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# Serum Uric Acid as a Biomarker of Disease Severity and Treatment Response in COPD Patients: A Hospital-Based Study in Tripura

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## ABSTRACT

**Background:** Chronic obstructive pulmonary disease (COPD) is characterised by persistent inflammation and oxidative stress. Serum uric acid (SUA) has been proposed as a potential biomarker reflecting disease activity.
**Aim:** To evaluate SUA levels among admitted COPD patients and assess their role in monitoring disease severity and treatment response.
**Methods:** A hospital-based observational study was conducted among COPD patients admitted to a tertiary care hospital in Tripura. SUA levels were measured at admission and discharge. Demographic, socioeconomic, occupational, and smoking-related factors were analysed for associations.
**Results:** Mean SUA levels were significantly elevated at admission (6.43 ± 1.21 mg/dL) compared to discharge (4.70 ± 0.75 mg/dL; $p < 0.001$). Younger patients aged ≤50 years (6.81 ± 1.23 mg/dL) and those from higher socioeconomic groups (7.03 ± 1.50 mg/dL) exhibited higher SUA values. Occupational exposure, particularly among housewives (6.89 ± 1.28 mg/dL), was associated with elevated levels. Interestingly, nonsmokers had slightly higher SUA than smokers (6.65 ± 1.28 vs 6.43 ± 1.21 mg/dL), though not statistically significant (p=0.64).
**Conclusion:** SUA levels decline significantly with clinical improvement, supporting its utility as a dynamic biomarker in COPD management. Its inexpensive measurement makes it particularly valuable in resource-limited settings. Contextual factors such as socioeconomic status and occupational exposure should be considered when interpreting SUA levels.
***Keywords:*** Chronic obstructive pulmonary disease, serum uric acid, biomarker, oxidative stress, socioeconomic determinants, Tripura.

## INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterised by persistent airflow limitation, chronic inflammation, and structural changes in the lungs. It encompasses chronic bronchitis and emphysema, both of which impair gas exchange and reduce lung

function.[1] COPD is a major global health challenge, affecting about 213 million people and causing 3.5 million deaths in 2021—nearly 5% of all worldwide mortality and posing a significant socioeconomic burden, particularly in low- and middle-income countries like India. In India, COPD prevalence is high due to widespread smoking, biomass fuel exposure, and occupational hazards, making it a pressing public health issue.[2]

### Pathophysiology and Role of Oxidative Stress

The pathogenesis of COPD involves chronic airway inflammation, oxidative stress, and tissue remodelling. Inhalation of noxious particles, including cigarette smoke or biomass-burning smoke, triggers inflammatory cascades mediated by cytokines including TNF-α, IL-6, and IL-8, leading to progressive lung damage.[3] Oxidative stress, resulting from an imbalance between free radicals and antioxidants, plays a central role in disease progression. Identifying biomarkers that reflect these processes is crucial for early diagnosis and monitoring disease severity.[4]

### Uric Acid as a Biomarker

Uric acid is the final product of purine metabolism and acts as a major antioxidant in plasma under physiological conditions. Hyperuricemia, defined as elevated serum uric acid (SUA), is associated with a wide range of conditions, including gout, cardiovascular disease, renal disorders, metabolic syndrome, and diabetes. It significantly reduces quality of life, and its prevalence continues to rise globally, particularly in developed nations.[5] In COPD, hypoxia and oxidative stress increase purine catabolism, leading to higher uric acid levels. This dual role—antioxidant defence versus pro-inflammatory mediator—makes uric acid a promising but complex biomarker.[6,7]

### Evidence Linking Uric Acid and COPD

Earlier studies have demonstrated that SUA levels are significantly higher in COPD patients in comparison to healthy controls, and levels correlate with disease severity and duration.[8] For instance, patients with advanced COPD (GOLD stage III/IV) exhibit higher uric acid levels than those with milder disease.[9] Hyperuricemia has also been associated with increased risk of exacerbations, systemic inflammation, and comorbid cardiovascular disease.[10]

A hospital-based case-control study in India by Sarangi et al. (2017) reported that COPD patients had mean uric acid levels nearly double those of controls, highlighting its potential utility in risk stratification and prognosis.[11] Similarly, meta-analyses by Li and Chen (2021) suggest uric acid could serve as a cost-effective, non-invasive biomarker for assessing disease severity and predicting outcomes.[12]

### Relevance to Tertiary Care Settings

In tertiary care hospitals, particularly in resource-limited regions such as Tripura, simple and inexpensive biomarkers are valuable for patient assessment. Measuring SUA offers a low-cost, accessible tool that can complement spirometry and imaging in the evaluation of COPD severity. It may also help identify patients at higher risk of poor outcomes, guiding timely interventions and management strategies. Therefore, the study aimed to evaluate SUA levels among admitted COPD patients and assess their role in monitoring disease severity and treatment response.

## MATERIAL AND METHODS

### Study Design and Setting

This investigation was conducted as a descriptive, cross-sectional study over a period of two years. Data collection and procedural work were completed within 1.5 years. The study was conducted in the Department of Biochemistry at Agartala Government Medical College (AGMC) & GBP Hospital, in collaboration with the Department of Respiratory Medicine at the same institution.

**Study Population**

The study population comprised admitted patients with COPD in the Department of Respiratory Medicine at AGMC & GBP Hospital. All participants were recruited consecutively during the study period, in accordance with the inclusion and exclusion criteria defined in the protocol.

**Sample Size**

An initial calculation yielded a sample size of 62 patients. To improve precision and enhance the robustness of the statistical analysis, the sample size was rounded up to 150 participants. This adjustment ensured adequate representation and strengthened the reliability of findings.

**Sampling Technique**

To achieve an estimated sample size of 150, study subjects were selected using census sampling from admitted COPD patients in the inpatient department (IPD) of the Respiratory Medicine unit at AGMC & GBP Hospital. All patients meeting the predefined inclusion and exclusion criteria during the study period were considered eligible for recruitment.

**Inclusion Criteria**

Patients were included if they met the following conditions:

- Diagnosed cases of COPD admitted to the Department of Respiratory Medicine.
- No acute exacerbation within the preceding three months.
- Absence of active lower respiratory tract infection at the time of recruitment.
- Willingness to provide informed consent and participate in the study.

**Exclusion Criteria**

Patients were excluded if they had any of the following:

- Coexisting pulmonary tuberculosis.
- History of chronic hypertension, cardiac disease, renal disease, diabetes mellitus, hypothyroidism, hyperuricemia, gout, malignancy, major systemic illness, or autoimmune disease.
- Pregnancy.
- Refusal to participate in the study.
- Current use of medications known to lower SUA levels.

**Procedure**

All COPD patients admitted to the Department of Respiratory Medicine who fulfilled the inclusion criteria were enrolled as study subjects. Post-treatment COPD patients at the time of discharge, meeting the same criteria, were considered as the comparison group (controls). Each participant was informed in detail about the purpose of the study and the procedures involved. Written informed consent was obtained prior to inclusion. A thorough history was recorded, followed by general physical examination, and all findings were documented in a structured case record form. For the study, venous blood samples were collected under aseptic precautions to estimate SUA levels. The analysis was performed on the same day to ensure the accuracy and reliability of the results.

**Estimation of SUA**

SUA concentration was determined using the Uricase–UV method.

**Sample and Reagents:**

- Serum: 5 µL
- Reagent 1: 200 µL
- Reagent 2: 50 µL

The assay was performed using the XL-640 Fully Automated Clinical Chemistry Analyser. Optical density (OD) was recorded at 505 nm, and uric acid concentration was calculated automatically by the analyser software based on calibration standards.

**Collection of Blood Samples**

Under strict aseptic precautions, 5 mL of venous blood was collected, preferably from the antecubital vein, using a sterile needle and syringe. The samples were transferred into clot activator tubes. Clotting was allowed to occur at room temperature, after which serum was separated by centrifugation at 3000 rpm for 3–5 minutes. The biochemical analysis was performed on the

same day to ensure sample integrity and accuracy.

**Data Management and Statistical Analysis**

Data entry and statistical analysis were carried out using SPSS software version 25 on a Windows-based system. Categorical variables were summarised using descriptive statistics and presented as text, tables, and charts. Statistical analysis was conducted using one-way ANOVA and paired t-test procedures. A p-value ≤ 0.05 was considered statistically significant.

**Ethical Considerations**

The study was initiated only after obtaining approval from the Institutional Ethics Committee of AGMC. Written informed consent was obtained from all participants prior to inclusion. Data collected during the study were kept strictly confidential and utilised solely for research purposes, in accordance with ethical standards for biomedical research.

## RESULTS

The majority of study participants were aged ≤ 50 years (27%), followed closely by those aged 61–70 years (23%) and 51–60 years (23%). A smaller proportion belonged to the 71–80-year group (17%), while only 10% were aged 81 years and older (Figure 1).

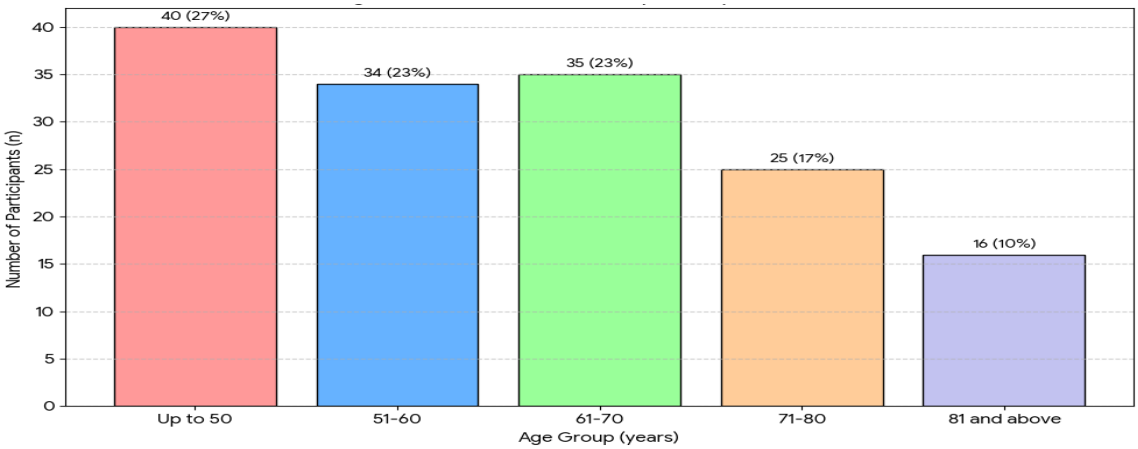


**Figure 1: Age-wise Distribution of Study Participants (n=150)**

Among the study participants, 82 (55%) were female, while 68 (45%) were male, indicating a slightly higher representation of women in the study population (Figure 2).

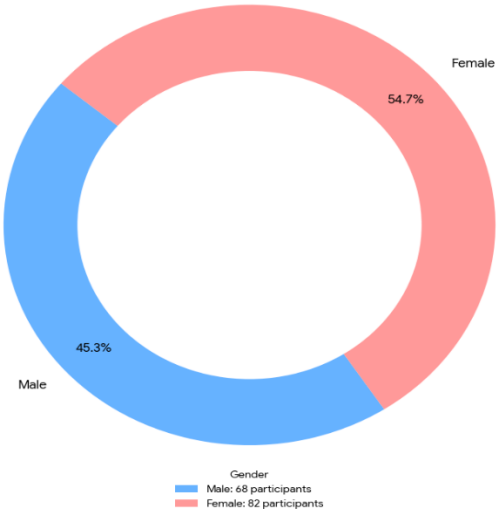


**Figure 2: Gender-wise Distribution of Study Participants (n=150)**

The occupation-wise distribution of COPD patients shows that the largest proportion is in the "Others" category (44%), likely including individuals engaged in informal or unclassified work. This was followed by businessmen (20%) and housewives (17%), while agricultural workers (12%) and government employees (7%) formed smaller groups (Figure 3).

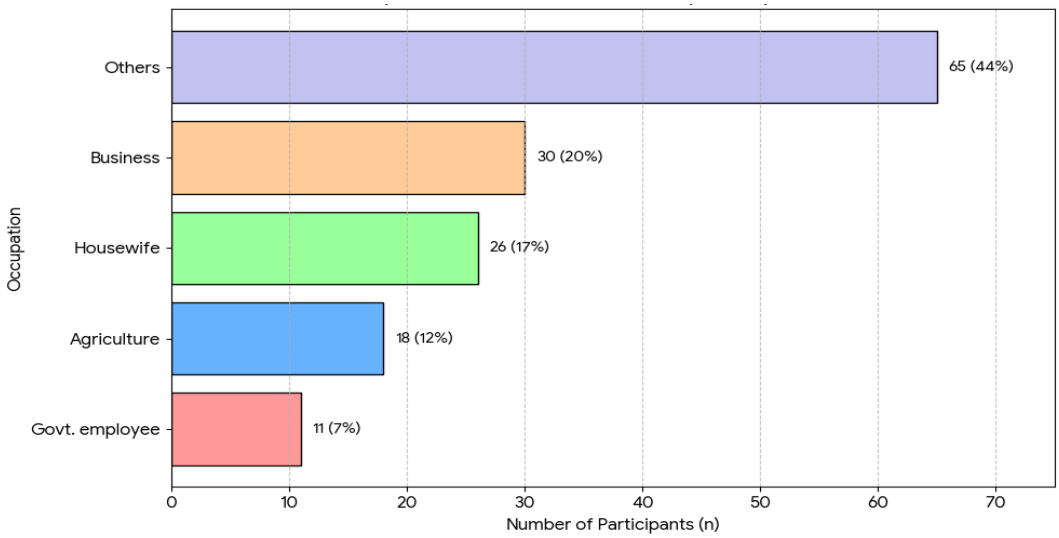


**Figure 3: Occupation-wise Distribution of Study Participants (n=150)**

The socio-economic status shows that the largest proportions of participants belonged to the upper-middle class (27.33%) and lower-middle class (27.33%), together accounting for more than half of the study population. The upper class represented 22.67%, while both the upper-lower and lower classes comprised 11.33% each (Figure 4).

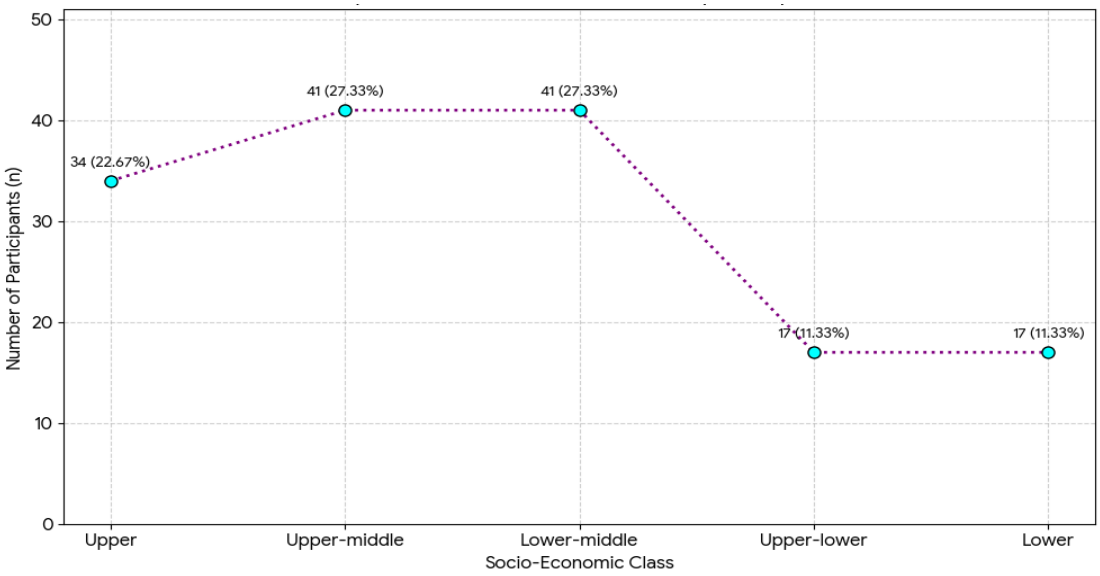


**Figure 4: Socio-economic Status of Study Participants (n=150)**

The distribution according to tobacco use shows that a clear majority, 99 participants (66%), reported tobacco use, while 51 participants (34%) did not. The distribution according to smoking habits shows three distinct groups. The largest proportion was current smokers (60 participants, 40%), followed by ex-smokers (39 participants,

26%), while non-smokers accounted for 51 participants (34%). The distribution of COVID-19 infection history shows that a large majority, 118 participants (79%), reported a prior history of COVID-19, while only 32 participants (21%) had no such history. The distribution according to family history of lung disease shows that 62 participants (41%) reported a positive family history of conditions such as asthma, COPD, or tuberculosis, while 88 participants (59%) had no such family history (Figure 5).

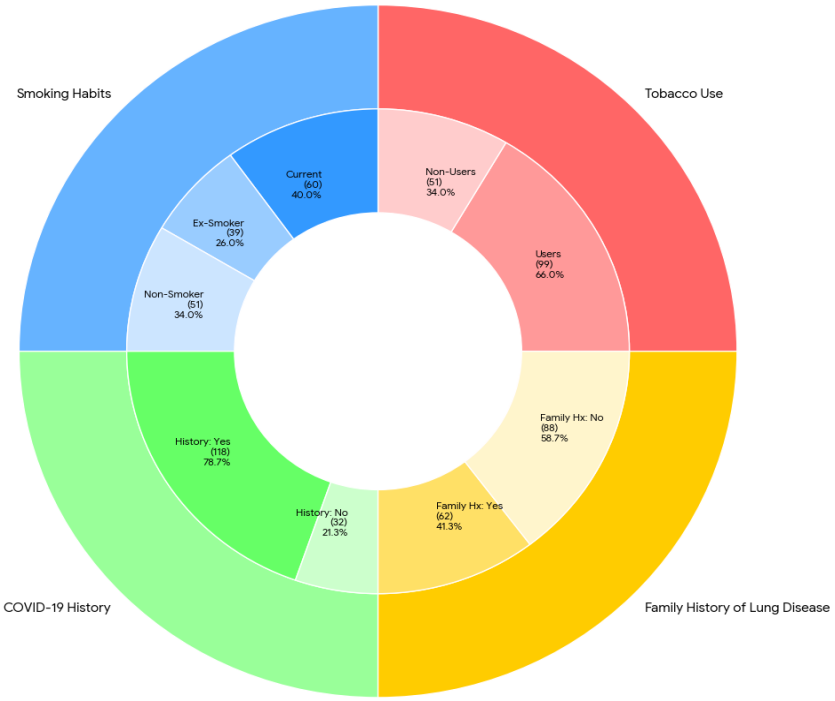


**Figure 5: Comprehensive Health Profile Analysis (n=150)**

**Table 1** presents the mean SUA levels (mean ± SD) across different demographic and socio-economic subgroups of COPD patients at admission (n = 150).

- **Age groups:** Uric acid levels were highest in patients ≤50 years (6.81 ± 1.23) and gradually declined with increasing age, reaching the lowest in the 71–80 years group (6.35 ± 1.44).
- **Gender:** Levels were almost identical between males (6.54 ± 1.14) and females (6.55 ± 1.28), indicating no significant gender difference in uric acid among COPD patients.
- **Occupation:** The lowest mean uric acid was seen in government employees (5.84 ± 0.97), while housewives (6.89 ± 1.28) and those in the "others" category (6.86 ± 1.29) had the highest values.
- **Socio-economic class:** The upper class (7.03 ± 1.50) showed the highest uric acid levels, while the lower class (6.26 ± 1.07) and upper-middle/lower-middle groups (6.36 ± ~1.3) had comparatively lower values.

**Table 1: Mean SUA Levels (Mean ± SD) Across Demographic and Socio-economic Subgroups of COPD Patients at Admission (n = 150)**

| Variable | Uric acid (mean ± SD) |
|---|---|
| **Age (in years)** | |
| Up to 50 (n =40) | 6.81 ± 1.23 |
| 51-60 (n =34) | 6.53 ± 1.19 |
| 61-70 (n =35) | 6.45 ± 1.12 |
| 71-80 (n =25) | 6.35 ± 1.44 |
| 81 and above (n =16) | 6.36 ± 1.13 |

| | |
|---|---|
| **Gender** | |
| Male (n=68) | 6.54 ± 1.14 |
| Female (n=82) | 6.55 ± 1.28 |
| **Occupation** | |
| Agriculture (n =18) | 6.22 ± 1.29 |
| Govt. employee (n =11) | 5.84 ± 0.97 |
| Housewife (n =26) | 6.89 ± 1.28 |
| Business (n =30) | 6.66 ± 1.07 |
| Others (n =65) | 6.86 ± 1.29 |
| **Socio-Economic Class** | |
| Upper (n=9) | 7.03 ± 1.50 |
| Upper-middle (n=38) | 6.36 ± 1.36 |
| Lower-middle (n=27) | 6.36 ± 1.24 |
| Upper-lower (n=36) | 6.64 ± 1.28 |
| Lower (n=40) | 6.26 ± 1.07 |

Figure 6 shows the mean blood uric acid levels of COPD patients at admission, categorised by smoking habits. The values were 6.43 ± 1.21 for current smokers, 6.58 ± 1.16 for ex-smokers, and 6.65 ± 1.28 for non-smokers. Although non-smokers had slightly higher mean uric acid levels compared to smokers, the differences across groups were minimal. The one-way ANOVA test yielded F = 0.44 with a p-value of 0.64, indicating that the variation was not statistically significant.

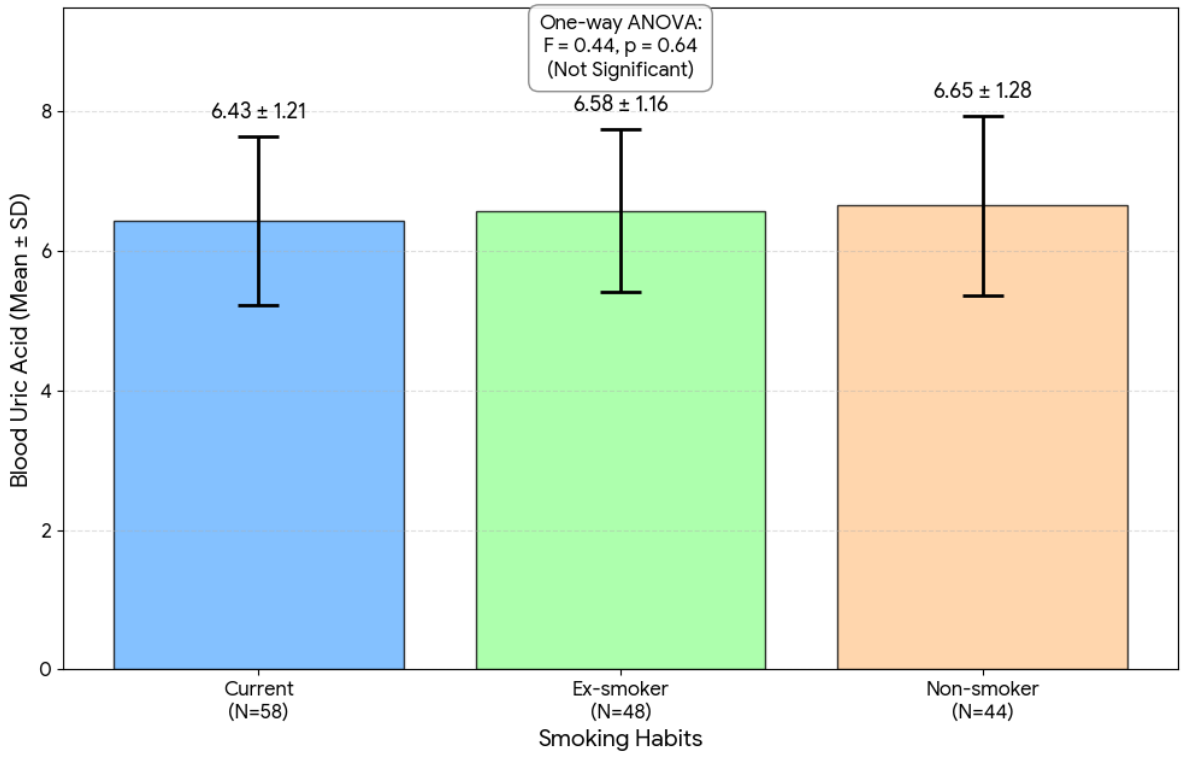


**Figure 6: Blood Uric Acid (Mean ± SD) Distribution by Smoking Habits (n=150)**

The mean SUA level in COPD patients at admission was 6.43 ± 1.21 mg/dL and decreased significantly to 4.70 ± 0.75 mg/dL at discharge. The paired t-test result ($t = 14.3$, $p < 0.001$) confirms that this reduction was highly statistically significant (Figure 7).

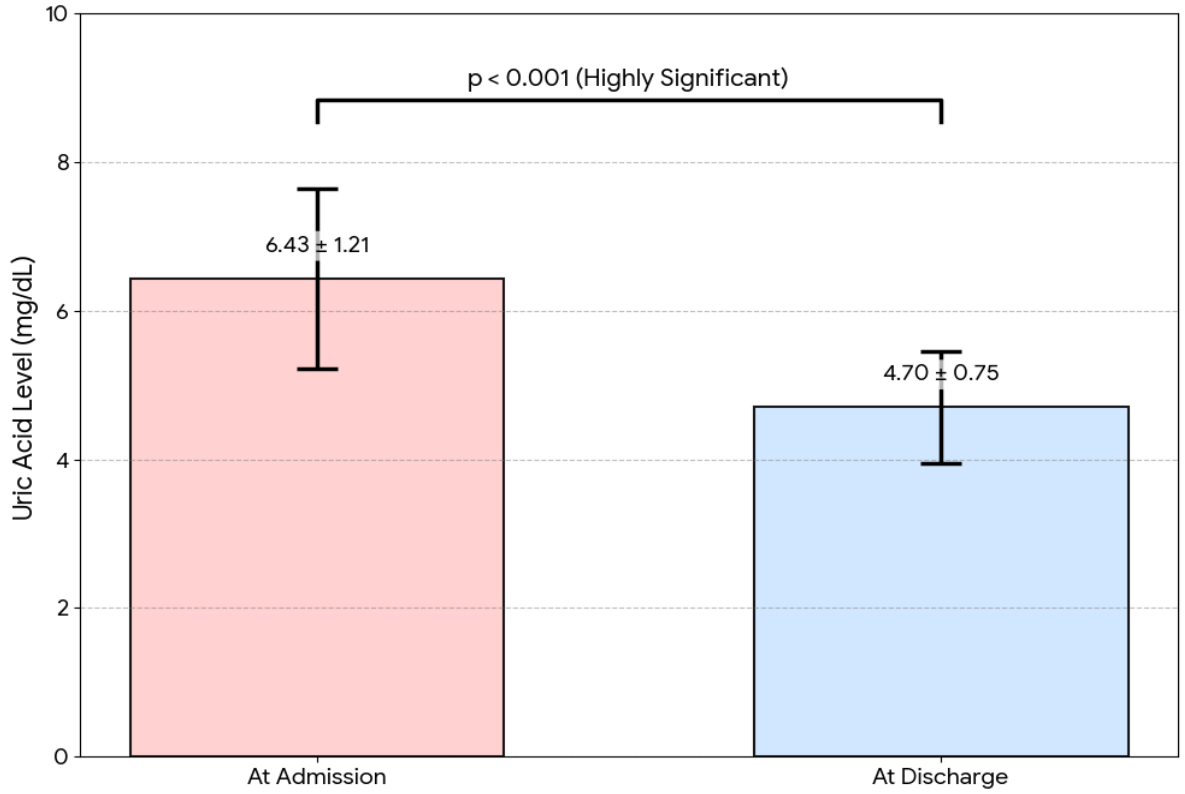


**Figure 7: Comparison of Blood Uric Acid Levels: Admission vs. Discharge**

## DISCUSSION

The present study investigated SUA levels among admitted COPD patients in a tertiary care hospital in Tripura, highlighting its potential role as a biomarker of disease severity. The findings demonstrated that mean SUA levels at admission (6.43 ± 1.21 mg/dL) were significantly higher than at discharge (4.70 ± 0.75 mg/dL), with the reduction highly statistically significant ($p < 0.001$). This supports the hypothesis that uric acid levels reflect the metabolic and oxidative stress burden in COPD and may serve as a useful adjunct in patient monitoring.

### Uric Acid and COPD Pathophysiology

COPD is characterised by chronic inflammation, oxidative stress, and progressive tissue damage. Hypoxia and increased purine catabolism in COPD patients contribute to elevated SUA levels, which act as both antioxidants and pro-oxidants depending on the context. This dual role has been described as "janus-faced," reflecting its protective antioxidant capacity and its potential to exacerbate systemic inflammation.[8,13] The significant decline in SUA levels observed at discharge in our study suggests that clinical improvement and resolution of hypoxemia reduce purine turnover, thereby lowering uric acid concentrations.

### Comparison with Previous Studies

Our findings are consistent with earlier reports that COPD patients exhibit higher SUA levels compared to healthy controls. Rao et al. (2024) demonstrated a strong positive association between SUA and COPD severity in a hospital-based cohort, while Yang et al. (2022) confirmed that SUA correlates with impaired lung function and hypoxia.[14,15] The admission SUA levels in our cohort (mean 6.43 mg/dL) align with these observations, reinforcing the biomarker's relevance in clinical practice.

Interestingly, we observed no significant gender differences in SUA levels, with males and females showing nearly identical values (6.54 ± 1.14 vs. 6.55 ± 1.28 mg/dL). This contrasts with population-based studies, in

which men typically exhibit higher SUA levels due to hormonal influences (Chen et al., 2022).[16] The lack of difference in our COPD cohort may reflect disease-related metabolic alterations overriding baseline gender variations.

**Influence of Demographic and Socioeconomic Factors**

Age-wise analysis revealed slightly higher SUA levels in younger patients (≤50 years: 6.81 ± 1.23 mg/dL), with levels declining with age. Similar patterns have been observed in recent studies, where younger COPD patients demonstrated higher SUA values linked to oxidative stress and disease severity, while older patients showed comparatively lower levels due to comorbid physiological changes. Earlier research on hyperuricemia supports this age-related oxidative stress burden, with younger patients exhibiting stronger associations between SUA, inflammation, and oxidative stress markers.[17] Occupation also influenced SUA levels, with housewives and individuals in the "others" category showing higher SUA levels than government employees.

Socioeconomic status showed a clear gradient, with upper-class patients exhibiting the highest SUA levels (7.03 ± 1.50 mg/dL). This finding may be related to dietary patterns, lifestyle factors, and comorbidities prevalent in higher socioeconomic groups. Conversely, lower-class patients had comparatively lower SUA levels, possibly due to nutritional deficiencies or reduced purine intake. These observations highlight the complex interplay between socioeconomic determinants and biochemical markers in COPD.

**Smoking and Uric Acid Levels**

Contrary to expectations, nonsmokers in our study had slightly higher SUA levels (6.65 ± 1.28 mg/dL) compared to current smokers (6.43 ± 1.21 mg/dL). However, the differences were not statistically significant ($p = 0.64$). This finding diverges from the conventional understanding that smoking exacerbates oxidative stress and elevates SUA. One possible explanation is that indoor air pollution, such as burning of biomass fuel, wood exposure, and coal fuel exposure, is associated with COPD in the non-tobacco smoker group.[18] This underscores the importance of considering non-smoking exposures in COPD pathogenesis in Indian populations.

**Clinical and Public Health Implications**

The significant reduction in SUA levels at discharge indicates that uric acid can serve as a dynamic biomarker reflecting disease activity and treatment response. Its measurement is inexpensive, widely available, and requires minimal infrastructure, making it particularly valuable in resource-limited settings such as Tripura. Incorporating SUA estimation into routine COPD management could aid in risk stratification, monitoring therapeutic efficacy, and identifying patients at higher risk of poor outcomes.

Furthermore, the absence of strong associations with gender or smoking status in our study indicates that SUA may provide independent prognostic information beyond traditional risk factors. However, the influence of socioeconomic and occupational exposures suggests that contextual factors must be considered when interpreting SUA levels.

**Limitations and Future Directions**

The study was limited by its cross-sectional design and hospital-based sampling, which may not fully capture community-level variations. Additionally, exclusion of patients with comorbidities such as hypertension, diabetes, and renal disease—conditions known to influence SUA—may restrict generalisability. Future longitudinal studies with larger, diverse populations are warranted to validate SUA as a prognostic biomarker and to explore its utility in predicting exacerbations and long-term outcomes.

## CONCLUSION

This study demonstrates that serum uric acid is a promising biomarker for assessing disease severity and treatment response in COPD patients. The significant reduction in SUA levels at discharge highlights its dynamic role in reflecting clinical improvement. Unlike traditional risk factors such as gender and smoking, SUA provided independent prognostic information, while socioeconomic and occupational exposures influenced baseline values. Given its affordability and accessibility, SUA estimation can be integrated into routine COPD management, especially in resource-constrained regions. Future longitudinal studies are warranted to validate its prognostic utility and explore its role in predicting exacerbations and long-term outcomes.

### *Declaration by Authors*

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**Source of Funding:** None
**Conflict of Interest:** The authors declare no conflict of interest.

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