International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

IJMRP

Original Research Article.

Serum Uric Acid Levels in Chronic Obstructive Lung Disease Patients

Sapna Jaiswal[1], Tabassum Yasmin[2*]

[1]Tutor, Department of Biochemistry,
Ram Manohar Lohia Institute of Medical Sciences, Gomti Nagar, Lucknow, Uttar Pradesh, India.
[2*]Associate Professor, Department of Biochemistry,
Heritage Institute of Medical Sciences, Varanasi, Uttar Pradesh, India.

ABSTRACT

Objective: This study was done to measure serum uric acid levels in chronic obstructive pulmonary disease patients.

Materials & Methods: 110 stable COPD patients in the age group 35-70 years were included in this study. Spirometric parameters like Forced vital capacity (FVC) and Forced Expiratory Volume (FEV) were measured using 1 standard technique with Spirometer.

Result: 110 stable patients with COPD, without comorbid conditions, were included as cases. 37 age and sex matched individuals were included as controls. Clinical, functional characteristics and serum UA levels were compared between cases and controls. Student's unpaired t test was used to compare serum uric acid levels in cases and controls. The serum uric acid levels were significantly higher in patients with COPD than in controls.

Conclusion: Prolonged exposure to cigarette smoke injures the respiratory system causing pulmonary diseases such as chronic obstructive pulmonary disease (COPD). Oxidants damage the lung tissue decrease the pulmonary function and cause tissue hypoxia. Serum uric acid, the purine degradation product is elevated in various clinical conditions associated with hypoxia.

Keywords: Cigarette Smoke, COPD, Uric Acid, Hypoxia, Spirometry.

***Correspondence to:**

Tabassum Yasmin,
Associate Professor,
Department of Biochemistry,
Heritage Institute of Medical Sciences,
Varanasi, Uttar Pradesh, India.

Article History:

Received: 23-09-2017, **Revised:** 17-10-2017, **Accepted:** 28-11-2017

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2017.3.6.067	

INTRODUCTION

Chronic cigarette smoking damages the respiratory system and causes pulmonary diseases including chronic obstructive pulmonary disease (COPD). Even passive cigarette smoke exposure and indoor air pollution resulting from biomass cooking or heating induces oxidative stress and lung inflammation in otherwise healthy individuals especially in developing countries. As a result of damage to lung tissues induced by oxidants and inflammation, pulmonary function declines.[1,2] Impairment of pulmonary function reduces oxygen intake, resulting in tissue hypoxia. Serum uric acid (UA) has been shown to be increased in hypoxic state.[3] Previous studies have shown that UA has antioxidant properties.[4] In addition proinflammatory effect of UA is more profound in those with high serum UA levels.[5] Hyperuricemia is defined as serum UA levels >7.1mg/dL (420 µmol/L) in men or >6.1mg/dL (360 µmol/L) in women.[6] Compared with individuals with normouricemia, individuals with hyperuricemia have more inflammation and oxidative stress injuries. Also studies have shown that hyperuricemia is strongly associated with increased cardiovascular mortality.[7,8] In this study we assessed whether the presence of higher values of serum UA is associated with changes in clinical and functional characteristics in patients with chronic obstructive pulmonary disease (COPD).

MATERIALS AND METHODS

The study was conducted in over a period of one year from June 2016 to June 2017 in GCRC medical college, Lucknow, India. 110 stable COPD patients in the age group 35-70 years were included in this study. Spirometric parameters like Forced vital capacity (FVC) and Forced Expiratory Volume (FEV) were measured using 1 standard technique with Spirometer.

Spirometry was done without the administration of any bronchodilator. The highest value out of three FVC measured by each subject was used in the analysis. Percent predicted values for spirometric parameters are presented as FEV % predicted and FVC 1 % predicted. Subjects with FEV /FVC <70% were identified as having 1 airflow limitation (COPD).

According to the Global initiative for Chronic Obstructive Lung Disease (GOLD) criteria, subjects with airflow limitation and FEV1 % predicted ≥80 were identified as having mild air flow limitation, those with FEV % predicted between 50 and 1 <80 were defined as having moderate air flow limitation, and those with FEV % predicted <50 were defined as having severe airflow 1.2 limitation Of the total 110 cases, 52 were mild, 42 were moderate and 16 were severe COPD patients.37 age and sex matched subjects were included as controls (mean age 48.7yrs). Clinical

assessment included detailed physical examination, information regarding smoking
history, biomass exposure and accompanying diseases were elicited from both cases and control groups. Exclusion criteria for the present study includes patients with history of pulmonary tuberculosis, asthma, coronary artery disease, renal disease, liver disease, diabetes mellitus, cancer and patients on chemotherapy and radiotherapy. Venous blood samples were drawn from controls and COPD patients. Renal function tests, liver function tests, uric acid and electrolytes were measured using Cobas c501 fully automated clinical chemistry analyser. Serum uric acid was analysed using uricase peroxidase methodology in autoanalyser.

Statistical Analysis

Clinical characteristics of the study population were compared using Chi square test and Student's t-test. p - value of < 0.05 were considered statistically significant. Comparison of serum uric acid levels between patients with COPD and controls were done using Student's unpaired t-test. Comparison of serum uric acid levels between various stages of patients with COPD was performed using ANOVA.

RESULTS

Clinical characteristics of COPD patients and controls are summarized in Table 1. FEV1% predicted was found to be highly significant among cases and controls. Smoking history, biomass exposure and FEV1/FVC ratio are found to be statistically significant. Serum uric acid levels in controls and COPD patients shown in Table 2.

Table 1: Clinical characteristics of the study population

	Controls (n=74)	COPD patients (n= 110)	p-value
Age (years)	50.0+/- 8.7	51.9+/- 10.3	0.36
Males (n%)	48(64.9%)	74(67.3%)	0.91
Smoking history	22(29.7%)	74(67.3%)	0.04*
Biomass exposure	34(45.9%)	76(69.1%)	0.03*
FEV1%predicted	87.6+4.8	71+ 15.7	0.002**
FEV1/FVC	80.7+ 2.0	63.8+ 4.2	0.01*
**** Highly significant *Significant**			

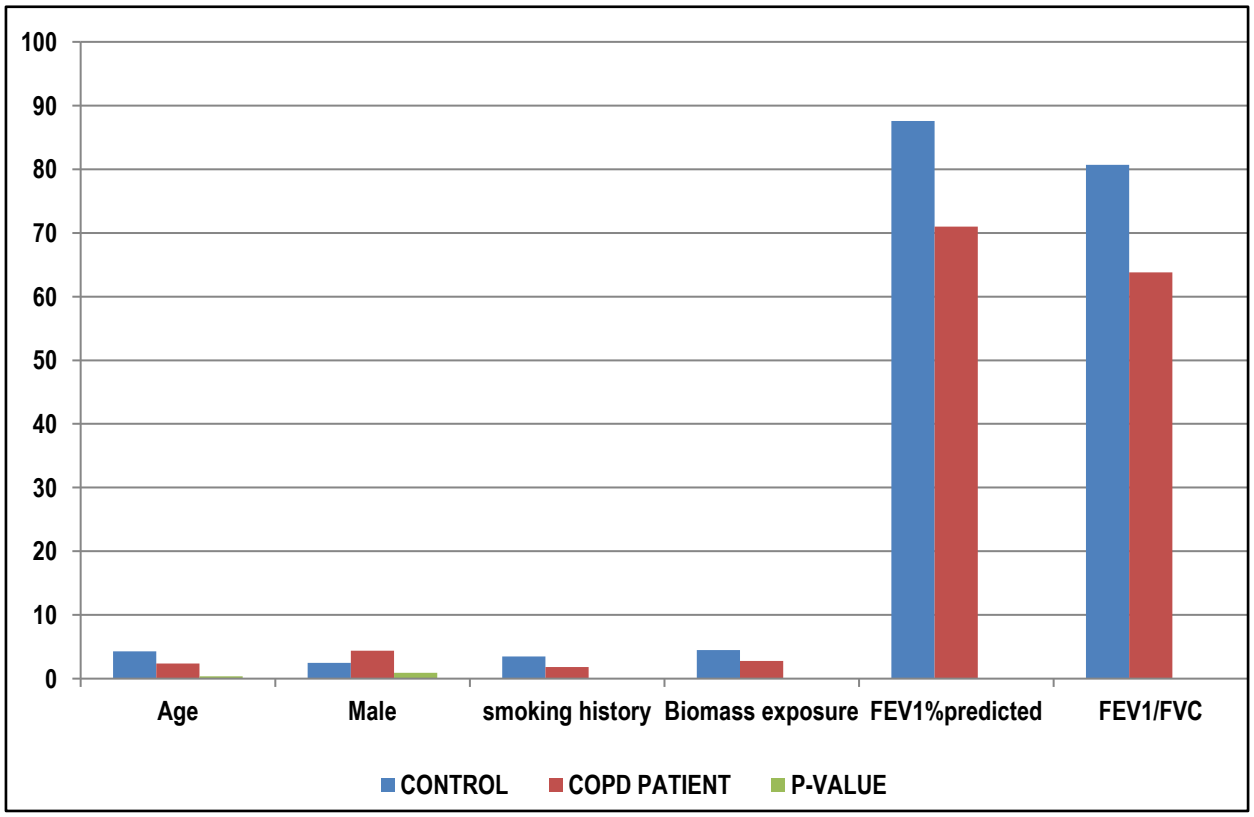

Fig 1: Clinical characteristics of the study population

Table 2: Serum uric acid levels in controls and COPD patients

Group	N	Mean(mg/dL)	Std. Deviation	SEM	p- value
Control	74	4.60	1.06	0.18	0.02*
COPD	110	5.20	1.26	0.17	

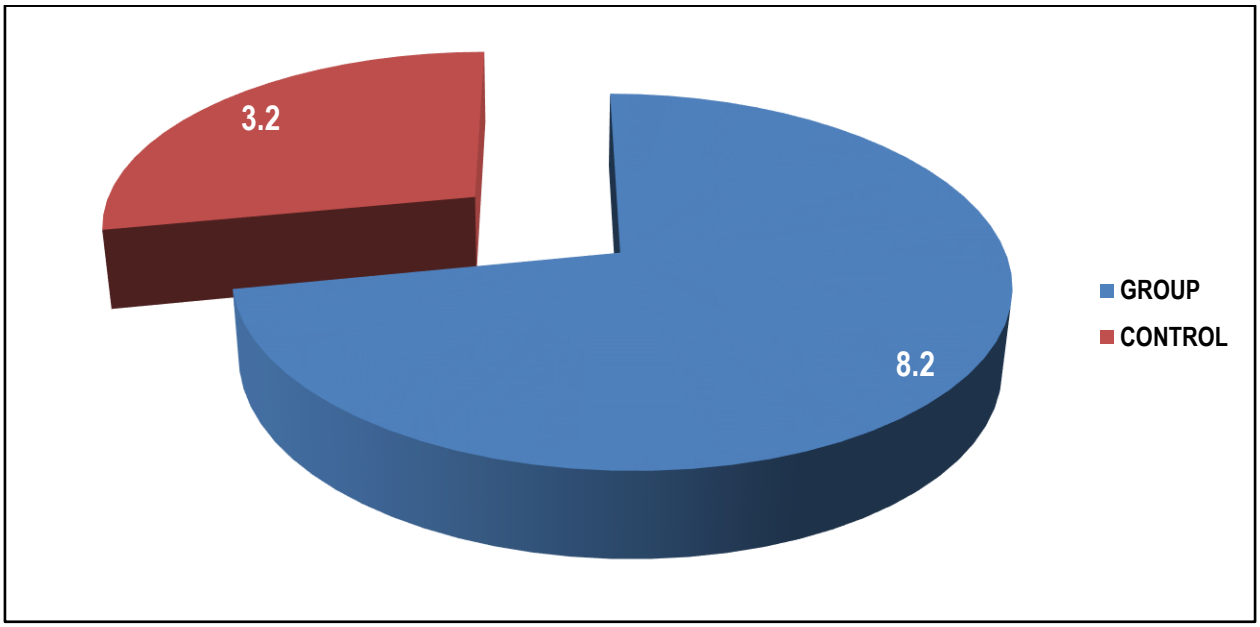

Fig 2: Serum uric acid levels in controls and COPD patients

Table 3: Comparison of serum uric acid levels between various stages of patients with COPD

COPD group total cases- 55	N	Mean uric acid (mg/dL)	Std. Deviation	p –value
Mild	52	4.41	1.04	
Moderate	42	5.73	0.96	<0.001**
Severe	16	6.35	1.05	

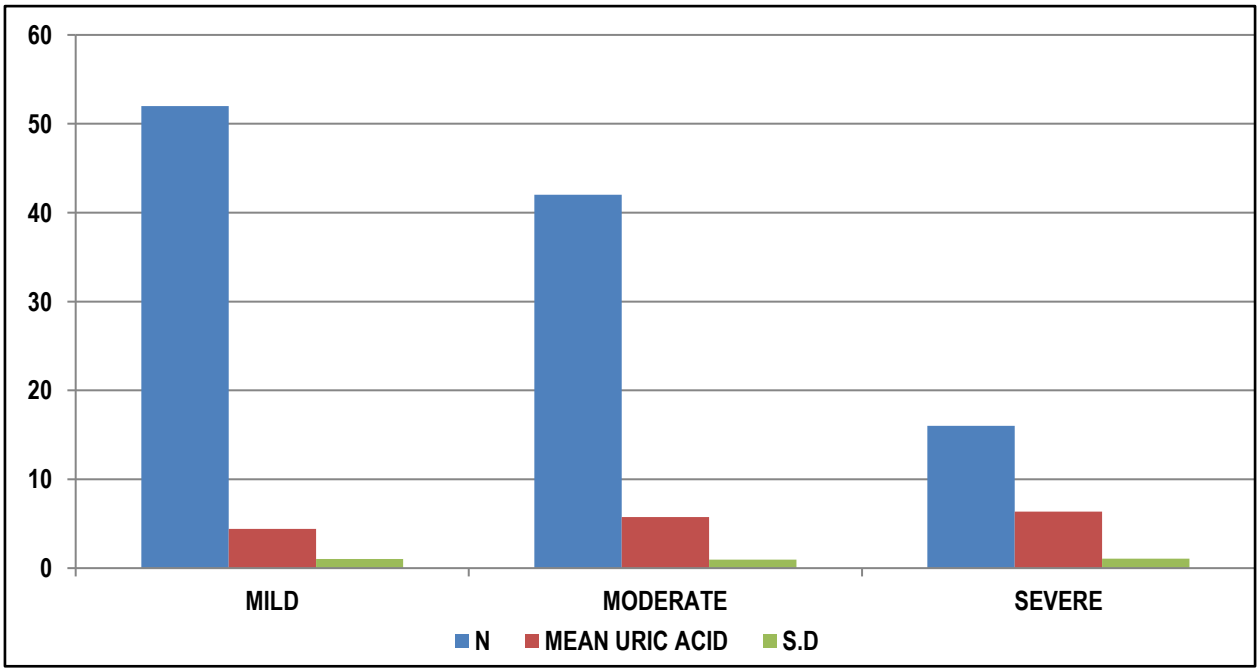

Fig 3: Comparison of serum uric acid levels between various stages of patients with COPD

DISCUSSION

UA is the end-product of purine degradation. Excessive intake of foods containing purine bases, alcohol consumption , renal dysfunction and genetic disorders of purine metabolism, such as hypoxanthine-guanine phosphoribosyl transferase deficiency (Lesch-Nyhan syndrome) and adenine phosphoribosyl transferase deficiency, result in elevation of serum UA levels.[6-9] In addition, other demographic and clinical factors, such as gender, BMI, smoking index, and serum glucose levels, are known to be associated with increased serum levels of UA.[10] Therefore, careful consideration of these factors is required when assessing the relationship between pulmonary function and UA levels. Tissue hypoxia has been reported to induce the degradation of adenosine.[11] This results in the release of purine intermediates

and end products of purine catabolism, such as uric acid (UA).[12] Elevation of serum UA (sUA) levels has been observed in hypoxic subjects, including patients with COPD.[13] UA is a biomarker of xanthine oxidase activity, which is known to be an important source of reactive oxygen species.[14] Several investigators have reported that elevated UA levels were associated with worsening of cardiovascular disease, heart failure and COPD.[15] In addition, positive associations were demonstrated between UA and inflammatory markers such as C-reactive protein and 6 interleukin- . These findings suggest that systemic UA levels are associated with oxidative stress and inflammation in vivo. UA activates leukocytes through the NALP3 inflammasome.[16] Activated leukocytes express selectins and adhere to endothelial cells, where they secrete various pro-inflammatory cytokines and chemical mediators, resulting in vessel wall damage and atherosclerosis. Possible explanations for the association between elevated sUA levels and pulmonary function includes 1) hypoxia due to impaired pulmonary function leading to purine catabolism, 2) impaired pulmonary function inducing pulmonary hypertension and resulting in the elevation of sUA levels, 3) Toxins in cigarette smoke causes oxidative stress in the alveolar spaces of the lungs. This oxidative stress induces lung inflammation contributing to the pathogenesis of chronic respiratory diseases, such as COPD and pulmonary fibrosis.[17] sUA levels may be elevated according to the severity of tissue damage or conversely, UA activates leukocytes through the NALP3 inflammasome. Subsequently, activated leukocytes cause damage to vascular endothelial cells, pulmonary endothelial dysfunction is involved in the pathogenesis of COPD. Hyperuricemia-induced endothelial dysfunction is associated with impaired pulmonary function in the general population.[18,19]

CONCLUSION

To conclude elevated serum uric acid level may serve as a noninvasive indicator for COPD severity and hypoxemia in stable COPD patients. Hence there is a need to evaluate serum UA levels as an additional parameter for predicting outcome in COPD patients.

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Source of Support: Nil.

Conflict of Interest: None Declared.

Cite this article as: Sapna Jaiswal, Tabassum Yasmin. Serum Uric Acid Levels in Chronic Obstructive Lung Disease Patients. Int J Med Res Prof. 2017 Nov; 3(6):330-33.
DOI:10.21276/ijmrp.2017.3.6.067