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Original Article

Association of Serum Uric Acid with Disease Severity in Hospitalized Patients with Acute Exacerbation of COPD in Bangladesh

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ABSTRACT

Background: Acute Exacerbation of COPD (AECOPD) is a major cause of morbidity and mortality. Serum uric acid (SUA) has emerged as a potential biomarker for oxidative stress and disease severity. **Aim of the study**: To characterize the clinical and epidemiological profile of hospitalized AECOPD patients. **Methods & Materials**: This observational study was conducted at a tertiary care chest hospital in Bangladesh from July 2019 to June 2020. Ninety-six patients admitted with AECOPD were enrolled via purposive sampling and stratified into two groups based on admission SUA levels: Group A (Low SUA, <6.9 mg/dL; n=48) and Group B (High SUA, ≥6.9 mg/dL; n=48). Data on socio-demographics, clinical history, and GOLD stage were collected. Statistical analysis was performed using SPSS version 20. **Results**: The mean age of participants was 55.18 (±8.52) years, with a majority being male, from rural areas, and of lower-middle socioeconomic status. Most were current or ex-smokers (69.79%). No significant differences were found in these baseline characteristics between the two SUA groups. However, a significantly higher proportion of patients in the high SUA group had severe/very severe COPD (83.33% vs. 22.92%, p<0.001) and experienced acute respiratory failure (64.58% vs. 22.92%, p=0.001). Median SUA levels showed a strong positive correlation with worsening GOLD stage (P < 0.001). **Conclusion**: Elevated serum uric acid on admission is significantly associated with more severe airflow limitation and a higher incidence of acute respiratory failure in hospitalized AECOPD patients, suggesting its utility as a prognostic biomarker in this population.

Keywords: Bangladesh, COPD, Exacerbation, Epidemiology, GOLD Criteria, Uric Acid

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) represents a major global health burden, with rising prevalence and standing as a foremost cause of illness and death globally. An acute exacerbation of COPD (AECOPD) is defined by a substantial and sudden worsening of symptoms that often requires an adjustment in routine medication [1]. The disease, which includes chronic bronchitis and emphysema, presents with persistent airflow limitation, dyspnea, cough, and other clinical manifestations. It is associated with significant illness and fatality, ranking as the third most common cause of mortality in developed nations [2]. While primarily linked to smoking and remaining a prevalent cause of death, COPD's pathological processes can originate early in life, leading to premature mortality, though it predominantly impacts individuals aged 40–50 years. Key risk factors for developing COPD later in life include tobacco use, diabetes, and vitamin D deficiency. The World Health Organization (WHO) estimated that in 2005, 64 million people were living with moderate to severe COPD, and the condition was responsible for over 3 million deaths, accounting for 5% of global mortality. Approximately 90% of these fatalities occur in low- and middle-income countries [3]. The prognosis for COPD is generally poor. Research into prognostic factors has been conducted to aid in grading disease severity, guiding management strategies, predicting the rate of lung function decline, and informing patient education and expectations. A reduced forced expiratory volume in one second (FEV1) is a recognized predictor of outcomes, alongside variables like smoking, a low body mass index (BMI), reduced exercise capacity, male sex, and comorbidities such as heart failure. The BODE index, which integrates Body mass index, airflow Obstruction, Dyspnoea, and Exercise capacity, was specifically created to assess risks of mortality and hospitalization in COPD patients [4]. Serum uric acid, the end product of purine metabolism, rises markedly under hypoxic conditions. Higher levels have been linked to systemic inflammation and an elevated risk of cardiovascular events. Accordingly, increased serum uric acid has been observed in respiratory conditions like obstructive sleep apnea and pulmonary hypertension. In COPD, cigarette smoke triggers oxidative stress and pulmonary inflammation, causing tissue damage and a decline in lung function. This impairment in pulmonary function leads to reduced oxygen intake and tissue hypoxia, a condition that becomes more pronounced during an AECOPD [4]. Uric acid (UA) serves as a primary non-enzymatic antioxidant within the lungs. In conjunction with other antioxidants, it helps neutralize oxidants generated by cigarette smoke. This antioxidant capacity is believed to confer beneficial effects by mitigating the development of both COPD and lung cancer [5]. This study was therefore designed to assess the potential utility of serum uric acid as a biomarker for predicting outcomes in patients hospitalized with AECOPD, including the duration of their hospital stay and their requirement for non-invasive ventilation (NIV) support.

METHODS & MATERIALS

This cross-sectional analytical study was conducted at the Department of Respiratory Medicine, National Institute of Diseases of the Chest and Hospital (NIDCH), from July 2019 to June 2020. A final cohort of 96 patients admitted with a primary diagnosis of Acute Exacerbation of COPD (AECOPD) was selected via purposive sampling. Purposive sampling was employed to deliberately select patients with confirmed AECOPD who fulfilled predefined inclusion and exclusion criteria,

thereby ensuring a clinically relevant and homogeneous study population for accurate assessment of the relationship with serum uric acid levels. Inclusion required patients to be >40 years with confirmed AECOPD. Key exclusions were a history of gout, chronic kidney disease, hepatic failure, other significant respiratory pathologies, or a debilitated state to minimize confounders affecting serum uric acid (SUA). After obtaining ethical approval from the Institutional Review Board (IRB) of the National Institute of Diseases of the Chest and Hospital (NIDCH), and after securing informed written consent from all participants, enrolled participants were stratified into two groups based on admission SUA levels: Group A (Low SUA, <6.9 mg/dL; n=48) and Group B (High SUA, ≥6.9 mg/dL; n=48) [6]. Data on socio-demographics, clinical history, smoking status, and GOLD severity stage were collected using a structured questionnaire as primary outcomes. All patients underwent standardized investigations, including spirometry, arterial blood gas analysis, chest X-ray, and comprehensive blood tests. Data were analyzed using SPSS version 20. Categorical and continuous variables were expressed as percentages and mean ± SD, respectively. Intergroup comparisons employed Pearson's chi-square and Student's t-test. Correlations were assessed using Pearson's test, with a p-value <0.05 deemed statistically significant.

RESULT

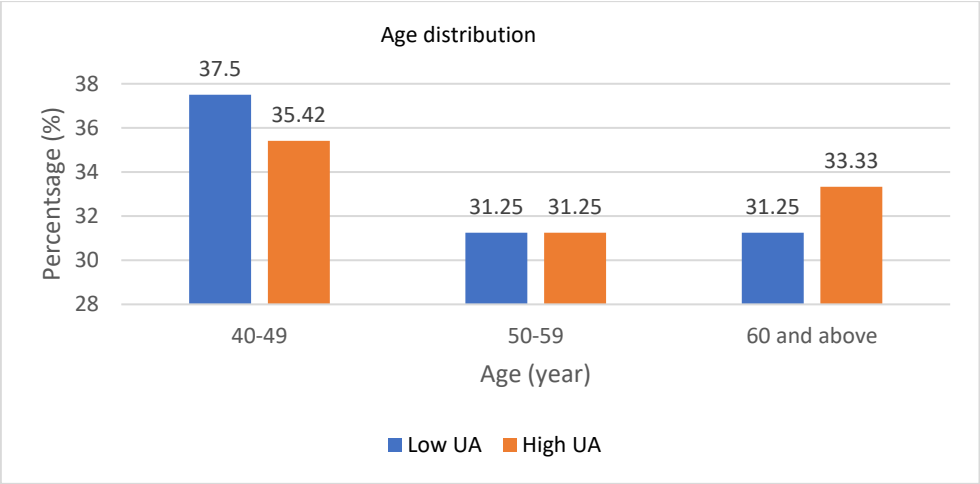

Figure 1 Age distribution of patients (*n*=96).

The study comprised 96 patients with a mean age of 55.18 ± 8.52 years (range: 40-73), with the majority (67.71%) belonging to the 40–59-year age group. There were no significant differences in baseline demographic characteristics between the groups with low and high serum uric acid (SUA) levels. This included age (p=0.385) *Figure 1*, sex distribution (p>0.05) *Table I*.

Table I
Distribution of study groups according to sex (*n*=96).

Sex	Group A (n=48) No. (%)	Group B (n=48) No. (%)	Total (n=96) No. (%)	P value
Female	11(22.92%)	6(12.5%)	17(17.71%)	0.189
Male	37(77.08%)	42(87.5%)	79(82.29%)	

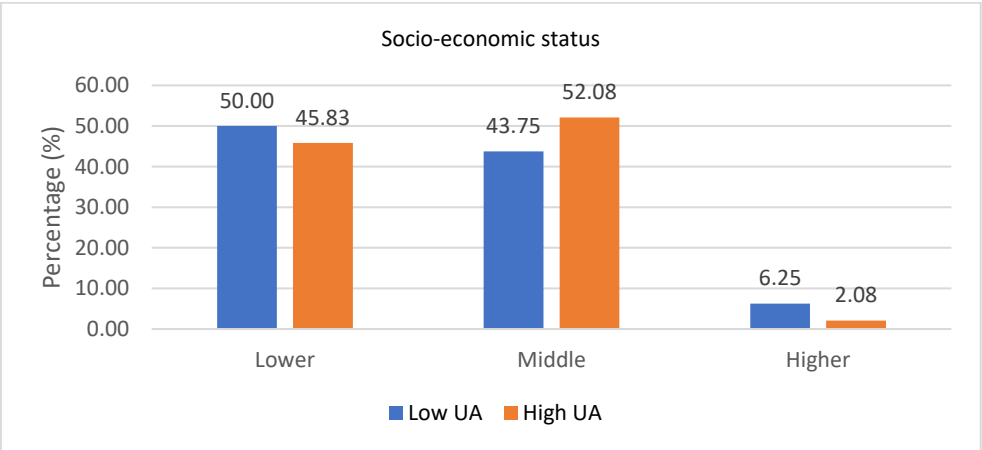

Figure 2 Socio-economic status of patients (*n*=96).

Socio-economic status (95.83% lower-middle class, *p*=0.488 (*Figure 2*).

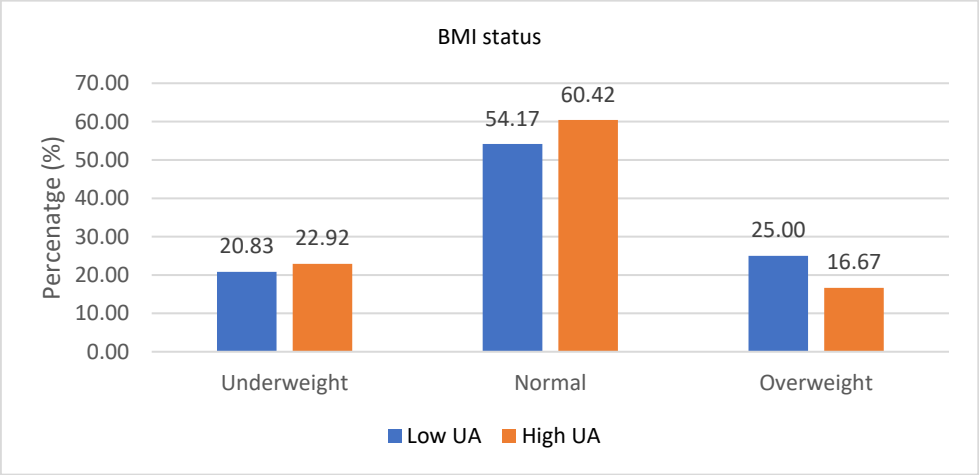

Figure 3 BMI distribution of patients (*n*=96).

The mean BMI was 21.83 ± 3.23, with most patients (57.29%) in the normal weight category, showing no intergroup difference (p=0.534) *Figure 3*.

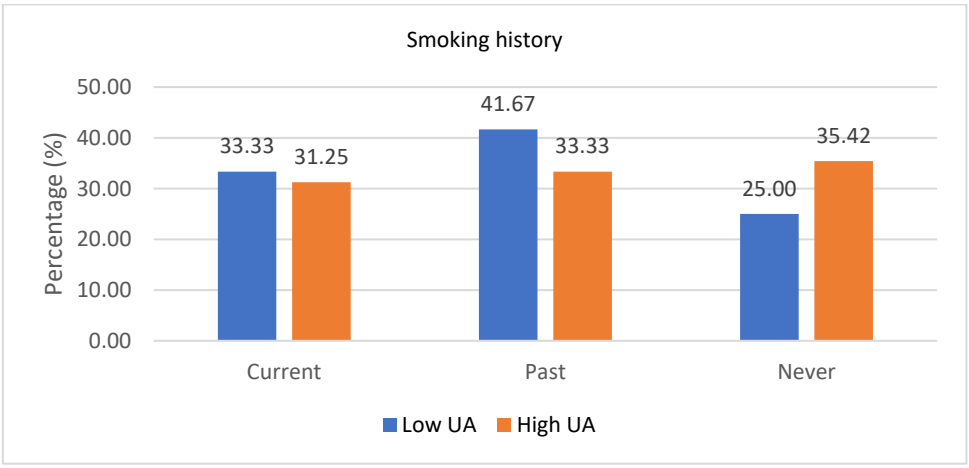

Figure 4 Smoking history of patients (*n*=96).

A significant majority (69.79%) were current or past smokers, with no difference between groups (p=0.512). Key significant differences were observed in clinical severity and outcomes (*Figure 4*).

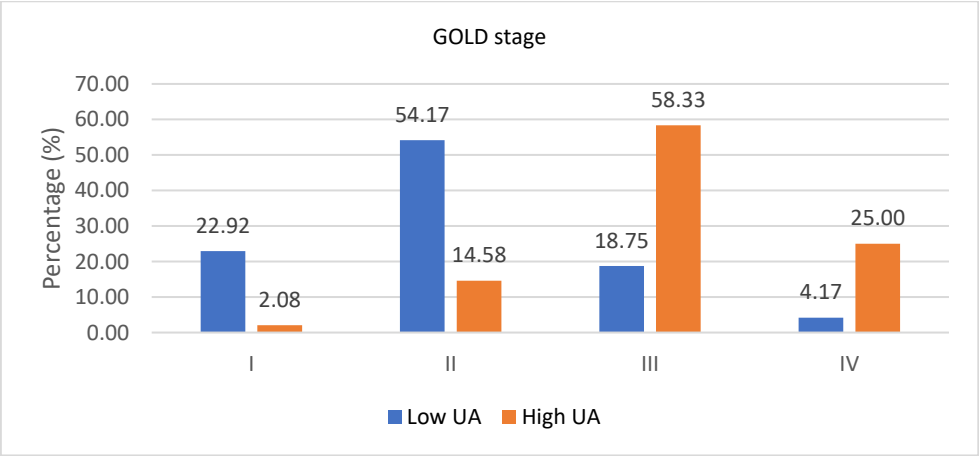

Figure 5 GOLD stage of COPD among patients (*n*=96).

The distribution of COPD severity by GOLD stage differed markedly between groups. The majority of patients with low SUA were classified as GOLD stage II (54.17%) (*Figure 5*) and had mild/moderate COPD (77.08%) *Figure 6*.

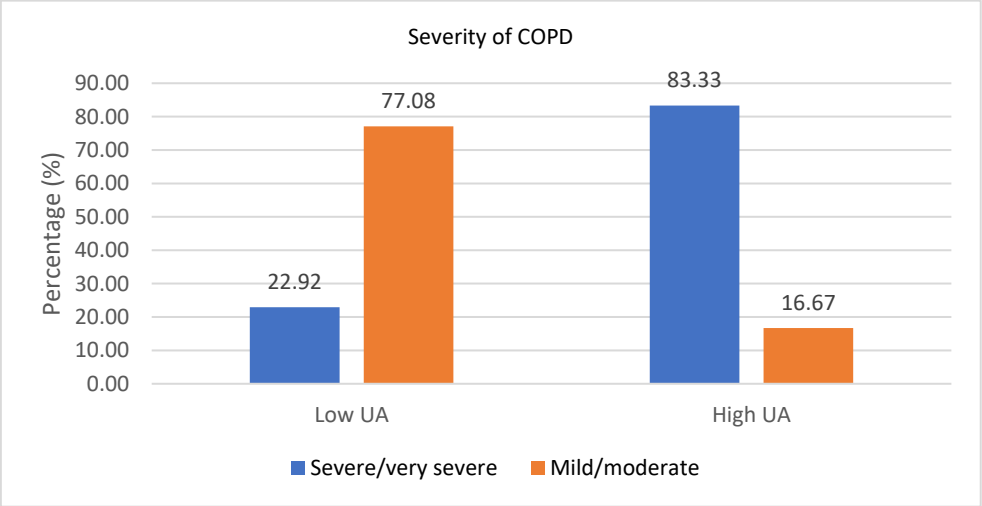

Figure 6 Severity of COPD among patients (*n*=96).

In contrast, the majority with high SUA were GOLD stage III (58.33%) (Figure 5) and had severe/very severe COPD (83.33%) (p<0.001 for both) *Figure 6*. Comparison of acute exacerbation severity of COPD patients between Group A and Group B showed a highly statistically significant difference (p<0.001). In Group A, mild exacerbation was observed in 11 (22.92%) patients, while no patients in Group B had mild disease. Moderate exacerbation was more frequent in Group A, 26 (54.17%), compared to 8 (16.67%) in Group B. In contrast, severe exacerbation was markedly higher in Group B, 40 (83.33%), compared to 11 (22.92%) in Group A. Overall, severe cases constituted the majority 51 (53.13%) of total patients, followed by moderate 34 (35.42%) and mild 11 (11.46%) cases. Regarding the classification of severe types of acute exacerbation of COPD, a significant difference was also observed between the two groups (p<0.001). No respiratory failure was seen equally in both groups, 9 (18.75%) each. However, acute respiratory failure that was non-life-threatening was more common in Group B, 19 (39.58%), compared to 2 (4.17%) in Group A. Life-threatening acute respiratory failure was observed only in Group B, 12 (25%), while none was recorded in Group A (Table II).

Table II
Classification of acute exacerbation of COPD patients (*n*=96).

Types	Group A (n=48) No. (%)	Group B (n=48) No. (%)	Total (n=96) No. (%)	p value
Mild	11 (22.92%)	0 (0%)	11 (11.46%)	<0.001
Moderate	26 (54.17%)	8 (16.67%)	34 (35.42%)	
Severe	11 (22.92%)	40 (83.33%)	51 (53.13%)	
Classes of severe types of acute exacerbation of COPD				
No respiratory failure	9 (18.75%)	9 (18.75%)	18 (18.75%)	<0.001
Acute respiratory failure is non-life-threatening	2 (4.17%)	19 (39.58%)	21 (21.88%)	
Acute respiratory failure life-threatening	0 (0%)	12 (25%)	12 (12.50%)	

p-value was determined by the Pearson Chi-square test

Laboratory findings showed statistically significant differences between the two groups across all measured variables (p<0.05). Mean serum uric acid level was significantly higher in Group B (8.42±1.02) compared to Group A (5.7±0.77) (p<0.001). Similarly, FEV1 (%) was significantly lower in Group B (37.92±12.03) than in Group A (62.10±14.91) (p<0.001). Arterial blood gas parameters also demonstrated significant variation: pH was lower in Group B (7.37±0.04 vs 7.40±0.03, p<0.001), while PaCO2 was higher in Group B (43.38±4.77 vs 41.02±3.26, p=0.006). PaO2 was significantly reduced in Group B (61.60±6.82 vs 64.73±5.34, p=0.014), and oxygen saturation (SpO2) was also lower in Group B (92.21±3.92 vs 95.19±2.62, p<0.001) *Table III*.

Table III
Laboratory findings of both groups (*n*=96).

Variables	Group A(n=48) Mean ± SD	Group B(n=48) Mean ± SD	P value
Uric acid	5.7±0.77	8.42±1.02	<0.001**
FEV1 (%)	62.10±14.91	37.92±12.03	<0.001**
pH	7.40±0.03	7.37±0.04	<0.001**
PaO_2	64.73±5.34	61.60±6.82	0.014**
$PaCO_2$	41.02±3.26	43.38±4.77	0.006**
SpO_2 (%)	95.19±2.62	92.21±3.92	<0.001**

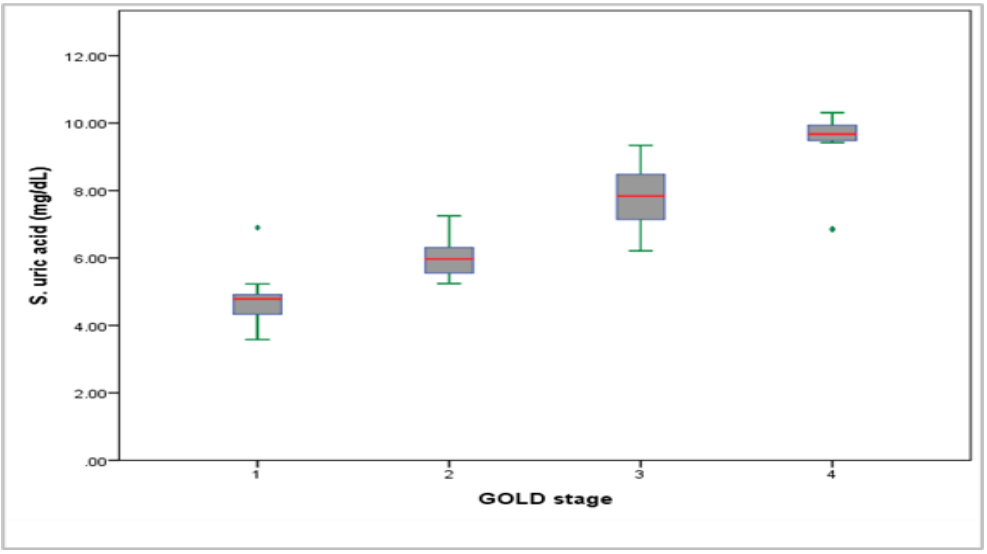

Figure 7 Serum uric acid levels on admission in all studied patients according to GOLD stage (*n*=96).

Figure 7 illustrates serum uric acid levels according to GOLD stage among all studied patients (N=96), showing a progressive increase in uric acid concentration with worsening disease severity.

DISCUSSION

Chronic obstructive pulmonary disease (COPD) remains a leading global cause of morbidity and mortality and is the third leading cause of death in developed countries [7]. This underscores the critical need to identify reliable prognostic biomarkers that can facilitate early, intensified therapy for high-risk patients to improve survival outcomes. In this study of 96 patients admitted with AECOPD, the mean age was 55.18 ± 8.52 years, with the majority (36.46%) in the 40–49-year age group. The prevalence of COPD increases with age, a trend supported by the well-documented physiological decline in respiratory function that begins around 30–40 years of age [8]. Furthermore, advanced age has been consistently identified as a significant risk factor for the disease [9]. We observed a pronounced male predominance (82.29%), which aligns with the classical epidemiological profile of COPD, where males have historically been at higher risk due to greater exposure to smoking and occupational inhalants [8]. This finding is consistent with other studies that also report a male preponderance [5,10]. The mean BMI across our cohort was 21.83 ± 3.23, and a significant majority (70%) had a positive smoking history. Both low BMI and smoking are well-established risk factors for COPD. Our findings corroborate those of Islam et al. (2013) [11], who identified low BMI and smoking as major risk factors, and Alam et al. (2015) [2], who found underweight and smoking to be significant predictors of the disease. The pathophysiological link is robust; cigarette smoke induces a complex inflammatory response in the airways and lung parenchyma [12,13]. This is characterized by infiltrates of CD8+ lymphocytes and neutrophils, accumulation of macrophages and epithelial cells, and the release of mediators that perpetuate inflammation and contribute to airway remodeling, a hallmark of COPD [14,15]. A central finding of our study is the strong association between elevated serum uric acid (SUA) levels and disease severity. The majority of patients with normal SUA were classified as GOLD stage II (54.17%), whereas most with high SUA were stage III (58.33%). Crucially, median SUA levels demonstrated a highly significant stepwise increase with worsening GOLD stage ($P < 0.001$) [16]. Receiver operating characteristic (ROC) analysis established a cut-off value of 7.38 mg/dL with high sensitivity (76.5%) and specificity (100%) for identifying severe disease. This finding is in strong agreement with the work of Bartziokas et al. (2014) [5], who also identified SUA as a significant predictor of COPD severity and outcomes. This suggests that SUA, potentially acting as a biomarker of hypoxia and oxidative stress during exacerbations, holds substantial promise as a practical and prognostic tool for risk stratification in clinical settings.

LIMITATIONS

This study has limitations, including a single-center design, modest sample size, and a purposive sampling method. These factors limit generalizability and may introduce selection bias, while the observational design prevents causal inferences.

CONCLUSION

This study demonstrated that elevated serum uric acid levels are significantly associated with greater COPD severity, higher GOLD stages, and an increased incidence of acute respiratory failure in hospitalized patients. These findings suggest that serum uric acid can serve as a valuable prognostic biomarker for identifying high-risk patients during acute exacerbations. Incorporating this measure into clinical assessments could improve risk stratification and guide more aggressive management strategies for severe COPD cases in Bangladesh.

RECOMMENDATION

Serum uric acid should be integrated into the routine assessment of AECOPD patients to improve risk stratification. Future multi-center studies with larger cohorts are recommended to validate these findings and explore their role in guiding targeted therapeutic interventions for high-risk individuals.

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