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Evaluation of C-Reactive Protein and Uric Acid/Creatinine Ratio as Biomarkers in the Assessment of COPD Patients Attending a Tertiary Care Centre of Tripura, North-Eastern India

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is a major global health burden, with systemic inflammation and oxidative stress contributing to its progression. C-reactive protein (CRP) and the uric acid/creatinine ratio (UCR) have emerged as promising biomarkers for disease monitoring.
Objective: To evaluate CRP and UCR levels among COPD patients admitted to a tertiary care centre in Tripura, North-Eastern India, and to assess their correlation with clinical severity and recovery.
Methods: A descriptive cross-sectional study was conducted on 150 COPD patients admitted to Agartala Government Medical College & GBP Hospital. Serum CRP, uric acid, and creatinine were measured using standard biochemical assays. Comparative analyses were performed between admission and discharge values, with subgroup analyses by age, gender, occupation, and socio-economic status.
Results: Mean CRP levels declined significantly from 11.34 ± 4.35 mg/L at admission to 2.35 ± 0.85 mg/L at discharge ($p < 0.001$). Similarly, UCR decreased from 5.45 ± 1.74 to 4.78 ± 1.04 ($p < 0.001$). Elevated CRP was consistently associated with systemic inflammation, while higher UCR values correlated with hypoxemia and socio-economic deprivation. No significant correlations were observed between CRP and UCR, underscoring biomarker heterogeneity.
Conclusion: CRP and UCR are cost-effective, accessible biomarkers that reflect systemic inflammation and oxidative stress in COPD. Their significant reduction during hospitalisation highlights their utility in monitoring therapeutic response and disease stability. Region-specific validation supports their relevance for resource-limited settings such as Tripura.

Keywords: Chronic obstructive pulmonary disease, C-reactive protein, Uric acid/creatinine ratio, Biomarkers, Correlation, Tripura

INTRODUCTION

Chronic obstructive pulmonary disease (COPD), which is one of the most common pulmonary disorders worldwide, is manifested as persistent respiratory symptoms due to airflow limitation. Usually, airway and/or alveolar abnormalities of COPD occur from significant exposure to noxious particles or gases, most notably due to tobacco smoke. [1] Chronic obstructive pulmonary disease (COPD) is a chronic respiratory disorder characterized by persistent airflow limitation resulting from abnormalities of the airways and/or alveoli. Chronic bronchitis may be associated with an obstructive ventilatory defect, characterized by persistent airway obstruction and a reduced ratio of forced expiratory volume in the first second to forced vital capacity (FEV_1/FVC), typically defined as $\leq 70\%$. Chronic respiratory failure represents an advanced manifestation of chronic obstructive airway disease and is characterized by persistent airflow obstruction accompanied by hypoxemia. Emphysema is defined anatomically by the abnormal and permanent enlargement of airspaces distal to the terminal bronchioles, associated with destruction of the walls of the alveoli and alveolar ducts, with relatively little or no obvious fibrosis. These pathological changes contribute to persistent airflow limitation and impaired pulmonary gas exchange, which are characteristic features of COPD. [2]

According to the World Health Organization (WHO), COPD is currently the fourth leading cause of death and is predicted to become the third leading cause of mortality by 2030. [3] Although the course of disease is progressive and often punctuated by acute exacerbations, it is a preventable and treatable disease. [4]

The development and progression of chronic obstructive pulmonary disease (COPD) are influenced by several environmental and host-related factors, including ageing, infectious diseases, and cigarette smoking. [5] Smoking is strongly associated with progressive deterioration in pulmonary function and contributes to a decline in lung function parameters. [6] Cigarette smoking remains one of the most important modifiable risk factors for the development of COPD. However, epidemiological evidence indicates that chronic airflow limitation can also occur in individuals who have never smoked, suggesting that other environmental and host-related factors may contribute to disease development. [7] Compared with non-smokers, cigarette smokers have a higher prevalence of respiratory symptoms and pulmonary function abnormalities, a more rapid annual decline in forced expiratory volume in the first second (FEV_1), and a higher mortality rate attributable to COPD. [8]

The morphological pattern of emphysema also varies according to its underlying etiology. Centrilobular emphysema is characterized by dilation and destruction predominantly involving the respiratory bronchioles and is strongly associated with cigarette smoking. In contrast, panlobular emphysema involves relatively uniform dilation and destruction of the entire pulmonary lobule and is more commonly associated with α1-antitrypsin deficiency. [9]

In addition to pulmonary manifestations, COPD is associated with several systemic complications and comorbidities. These include metabolic syndrome, cachexia, skeletal muscle abnormalities, osteoporosis and osteopenia, depression, anaemia, cardiovascular disease, asthma, cancer, hypertension, and stroke. [10] The presence of these systemic manifestations may contribute substantially to disease burden, functional impairment, and overall morbidity and mortality among individuals with COPD. [10]

C- Reactive Protein (CRP), an acute-phase protein, is the most widely used biomarker when investigating and monitoring

respiratory infections. The CRP level is consistently higher in AECOPD (Acute Exacerbation of COPD) than during recovery. Acute Exacerbation of COPD associated with H influenzae or S pneumoniae incurred the highest CRP levels. Elevated C-reactive protein (CRP) levels have been associated with increased disease severity and poorer clinical outcomes. Higher CRP concentrations are correlated with a greater likelihood of intensive care unit (ICU) transfer, endotracheal intubation and the requirement for mechanical ventilation, as well as the development of congestive heart failure. Furthermore, elevated CRP levels have been associated with increased in-hospital mortality and higher mortality following hospital discharge, suggesting that CRP may serve as an important marker of disease severity and adverse prognosis. [11] Although CRP is considered a non-specific but reliable marker for systemic inflammation and disease activity in COPD, its measurement is simple, cost-effective, and accessible in most clinical laboratories.

Serum uric acid (sUA), a major end product of purine catabolism, is an important extracellular antioxidant present within the respiratory tract. Because the respiratory tract is continuously exposed to increased oxidative stress from cigarette smoke, biomass fuel combustion, and industrial air pollution, antioxidants such as uric acid, ascorbic acid, α-tocopherol, and ferritin present along the respiratory epithelium play an important protective role by inhibiting lipid peroxidation and scavenging reactive oxygen and nitrogen species. [12] Hyperuricemia has been implicated in the pathogenesis and progression of several respiratory disorders, including pulmonary arterial hypertension and obstructive sleep apnea. [13] However, its role in COPD and acute exacerbations remains unclear, with studies suggesting both potentially protective and detrimental effects, thereby highlighting the need for further prospective investigations. [14]

In patients with COPD, coexisting hyperuricemia has been associated with greater deterioration in pulmonary function compared with patients with COPD without elevated uric acid levels. Histopathological examination of lung tissue from patients with concomitant COPD and hyperuricemia has demonstrated increased inflammatory cell infiltration, more extensive airway destruction, and greater degrees of pulmonary fibrosis. Furthermore, elevated uric acid concentrations may contribute to the progression of airway inflammation in COPD through mechanisms involving the transfer of extracellular vesicles, potentially amplifying local inflammatory responses and structural airway damage. [15]

The uric acid/creatinine ratio (UCR) has emerged as a more refined and stable biomarker compared to serum uric acid alone, especially in patients with varying renal function. Since creatinine is a marker of renal filtration, UCR adjusts uric acid levels for the patient's kidney function, improving its reliability and interpretability. [16] In particular, UCR has shown promising results as a cost-effective and non-invasive marker for identifying disease severity, with a study reporting a cut-off value around 8–9 mg/g predicting exacerbation risk with high sensitivity and specificity. [17]

To the best of our knowledge, no study has been conducted in Tripura on the predictors or severity of COPD. Tripura, being a Northeastern state with a predominantly rural population, faces unique challenges in managing chronic diseases like COPD. Patients often present late with advanced symptoms and co-morbidities such as cor pulmonale. There is a pressing need for simple, affordable, and locally applicable biomarkers to aid in early identification of exacerbations and complications.

Despite the established value of CRP and UCR in the global literature, region-specific studies of these parameters from North-Eastern India are lacking. Moreover, the combinations of these biomarkers have not been systematically evaluated among admitted COPD patients in Tripura, where

the burden of the disease is increasing steadily. This study aims to evaluate and compare CRP and UCR levels in patients admitted with COPD at a tertiary care teaching hospital in Tripura. By doing so, the study seeks to assess their correlation with clinical severity, exacerbation frequency, and the presence of complications such as cor pulmonale.

MATERIALS AND METHODS

Study Design and Setting

The present investigation employed a descriptive, cross-sectional design conducted over two years. Data acquisition and laboratory procedures were completed within 8 months. The study was carried out in the Department of Biochemistry at Agartala Government Medical College (AGMC) & GBP Hospital, with collaborative support from the Department of Respiratory Medicine at AGMC & GBP Hospital.

Study Population

Participants were COPD patients admitted to the Respiratory Medicine unit at AGMC & GBP Hospital. Recruitment was consecutive and based on predefined eligibility criteria, ensuring that all eligible patients during the study period were enrolled.

Sample Size and Sampling

An initial calculation suggested a minimum sample size of 62. [18] To enhance statistical power and precision, the sample size was expanded to 150 patients. A census sampling approach was adopted, in which all admitted COPD patients who met the inclusion criteria were invited to participate.

Eligibility Criteria

Inclusion:

- Confirmed COPD cases admitted to the Respiratory Medicine department.
- Clinically stable patients without exacerbation in the preceding three months.
- No evidence of active lower respiratory tract infection at enrolment.
- Provision of written informed consent.

Exclusion:

- Patients with pulmonary tuberculosis.
- History of chronic systemic illnesses (e.g., hypertension, cardiac disease, renal disease, diabetes, hypothyroidism, gout, malignancy, autoimmune disorders).
- Pregnant women.
- Patients unwilling to participate.
- Current use of uric acid-lowering medications.

Procedures

Eligible COPD patients were enrolled at admission, and post-treatment patients were enrolled at discharge; the latter served as the comparison group. Each participant underwent detailed history taking, clinical examination, and documentation in a structured case record form. Venous blood samples were collected under aseptic precautions for biochemical analysis.

Estimation of Serum Creatinine

Serum creatinine was measured using the Enzymatic Method. [19]

- **Sample volume:**4µL serum
- **Reagent 1:**180µL
- **Reagent 2:**60 µL

Analysis was performed on the XL-640 Fully Automated Clinical Chemistry Analyser, and OD was recorded.

Estimation of Serum Uric Acid

Serum uric acid was measured using the Uricase–UV method. [20]

- **Sample volume:** 5 µL serum
- **Reagent 1:** 200 µL
- **Reagent 2:** 50 µL. Analysis was performed on the XL-640 Fully Automated Clinical Chemistry Analyser. Optical density was read at 505 nm, and SUA concentration was calculated automatically using calibration standards.

Estimation of C-Reactive Protein

Serum C-Reactive Protein was measured by Latex-enhanced Immuno-turbidimetric Assay. [21]

Blood Sample Collection

5 (Five) millilitres of venous blood were drawn aseptically, preferably from the antecubital vein, and transferred into clot activator tubes. After clotting at room temperature, serum was separated by centrifugation at 3000 rpm for 3–5 minutes. All assays were performed on the same day to maintain sample integrity.

Data Management and Statistical Analysis

Data were entered into SPSS version 25 (Windows platform). Descriptive statistics were used for categorical variables, which were presented in the text, tables, and figures. Comparative analysis was performed using paired t-tests. A p-value < 0.05 was considered statistically significant.

Ethical Considerations

Approval was obtained from the Institutional Ethics Committee of AGMC prior to study initiation (Ref. No. F.4(6-13)/AGMC/ Medical Education/IEC Approval/2022/ 18835). Written informed consent was secured from all participants. Confidentiality of patient data was strictly maintained, and information was used solely for research purposes in compliance with biomedical ethics guidelines.

RESULTS

The study included 150 participants. Among 150 study participants, 45% are males. The remaining 55% of participants are female, indicating slightly higher female representation in the study (Table 1/Fig 1).

Table-1: Distribution of study participants according to gender (n=150)

Variable	n (%)
Gender	
Male	68 (45)
Female	82 (55)
Total	**150 (100)**

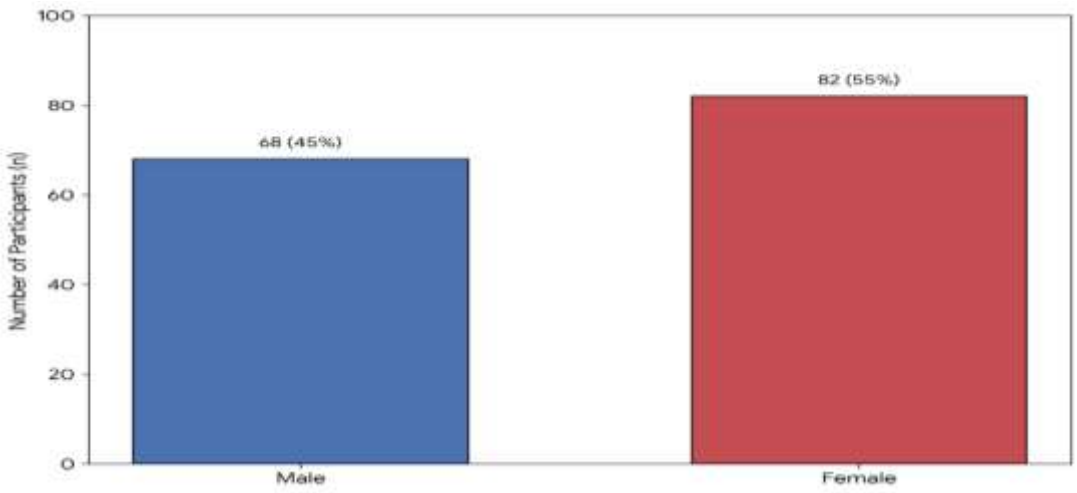

Fig 1: Distribution of Study Participants according to Gender

Table 2: Blood level of CRP (Mean ± SD) of COPD patients at the time of admission according to Age (n=150)

Variable	CRP (mean ±SD)
Age (in years)	
Upto 50 (n=40)	12.19±4.70
51-60 (n=34)	11.26±3.39
61-70(n=35)	12.63±4.33
71-80(n=25)	12.33±5.14
81and above (n=16)	13.15±4.50

Mean CRP levels were compared across age groups among study participants. The CRP values showed slight variation with age, ranging from 11.26 ± 3.39 mg/L to 13.15 ± 4.50 mg/L. The highest mean CRP level (13.15 ± 4.50 mg/L) was observed in participants aged 81 years and above (Table 2/Fig-2).

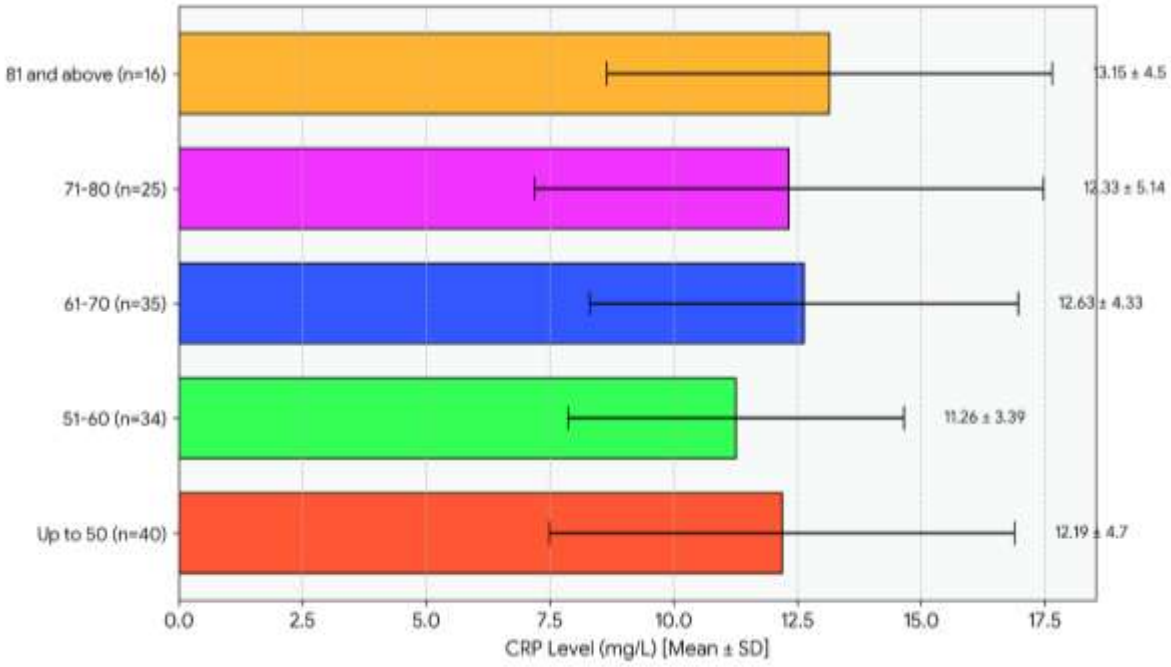

Fig 2: Assessment of C-Reactive Protein (CRP) Levels Across Age Demographics in COPD Patients

Table 3: Blood level of CRP (Mean ± SD) of COPD patients at the time of admission according to Gender (n=150)

Variable	CRP mean±SD
Gender	
Male (n=68)	12.06±4.19
Female(n=82)	12.26±4.51

The mean CRP levels were analysed according to gender. It was found that male participants had a mean CRP level of 12.06 ± 4.19 mg/L, while females had 12.26 ± 4.51 mg/L. So, it was obvious that among the study participants, CRP levels were slightly higher in females than in males (Table 3).

Table 4: Blood level of CRP (Mean ± SD) of COPD patients at the time of admission according to individual Occupation (n=150)

Variable	CRP mean ±SD
Occupation	
Agriculture (n =18) Govt.	12.31±4.52
employee (n=11)	12.38±3.85
Housewife (n =26)	12.75±5.01
Business(n=30)	13.19±3.92
Others(n=65)	10.87±3.78

When comparing blood CRP levels among study participants across different occupations, the levels ranged from 10.87 ± 3.78 mg/L to 13.19 ± 3.92 mg/L. Participants involved in business had the highest mean CRP levels, while those in other services had the lowest (Table 4).

Table 5: Blood level of CRP (Mean±SD) of COPD patients at the time of admission according to Socio-economic status (n=150)

Variable	CRP mean±SD
Socio-Economic Class	
Upper (n=15)	12.17±2.14
Upper-middle (n=30)	11.32±4.12
Lower-middle (n=20)	12.74±4.53
Upper-lower (n=35)	12.92±4.67
Lower (n=50)	12.39±4.51

The mean CRP levels are analysed across different socio-economic classes. CRP levels were relatively similar across groups, ranging from 11.32 ± 4.12 mg/L to 12.92 ± 4.67 mg/L. Participants in the lower-middle class showed a slightly higher mean CRP level than other groups (Table 5).

Table 6: Socio-economic status-wise blood level of CRP (Mean ± SD) of COPD patients at admission (n=150)

Variable	UA/Cr (Mean±SD)
Age (in years)	
Upto 50 (n=40)	6.02±1.90
51-60 (n =34)	5.57±1.56
61-70 (n =35)	5.73±1.76
71-80 (n =25)	5.62±2.03
81 and above (n=16)	5.12±1.15

Participants aged 0-50 years had the highest uric acid–creatinine ratio, with a mean of 6.02 ± 1.90, indicating relatively elevated levels in this younger age group. The UA/C ratio showed a slight decline in the subsequent age groups, with mean values of 5.57 ± 1.56 (51–60 years), 5.73 ± 1.76 (61–70 years), and 5.62 ± 2.03 (71–80 years). The lowest mean uric acid–creatinine ratio was observed in individuals aged 81 years and older, at 5.12 ± 1.15, suggesting an overall decreasing trend with advancing age (Table 6).

Table 7: Uric acid/Creatinine ratio (mean ± SD) of COPD patients at the time of admission according to Gender (n=150)

Variable	UA/Cr (mean ±SD)
Gender	
Male (n=68)	5.58±1.72
Female (n=82)	5.79±1.77

The mean blood UA/Creatinine ratio was slightly higher in females (5.79 ± 1.77) in comparison to the male group (5.58 ± 1.72). Both genders showed comparable variability in values, as indicated by similar standard deviations. Overall, the data suggested minimal gender-based differences in blood uric acid–creatinine ratio levels (Table 7).

Table 8: Blood UA/Cr ratio (mean ± SD) of COPD patients according to occupation at the time of admission (n=150)

Variable	UA/Cr (mean ±SD)
Occupation	
Agriculture (n =18)	5.34±1.98
Govt. employee (n=11)	4.57±1.02
Housewife (n =26)	6.09±1.85
Business (n=30)	5.29±1.32
Others (n=65)	6.52±1.89

The mean uric acid–creatinine ratio varied across occupational groups, with the highest values observed among individuals in other services (6.52 ± 1.89) and housewives (6.09 ± 1.85). Government employees showed the lowest mean ratio (4.57±1.02), indicating comparatively lower levels in this group. Overall, uric acid–creatinine ratios showed noticeable variation by occupation, suggesting that occupational lifestyle factors might influence these biochemical levels (Table 8).

Table 9: Uric acid/Creatinine ratio (Mean ± SD) of COPD patients at the time of admission according to Socio-economic status (n=150)

Variable	Uric acid-creatinine ratio
Socio-Economic Class	
Upper (n=15)	4.55±1.51
Upper-middle (n=30)	5.12±1.55
Lower-middle (n=20)	5.24±1.46
Upper-lower (n=35)	6.07±2.14
Lower (n=50)	6.33±1.99

The mean uric acid–creatinine ratio showed a gradual increase from the upper socio-economic class (4.55 ± 1.51) to the lower socio-economic class (6.33 ± 1.99). Individuals belonging to the lower-middle class had intermediate values, with a mean ratio of 5.24±1.46. Overall, the findings suggested that lower socio–economic status was associated with slightly higher uric acid–creatinine ratios (Table 9).

Table10: Comparison of blood Uric acid, CRP, UA/Creatinine ratio values at the time of admission and discharge

Variable	At Admission (Mean±SD)	At Discharge (Mean±SD)	Paired t-test p-value (2-tailed)
CRP (mg/L)	11.34±4.35	2.35±0.85	22.9(<0.001)
UA/Creatinine ratio	5.45±1.74	4.78±1.04	3.7(<0.001)

There was a significant improvement in various laboratory parameters from admission to discharge ($p<0.001$). The uric acid/creatinine ratio also decreased

significantly, reflecting improved renal function and reduced oxidative stress (t-approx. 3.7, p<0.001). Similarly, CRP levels dropped substantially, signifying a significant reduction in systemic inflammation (t-approx. 22.9, p<0.001). Overall, the observed changes in these parameters between admission and discharge were statistically highly significant, underscoring clinical improvement during hospitalisation (Table 10).

DISCUSSION

In the present study, COPD patients admitted to a tertiary care centre in Tripura showed significant changes in both C-reactive protein (CRP) and the uric acid-to-creatinine ratio (UCR) between admission and discharge, underscoring the utility of these biomarkers for assessing systemic inflammation and oxidative stress. The demographic profile revealed a slight female predominance (55%), which aligns with recent Indian studies reporting increasing COPD prevalence among women due to biomass fuel exposure and underdiagnosis. [22] Salvi and Barnes highlighted that nearly half of rural Indian women with COPD are affected by household smoke exposure, while Aryal et al. suggested greater biological susceptibility among women even with lower cumulative smoking exposure. [23,24] These findings reinforce that COPD is no longer confined to male smokers but is increasingly a disease of women in resource-limited settings.

CRP as a marker of systemic inflammation

Our cohort showed a marked decline in CRP from 11.34 ± 4.35 mg/L at admission to 2.35 ± 0.85 mg/L at discharge ($p < 0.001$), confirming its role in monitoring disease activity. Elevated CRP has consistently been associated with poor outcomes in COPD, including ICU transfer and mortality. [25] Rath et al. reported significantly higher CRP levels in patients with stable COPD compared with controls ($p < 0.001$). [26] Aksu et al. similarly found elevated CRP in stable COPD patients independent of smoking or biomass exposure. [27] Treatment-related reductions have also been documented: De Torres et al. showed CRP ≥3 mg/L was associated with poor prognosis and higher mortality, [28] while Banerjee et al. confirmed elevated CRP predicts mortality risk in COPD. [25] These findings are consistent with our results and confirm CRP's utility in monitoring therapeutic response. These findings are consistent with our results and confirm CRP's utility in monitoring therapeutic response.

Importantly, CRP variations across gender, occupation, and socioeconomic groups in our study were modest and not statistically significant. De Torres et al. noted that CRP correlated more strongly with lung function impairment and BODE index than with demographic factors. [28] Gallego et al. further demonstrated that CRP was independently associated with COPD severity and hospitalisation risk, with levels >100 mg/L predicting a fourfold increased risk of hospital admission. [29] Sönnerfors et al. highlighted that systemic inflammation persists in COPD patients even among ex-smokers and never-smokers. [30] Thus, once COPD is established, inflammation persists due to ongoing airway injury and comorbidities rather than demographic variables.

The prognostic value of CRP is well established. Banerjee et al. showed that elevated CRP was associated with significantly higher all-cause mortality (HR 2.70; 95% CI 1.08–6.81) compared with patients without elevated CRP. [25] Therefore, the marked reduction in CRP observed in our study not only reflects symptomatic improvement but may also indicate reduced future risk of exacerbation and mortality.

Uric acid/creatinine ratio (UCR) as a novel biomarker

Uric acid metabolism in COPD is complex, with both antioxidant and pro-inflammatory roles. Adjusting for renal function via UCR enhances interpretability. In our study, UCR decreased significantly from 5.45 ± 1.74 at admission to 4.78 ± 1.04 at discharge ($p < 0.001$), reflecting reduced oxidative stress

during recovery. Rumora et al. reported elevated UA/Cr ratios in COPD patients compared with controls, with higher values in GOLD III–IV stages and correlations with smoking pack-years. [31] Abdel Halim et al. similarly found higher UA/Cr ratios in COPD (5.6 ± 2.1 vs 3.8 ± 1.4 mg/mmol, $p < 0.001$), which were strongly correlated with arterial hypoxemia and predicted severity with a cutoff of 4.2 mg/mmol. [32] Büyükbayram et al. demonstrated that the UA/Cr ratio was significantly higher during exacerbations (6.33 ± 2.14) compared to stable COPD (4.82 ± 1.53) and controls (3.79 ± 1.17; $p < 0.001$), with elevated values linked to longer hospital stay and higher CRP (35). Rumora et al. demonstrated that the UA/Cr ratio was significantly higher in COPD patients than in controls, and elevated values were strongly correlated with systemic inflammatory markers (CRP, IL-6, TNF-α) and disease severity. [31] These findings corroborate our results, highlighting UCR as an integrated marker of systemic inflammation and instability.

Our study also found slightly higher UCR values among lower socioeconomic classes, consistent with evidence that deprivation increases oxidative stress and worsens outcomes. Eisner et al. reported that lower socioeconomic strata had significantly worse inflammatory profiles and poorer COPD outcomes regardless of smoking status. [33] This suggests that socioeconomic factors may indirectly influence biomarker levels through environmental exposures and healthcare access.

Biomarker heterogeneity and clinical implications

Interestingly, no significant correlations were observed between CRP and uric acid, CRP and UCR, or uric acid and creatinine at admission or discharge, reflecting the heterogeneous nature of systemic inflammation and metabolic dysregulation in COPD. Eagan et al. similarly reported weak or absent correlations among CRP, fibrinogen, leukocyte count, and IL-6. [34] Agustí et al. emphasised COPD's biological heterogeneity, noting that only subsets of patients show persistent systemic inflammation. [35] Endotype-based studies further support this: Xie et al. identified distinct inflammatory endotypes in COPD, showing that metabolic-inflammatory endotypes were associated with higher uric acid and UA/Cr, while classic inflammatory endotypes were characterised by elevated CRP and IL-6. [36] Thus, CRP and UCR are complementary rather than interchangeable biomarkers in COPD.

CONCLUSION

Overall, our findings confirm that CRP and UCR are powerful, cost-effective biomarkers for COPD management. Their significant reduction during hospitalisation reflects clinical improvement and underscores their role in monitoring systemic inflammation and oxidative stress. Fresh evidence from 2024–2026 supports their correlation with disease severity, making them highly relevant for routine use in Tripura and similar resource-limited settings.

Limitations and future directions

Our study was cross-sectional and single-centred. Future multicentric, longitudinal studies should validate CRP and UCR as predictors of exacerbation frequency, hospitalisation, and mortality. Correlating biomarker levels with spirometry (FEV1 decline) would further establish their prognostic value. Additionally, exploring biomarker cut-offs specific to Indian populations could enhance clinical applicability.

Declaration

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