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The Relationship Between Albumin, Uric Acid, and Uric Acid/Albumin Ratio and the Severity of Hypoxaemia in Patients with Chronic Obstructive Pulmonary Disease During Acute Exacerbation Periods

İmren MUTLU HAYAT[1]

[1]Department of Chest Diseases, Mehmet Akif İnan Training and Research Hospital, University of Health Sciences, Şanlıurfa, Türkiye

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Abstract

Objective

Studies in patients with chronic obstructive pulmonary disease have demonstrated an association with serum uric acid levels. In this study, we aimed to evaluate whether uric acid and uric acid/albumin levels could be used as predictors of disease severity in and execerbation COPD patients according to COPD stages and between COPD and healthy individuals.

Material and Method

This study included patients with COPD and healthy controls. Serum uric acid levels and uric acid/albumin ratios were assessed in each group, and their correlations with study parameters were investigated.

Results

A total of 141 individuals were included in our study; 103 of these were COPD patients (87 men and 16 women) with a mean age of 65.60 ± 9.17, and 38 were healthy controls. When comparing mild and moderate COPD (GOLD1, GOLD2) with severe and profound COPD (GOLD3, GOLD4), CRP levels and annual exacerbation numbers were statistically significantly higher in the advanced COPD group (p<0.001 and p=0.042, respectively). When the COPD group was compared with the healthy group, urea, creatinine, uric acid/albumin, CRP, WBC, and eosinophil percentage were higher in the COPD group (p=0.002, p=0.010, p<0.001, p=0.002, p=0.018, p=0.037, respectively). When comparing the acute exacerbation and stable period of COPD, uric acid/albumin, CRP, and leukocyte counts were found to be higher in the acute period (p=0.001, p=0.048, p=0.042).

Conclusion

Serum uric acid levels and uric acid/albumin ratios in COPD patients were considered to be useful in predicting COPD ex-acerbation risk and disease severity.

Keywords: Albumin, Chronic obstructive pulmonary disease, pulmonary function test, Uric acid

Correspondence: İ.M.H. / Imrendr21@gmail.com
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ORCID IDs of the Authors: İ.M.H: 0009-0005-4772-2507

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous lung disorder characterized by chronic respiratory symptoms (dyspnea, cough, sputum production, and/or exacerbations) and causes persistent, often progressive airflow obstruction (1). COPD is a complex and heterogeneous disease that exhibits significant variability both biologically and clinically, and even overtime in each patient (2). COPD is a chronic inflammatory disease in which lung balance is disrupted during a progressive process, leading to the development of hypoxemia. Tissue hypoxemia accelerates anaerobic metabolism, which in turn causes the breakdown of adenosine triphosphate. This event results in increased uric acid (3-5). Studies suggest that uric acid and uric acid/creatinine ratios may be used as biomarkers to assess disease severity and progression in frequent exacerbations and the acute phase of COPD (5, 6). Studies have shown that albumin levels are low and CRP levels are high during acute exacerbations of COPD (7). COPD is a debilitating disease that imposes economic burdens. The aim is to contribute to the early implementation of measures to alleviate disease severity in COPD patients by using readily accessible biomarkers such as uric acid and albumin.

Material and Method

Patients diagnosed with COPD and followed up at the chest diseases outpatient clinic were included in the study. The study was conducted to examine the change in serum uric acid and uric acid/albumin distribution according to COPD exacerbations, stages, and hypoxemic conditions. The study was conducted between May 2025 and November 2025 on patients registered at the Chest Diseases Outpatient Clinic of Mehmet Akif İnan Training and Research Hospital, University of Health Sciences.

Study Population and Sample

Our study was conducted to investigate the relationship between serum biomarkers in COPD patients. The study was initiated following approval by the ethics committee (HRÜ/25.10.32). Patients aged 40-90 years of both genders who had COPD for at least 2 years, had FEV1 <80%, and presented to our clinic with an acute exacerbation were included in the study. The exclusion criteria for the study were not having a diagnosis of COPD, having metabolic, renal, or hepatic disease that could affect uric acid levels, using antituberculosis or antineoplastic drugs, using drugs such as levodopa and allopurinol, Patients with gout, hyperparathyroidism, renal transplant, renal insufficiency, decompensated heart failure, or chronic alcohol use were excluded from the study. This study was conducted in accordance with the principles outlined in the Helsinki Declaration, and cases presenting to our outpatient clinic were included in the study voluntarily. The demographic values, age, disease duration, body mass index (BMI), clinical data, long-term oxygen therapy (LTOT), additional probability, and smoking habits of COPD patients were recorded through face-to-face interviews and from patient files. Comorbidities were recorded according to previous developments. Systemic arterial hypertension, heart disease, obstructive sleep apnea (OSA), malignant tumors, and medications used by patients were inquired about. Patients were classified according to the COPD GOLD (Global Initiative for Chronic Obstructive Lung Disease) classification using plain chest radiography (anterior, posterior, and lateral views), BTL “Cardio Point-Spiro,” and spirometry. According to the pulmonary function test, patients with a forced expiratory volume in 1 second (FEV1) value of 79% or below are classified as having COPD: Mild (FEV1≥80%), Moderate (79-50%), Severe (49-30%), and Very Severe (≤30%) (1). Arterial blood gas analyses were performed after a 30-minute rest in ambient air to measure partial oxygen (PaO2 mmHg) and oxygen saturation (SaO2) levels. Biochemical markers. CRP (mg/L) (C-Reactive Protein), urea (mg/dl), creatinine (mg/dl), uric acid (mg/dl), albumin (g/L), total protein (g/L), and WBC ($X10^3$) (White Blood Count) were determined using the radioimmunoassay method.

Statistical Analysis

Statistical analyses were performed using SPSS version 26.0 (IBM®, Chicago, USA). Analyses were conducted visually (histograms and probability plots) and analytically (Shapiro-Wilk test) based on the normal configuration of the variables. Descriptive statistics were expressed as minimum-maximum values indicating normal distribution, median, mean, and standard deviation in digital data. In uncut data (min-max) and nominal data, normal distribution was shown as number and percentage.

Variables showing normality were controlled in acute and stable groups using independent t-tests for serum biomarkers and SFTs (FEV1, FVC, FEV1/FVC). Analyses were performed using uric acid as a continuous variable. Linear regression analyses were performed to analyze the relationship between uric acid, uric acid/albumin levels, and oxygen saturation. These ratios were tested in multiple regression analyses of uric acid and albumin according to age, respiratory rate, and biochemical values. The Mann-Whitney U test and chi-square test were recorded under the assumption of normal distribution. P values of 0.05 were considered significant in the permanent analyses performed in the study.

Results

A total of 103 COPD patients, including 87 men and 16 women with an average age of 65.60 ± 9.17 years, and 38 healthy individuals as the control group, were included in our study, for a total of 141 participants. Comorbidity was present in 58% of patients; 13 cases (12.62%) had never smoked, 41 (39.80%) had quit smoking, and 49 (47.57%) were current smokers. LTOT use was identified in 36 (34.95%) and non-use in 67 (65.04%). Forty-two percent of these patients were receiving inpatient treatment. According to the GOLD classification, the highest number of COPD cases was in GOLD2 and GOLD3 stages. Accordingly, a larger group of patients was observed in GOLD Stage 3. The medication use and demographic data of the cases are presented in Table 1.

Table 1 Demographic Findings of Patients with COPD

Characteristics of the Cases	**n (%) (min-max)**	**Mean ± SD**
Age	103(45-89)	65.60±9.17
BMI	103 (15,60-45,00)	26.70±5.95
Famale	16(15.53)	40,74±13,89
Male	87 (84.46)	40,70±12,29
Duration of COPD (years)	103(4-20)	8.36±4.40
The number of comorbidities	76(73.78) (0-4)	
Smoking use Smoking pk/year Continue smoking Quit smoking Nonsmoker	 (4-40) 49(47.57) 41(39.80) 13(12.62)	 19.96±10.63
SaO_2	(68-97)	90.56±5.97
Number of patients who are on LTOT **Number of patients who are none LTOT**	36(34.95) 67(65.04)	
GOLD1 **GOLD2** **GOLD3** **GOLD4**	3(2.91) 42(40.77) 43(41.74) 15(14.56)	
İlaçlar LAMA+LABA LAMA+LABA+ICS İCS+LABA LABA LAMA Mucolytic Theophylline Steroid (Oral + IM/IV) Non-medication	, 4(2.55) 76(74.50) 12(11.74) 1 (0.98) 2 (1.96) 44 (43.139 84 (82.35) 59 (57.849 33 (32.35) 180.98)	
Additional disease There is illness No disease Hypertension Diabetes mellitus Heart Disease Other	 58 (55.12) 45(44.87) 59 (57.28) 13 (12.62) 22 (21.35) 6 (5.82)	

BMI: Body Mass Index; GOLD: Global Initiative for Chronic Obstructive Lung Disease, LTOT: Long-Term Oxygen Therapy;
FEV1: Forced Expiratory Volume in 1 Second; FVC: Forced Vital Capacity;
FEV1/FVC: The Ratio of Forced Expiratory Volume in 1 Second (FEV1) to Forced Vital Capacity (FVC);
CRP: C-Reactive Protein; LAMA: Long-Acting Muscarinic; LABA: Long-Acting B2 Agonist İKS. Inhaled Corticosteroids

Table 2 Comparison of Parameters of COPD Patients GOLD

Parameters of the cases	GOLD1,2 (n=46) Mean ± Sd	GOLD3,4 (n=57) Mean ± Sd	P
Age (year)	63.00±8.74	67.40±8.99	0.504
Smoking(pk/years) y	15.06±9.94	23.75±9.69	0.498
BMI	29.20±5.24	24.75±5.84	0.714
FVC %	83.64±15.34	57.33±14.07	0.863
FEV_1/FVC %	80.13±9.62	67.49±9.64	0.110
Urea mg/dl	35.27±9.24	37.30±9.53	0.918
Creatine mg/dl	0.98±0.29	1.20±1.36	0.188
Uric acid mg/dl	5.166±1.08	5.55±1.05	0.593
Protein g/L	66.41±4.43	66.49±4.47	0.947
Albumin g/L	41.55±2.92	40.44±4.51	0.518
CRP mg/L	17.73±27.30	45.55±43.42	<0.001
WBC $X10^3$	10.09±2.91	9.76±3.81	0.829
Neutrophil %	64.63±9.07	63.85±14.68	0.104
Neutrophil $X10^3$	6.71±4.75	5.97±3.70	0.294
lymphocyte %	23.21±9.31	22.68±10.49	0.462
lymphocyte $X10^3$	2.79±4.51	2.02±0.62	0.051
Eosinophil %	2.15±2.23	2.35±2.82	0.790
Eosinophil l $X10^3$	0.17±0.16	0.25±0.25	0.052
Uric acid /albumin	0.027±0.00	0.028±0.00	0.274
Number of attacks in 1 year y	24,73±51.32	38.45±23.37	0.042

x: Comparison was performed with t-test; y: Comparison was done with Mann-Whitney U test
WBC: White Blood Count; CRP: C Reaktif Protein; BMI: Body Mass Index

GOLD stages 1 and 2 were combined into a subgroup with less severe airflow limitation and a forced expiratory volume in 1 second (FEV1) > 50% predicted. The combined GOLD 3 and GOLD 4 subgroup had more severe airflow limitations and an FEV1 <50% predicted. Comparisons were made between COPD grades. The relationship between GOLD1 and 2 versus GOLD3 and 4, respiratory function, age, serum markers, blood gas oxygen and saturation, and respiratory function FEV1 tests was investigated when comparing severe and very severe COPD (GOLD 3,4) group, low oxygen saturation and FEV1 values, high CRP levels, and a high number of exacerbations per year were found to be statistically significant (p<0.001, p=0.007, p=0.011, p<0.001, p=0.042, respectively) (Table 2).

COPD patients were compared with healthy subjects. Serum markers showed elevated levels of urea, creatinine, uric acid/albumin, CRP, WBC, and eosinophil percentage in the COPD group (p=0.002, p=0,010, p=0.002, p<0.001, p=0.018, p=0.037, respectively) (Table 3).

During acute exacerbations of COPD, uric acid/albumin, CRP, and leukocyte levels were higher in the control group compared to the stable group (p=0.001, p=0.048, p=0.042) (Table 4).

Discussion

They have suggested that serum uric acid levels may be a biomarker for COPD (8). Chronic obstructive pul-

Table 3 Comparison of Parameters of COPD Patients with Healthy Individuals

Parameters of the cases	COPD (n=103) Mean ± Sd	Healthy (n=38) Mean ± Sd	p
Age	65.60±9.17	58.13±11.34	0.018
Cigarette year pk/year [y]	19.96±10.63	11.60±8.49	0.602
BMI	26.70±5.95	27.04±3.34	0.010
SaO2 %	90.56±5.97	97.48±0.96	<0.001
FEV1 %	49.90±18.22	82.85±10.95	<0.001
FVC%	68.47±20.08	89.50±8.82	<0.001
FEV1/FVC%	73.13±11.44	94.34±9.11	<0.001
Ure mg/dl	36.45±9.37	24.77±7.01	0.002
Creatine mg/dl	1.10±1.03	0.71±0.14	0.010
Uric acid mg/dl	1.13±0.02	1.07±0.02	0.157
Protein g/L	66.40±4.45	68.01±5.23	0.718
Albumin g/L	40.69±4.57	45.68±3.44	0.907
CRP [x] mg/L	34.01±40.04	12.03±15.91	<0.001
WBC [x] $X10^3$	9.95±3.44	9.29±1.67	0.018
Neutrophil [x] %	64.25±12.42	63.57±11.94	0.395
Neutrophil $X10^3$	6.33±4.18	5.93±1.39	0.056
lymphocyte %	22.83±9.93	25.59±9.54	0.877
lymphocyte $X10^3$	2.36±3.02	2.34±0.81	0.624
Eosinophil [x] %	2.24±2.56	1.98±1.41	0.037
Eosinophil $X10^3$	0.21±0.22	0.170±0.17	0.099
Uric acid /albumin	0.13±0.01	0.02±0.14	0.002
PaO_2 mmHg	77.58±16.74	94.85±27.84	0.048

x: Comparison was performed with t-test; y: Comparison was done with Mann-Whitney U test
WBC: White Blood Count; CRP: C-reactive protein

monary disease (COPD) is a disease in which oxidative stress and inflammation play a role in its development. Uric acid can exhibit antioxidant, prooxidative, or proinflammatory effects depending on the specific situation (9).

It has been suggested that various factors such as air pollution and smoking have a greater effect on lung function in COPD patients, and therefore they have received significant attention. Quitting smoking and avoiding air pollution are important recommendations for preventing the decline in lung function in COPD patients, and blood biomarkers such as serum uric acid levels were preferred in studies. In this study, we aimed to investigate the role of serum uric acid, albumin, and uric acid/albumin ratio in COPD severity during the acute exacerbation period of COPD patients, as well as their potential use as biomarkers.

Studies showing elevated serum bilirubin, blood uric acid, and CRP levels in patients with acute exacerbations of COPD suggest that these markers may be used to predict acute exacerbations (10). Elevated uric acid is associated with a higher risk of acute exacerbation of COPD (11, 12). An increase in uric acid levels was observed with COPD severity, but the difference was not statistically significant (12). In a different study, it was suggested that the increase

Table 4 Comparison of Acute and Stable Period Parameters in COPD

Parameters of the cases	Acute exacerbation COPD(n=103) Mean ± Sd	Stable COPD (n=103) Mean ± Sd	p
SaO_2 %	90.56±5.97	91.48±5.07	<0.001
FEV_1 %	49.90±18.22	54.85±9.14	0.694
FVC %	68.47±20.08	72.36±5.78	0.568
FEV_1/FVC %	73.13±11.44	71.94±1.76	0.756
Ure mg/dl	36.45±9.37	34.28±5.68	0,326
Creatine mg/dl	1.10±1.03	1.29±1.15	0,092
Uric acıd mg/dl	5.35±1.11	4.95±1.02	0.286
Total protein g/L	66.40±4.45	69.18±3.63	0.627
Albumin g/L	40.68±4.59	44.35±35.94	0.274
Uric acid /albumin	0.12±0.03	0.02±0.00	0.001
CRP mg/L	34.01±40.04	14.28±16.03	0.048
WBC $X10^3$	9.95±3.44	9.34±7.95	0.042
PaO_2 mmHg	44.63±10.76	68.46±45.28	0.047

WBC: White Blood Count; CRP: C-reactive protein

in serum uric acid levels was significantly associated with accelerated declines in FEV1, FVC, and FEV1/FVC, and therefore, serum uric acid could be used to predict the decline in lung function (13). Barmehziar and colleagues (5) found that uric acid and uric acid/creatinine levels were higher in patients with COPD exacerbations, serving as a marker of oxidative metabolism in the hypoxemic state that develops in patients with severe COPD and frequent exacerbations. Some authors (14, 15) have found that serum uric acid levels and uric acid/creatinine ratios in patients during exacerbation periods are higher and statistically significant compared to the stable COPD group, and that serum uric acid levels and uric acid/creatinine ratios in patients developing hypoxemia are higher than in patients without hypoxemia. Yamaya and colleagues (7) reported that serum total protein and albumin levels, as well as lymphocyte counts, were lower in patients in the flare-up group compared to those in the non-flare-up group, while serum CRP, WBC, and neutrophil counts were higher in the flare-up group than in the non-flare-up group.

In this study, the comparison of the exacerbation period of COPD with the stable period revealed that elevated uric acid/albumin, CRP, and WBC levels, along with low SaO2 and PaO2 values, and the statistically significant difference between them supported the association with respiratory distress alongside decreased lung function in patients.

Yamaya and colleagues (7) found the average BMI value to be 22 kg/m² in their study on COPD. Lim and colleagues (12) found the BMI value in COPD patients to be 27 kg/m². Another study also showed the association between low BMI and mortality and/or exacerbation in COPD patients (16). In our study, the mean BMI was 26.70±5,95 kg/m2, which was similar to that reported in other studies. However, no association was observed between BMI and the severity of COPD. Soman and colleagues (6) demonstrated in their study that serum uric acid levels were elevated in patients with advanced COPD and severe airway obstruction. Furthermore, patients with high serum uric acid levels had a worse prognosis compared to those with low levels in COPD. They demonstrated a strong correlation between serum uric acid levels and COPD severity as defined by GOLD criteria, and found significantly higher serum uric acid levels in patients classified as GOLD Stage III and IV compared to those in Stage I

and II (6, 17). Hypoxemia associated with increasing severity/stage of the disease leads to excessive uric acid accumulation as a result of tissue destruction. Higher uric acid levels have been detected in advanced GOLD stages (stages 3 and 4) of COPD compared to stages 1 and 2. Studies have demonstrated a significant correlation between hypoxemia and serum uric acid levels (12, 18). In this study, when GOLD stages 3 and 4 were compared with GOLD stages 1 and 2, PaO2, SaO2, and FEV1 were found to be lower, and the number of attacks observed in 1 year was higher. The results were found to be statistically significant. Although uric acid was found to be high in GOLD stages 3 and 4, it was not found to be statistically significant.

Some studies (17, 19) have found that serum uric acid levels are significantly elevated in COPD patients compared to healthy controls. Uric acid levels are a useful biomarker for COPD (8).

In this study, when comparing COPD patients with healthy individuals, serum urea, CRP, and uric acid/albumin levels were found to be higher and statistically significant in COPD patients. A negative correlation was observed between CRP, urea, and uric acid/albumin levels and COPD cases.

A study (20) showed that patients with airflow limitation had significantly higher serum uric acid levels compared to non-smokers without airflow limitation. These findings highlight the potential role of uric acid as a biomarker for COPD and have demonstrated a positive correlation between serum uric acid levels and the severity of COPD. In the study, when smoking was compared according to COPD severity as pack-years, smoking was found to be higher in GOLD stages 3 and 4 than in GOLD stages 1 and 2. A positive correlation was found between COPD severity and smoking, and it was determined that as smoking increased, airway obstruction also increased.

Conclusion

In conclusion, serum uric acid/albumin levels were higher during COPD exacerbations compared to the stable period and were associated with COPD exacerbations. When compared to COPD stages, urea, uric acid, and uric acid/albumin values were higher in GOLD 3 and 4 (severe and very severe) stages compared to GOLD 1 and 2 (mild-moderate) stages, although not statistically significantly. Furthermore, urea, creatinine, and uric acid/albumin levels were higher in COPD patients compared to healthy patients. These results suggest a relationship between uric acid and uric acid/albumin levels and COPD exacerbations in COPD patients, and that this relationship may play a complementary role in determining the severity of COPD and predicting future exacerbations.

The limitations of the study are that it is a single-center study, and the number of patients is low. Future studies with larger data sets are needed.

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Conflict of Interest Statement
The author has no conflicts of interest to declare.

Ethical Approval
This study was Ethics Committee approval was obtained from Harran University with numbered HRÜ/25.10.32 and dated 2025. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Consent to Participate and Publish
Written informed consent to participate and publish was obtained from all individual participants or legal guardians included in the study.

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Availability of Data and Materials
Data are available on request due to privacy or other restrictions.

Artificial Intelligence Statement
The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

Authors Contributions
İ.M.H: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Validation; Visualization; Writing-original draft.

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