3%)	48 (94.1%)	**0.033**[b]
No	22 (19.8%)	89 (80.2%)	

b= fisher's exact test

Exacerbation of COPD and serum uric acid level

Patients with acute exacerbation history in the last year had 94.1% of patients with increased serum uric acid level (p=0.033).

DISCUSSION

Under normal physiological conditions, uric acid functions as an antioxidant, protecting against oxidative damage from reactive oxygen and nitrogen species (ROS/RNS) generated by inhaled pollutants, cigarette smoke, and microorganisms.[26] In the respiratory tract, uric acid is secreted with mucus and plays a key role in counteracting oxidative stress.[27] However, its levels are influenced by various demographic and clinical factors, including age, smoking, BMI, serum glucose, creatinine, lipid profile, and systemic inflammation.[18, 28, 29] Pulmonary function declines with long-term smoke exposure, leading to tissue hypoxia in COPD patients. Hypoxia triggers purine metabolism, resulting in increased uric acid production. While uric acid initially serves as an antioxidant, excessive levels may contribute to oxidative stress and inflammation, potentially worsening disease progression.

In the present study, mean age of patients was 64.13±9.94 years with male predominance (97.5%). It was found that COPD incidence was higher after the age of 40 and in male gender.[28–31] More than half of the participants were moderate smoker; consuming for about 10-19 pack year which was evidenced that smoking and COPD have a significant interdependent relation.[28, 29, 31–33] 16.7% of patients had mild COPD, 38.3% had moderate COPD, 29.6% had severe COPD, and 15.4% had very severe COPD. Our study found that an increase in uric acid levels with increasing severity of the disease. Advanced GOLD stages (stages 3 and 4) Chronic obstructive pulmonary disease cases had higher uric acid level in comparison to stage 1 and 2. The most probable reasons between the association of deranged pulmonary functions and raised uric acid may be hypoxia leading to purine catabolism, pulmonary hypertension resulting in the elevation of uric acid, toxins in cigarette smoke causing oxidative stress in the alveolar spaces of the lungs, leading to lung inflammation which leads to the development of chronic respiratory diseases, such as Chronic obstructive pulmonary disease .[34] Similar observations were found in numerous international original articles.[28–33, 35–37]

The present study also found longer duration of disease had a linear positive significant correlation with serum uric acid level. This notion supports the hypothesis of chronic inflammation and its link to hyperuricemia .[30] In the past year, who had a history of acute exacerbation of COPD, about 94% of them had elevated level of serum uric acid. A close observation was found in several case control studies .[29, 32, 33, 37, 38]

Summarizing the finding of this study, it is evident that there is a strong association between elevated serum uric acid level on different stages of COPD.

CONCLUSION

Higher uric acid levels were observed in patients with advanced disease stages and longer disease duration, suggesting its potential role as a biomarker for disease progression. Serum uric acid may serve as a useful adjunct in assessing COPD severity, particularly in resource-limited settings where spirometry is unavailable.

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