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Beyond Standard Biomarkers: Investigating the Role of Hemostatic Biomarkers in Predicting Outcomes of Acute Exacerbation of Chronic Obstructive Pulmonary Disease. A Prospective Cohort Study

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ABSTRACT

Background: To evaluate the prognostic role of admission hemostatic biomarkers—mean platelet volume (MPV), D-dimer, and fibrinogen—in patients with chronic obstructive pulmonary disease (COPD), with particular emphasis on adverse outcomes and in-hospital mortality during acute exacerbations of COPD (AECOPD).

Methods: This was a Prospective cohort study conducted in Pak Emirates Military Hospital (PEMH), Rawalpindi, Pakistan, from August 11, 2024, to January 11, 2025. A total of 300 adults aged 40–65 years with spirometry-confirmed COPD (post-bronchodilator $FEV_1/FVC < 0.70$) were enrolled, including 159 patients classified as stable COPD and 141 presenting with AECOPD. Demographic and clinical characteristics, spirometric indices, fibrinogen, D-dimer, and MPV were recorded. Fibrinogen was assessed by the Clauss method, D-dimer by a high-sensitivity immunoturbidimetric assay, and MPV from the admission complete blood count. Outcomes included in-hospital mortality, intensive care unit (ICU) admission, length of hospital stay, and clinical deterioration requiring oxygen or ventilatory support. Independent-samples t tests, chi-square testing, effect-size estimation, and receiver operating characteristic (ROC) curve analysis were performed using IBM SPSS Statistics version 25.

Results: Of 300 patients, 240 (80.0%) were male and 60 (20.0%) were female; 22 patients died during hospitalization. AECOPD patients had higher fibrinogen (568 ± 112 vs 378 ± 68 mg/dL) and D-dimer concentrations (1,650 ± 850 vs 450 ± 180 µg/L) than stable COPD patients, while mean MPV in AECOPD was 10.8 ± 1.4 fL. Compared with survivors, patients who died had lower FEV_1 (38.2 ± 12.1% vs 53.1 ± 16.4%) and higher MPV (11.6 ± 1.5 vs 9.8 ± 1.3 fL), D-dimer (2,520 ± 1,245 vs 1,148 ± 852 µg/L), and fibrinogen (710 ± 148 vs 468 ± 112 mg/dL). ROC analysis demonstrated good discriminatory performance for reverse-coded FEV_1 (AUC 0.920), fibrinogen (AUC 0.830), MPV (AUC 0.823), and D-dimer (AUC 0.765) for in-hospital mortality. ICU admission was also associated with mortality in the reported analysis ($\chi^2 = 5.42$, $p = 0.022$).

Conclusion: Admission fibrinogen, D-dimer, and MPV showed clinically relevant associations with adverse outcomes in COPD, and ROC analysis indicated potential value for mortality risk stratification. These inexpensive, readily available markers may complement spirometric and clinical assessment in hospitalized patients, although validation in larger multicenter cohorts and adjusted prognostic models is required.

Keywords	Acute exacerbation of chronic obstructive pulmonary disease; COPD; D-dimer; fibrinogen; mean platelet volume; mortality; prognosis.
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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a heterogeneous respiratory disorder characterized by persistent respiratory symptoms and airflow limitation, with a substantial and continuing global burden of morbidity and mortality. Contemporary burden estimates indicate that COPD affects hundreds of millions of people worldwide and remains a major cause of death, particularly in low- and middle-income settings where exposure to tobacco smoke, household air pollution, ambient particulate matter, and occupational inhalants remains common. Acute exacerbations of COPD (AECOPD) are clinically important events because they accelerate functional decline, increase hospitalization and healthcare utilization, and are associated with both short- and long-term mortality (1,2). Although airflow obstruction measured by spirometry remains central to COPD diagnosis, the clinical course of an exacerbation cannot be explained by lung function alone. AECOPD is accompanied by variable degrees of systemic inflammation, endothelial activation, platelet activation, and perturbation of the coagulation–fibrinolytic system. These mechanisms provide a biological rationale for investigating readily available hemostatic markers as indicators of disease severity and prognosis (3,4).

Fibrinogen is an acute-phase glycoprotein with central roles in coagulation, inflammation, and blood viscosity. Elevated circulating fibrinogen has been associated with greater COPD severity and increased exacerbation burden. In hospitalized AECOPD, higher fibrinogen concentrations have also been linked with adverse clinical features and failure of non-invasive ventilatory support, supporting its potential as a severity and prognostic biomarker (5,6). D-dimer is a fibrin degradation product generated following clot formation and fibrinolysis. Its concentration may rise during AECOPD because of systemic inflammation, endothelial dysfunction, immobilization, comorbidity, and activation of coagulation. Although D-dimer is nonspecific and must be interpreted in the clinical context, elevated values have been repeatedly associated with more severe acute illness and poor outcomes in hospitalized COPD populations (7,8).

Mean platelet volume (MPV) reflects average platelet size and is routinely available from automated hematology analysis. Larger platelets are metabolically and enzymatically more active, providing a plausible link between MPV, platelet activation, and systemic inflammatory states. Evidence regarding MPV in AECOPD remains inconsistent, with some studies suggesting prognostic value and others reporting no clear relationship with outcome. This uncertainty makes MPV useful to study alongside better-established hemostatic markers rather than as an isolated predictor (9,10). Risk prediction in AECOPD remains challenging. Recent reviews show that many prognostic models have methodological limitations and that readily measurable biomarkers may be useful as components of broader risk-stratification approaches rather than as stand-alone decision tools. In resource-constrained settings, inexpensive tests that are already part of routine admission assessment may be particularly attractive. The present study therefore evaluated fibrinogen, D-dimer, and MPV in patients with COPD and assessed their relationship with clinical outcomes, including in-hospital mortality, ICU admission, and length of stay, with a particular focus on the prognostic profile of AECOPD (11,12).

METHODS

This prospective cohort study was conducted at Pak Emirates Military Hospital (PEMH), Rawalpindi, Pakistan, from August 11, 2024, to January 11, 2025. Ethical approval was obtained from the institutional ethical review board before commencement of data collection. Participants were enrolled through non-probability purposive sampling after assessment of eligibility. Adults aged 40–65 years with a confirmed diagnosis of COPD were eligible. COPD was defined by a documented post-bronchodilator FEV_1/FVC ratio <0.70 in accordance with contemporary GOLD diagnostic principles.1 Participants were categorized clinically as stable COPD or AECOPD. AECOPD was defined as an acute worsening of respiratory symptoms beyond usual day-to-day variation that required additional treatment and/or hospital admission. Patients whose acute presentation was primarily explained by an alternative diagnosis such as pneumonia, pulmonary embolism, or lung malignancy were excluded. Patients with end-stage renal disease, hepatic cirrhosis, or active systemic inflammatory disease were also excluded because these conditions may independently alter hemostatic biomarkers (13).

The final study sample comprised 300 participants, including 159 patients with stable COPD and 141 patients with AECOPD. Demographic data, smoking status, COPD severity, hospital length of stay, ICU admission, and in-hospital survival status were recorded on a structured data collection form. COPD severity was classified using the available spirometric severity categories in the study dataset (14). Blood samples for hemostatic assessment were obtained during the initial 24 hours of admission or clinical assessment. Plasma fibrinogen was measured using the Clauss method. D-dimer was measured using a high-sensitivity immunoturbidimetric assay, with 500 µg/L used as the laboratory reference cut-off in the study protocol. MPV was obtained from the automated complete blood count performed at initial assessment. The units used for analysis were mg/dL for fibrinogen, µg/L for D-dimer, and femtoliters (fL) for MPV (15).

The primary outcome was in-hospital mortality. Secondary outcomes included ICU admission, hospital length of stay, and clinical deterioration requiring escalation of oxygen therapy or ventilatory support. For prognostic analyses, biomarker levels and FEV_1 were compared according to survival status. ROC curve analysis was used to quantify discrimination for mortality. Because lower FEV_1 indicates greater risk whereas higher biomarker values indicate greater risk, FEV_1 was reverse-coded before ROC analysis so that all ROC curves had a consistent direction of risk. Data were analyzed using IBM SPSS Statistics version 25. Continuous variables were summarized as mean ± standard deviation (SD) where appropriate, and categorical variables as frequency and percentage. Independent-samples t tests were used for reported comparisons of continuous measures, with Levene's test used only to assess

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equality of variances. Categorical associations were assessed using chi-square testing and Fisher's exact test where indicated. Effect sizes were expressed using Cohen's d, and odds ratios (ORs) with 95% confidence intervals (CIs) were reported for categorical outcomes. ROC curves were used to calculate the area under the curve (AUC) with 95% CIs. A two-sided p value <0.05 was considered statistically significant.

RESULTS

The study included 300 patients with COPD, comprising 159 (53.0%) with stable COPD and 141 (47.0%) with AECOPD. Overall, 240 (80.0%) participants were male and 60 (20.0%) were female. The observed age range was 42.3–65 years, with a mean age of 54.21 ± 7.15 years. Table 1 presents the demographic and clinical characteristics. Percentages in Table 1 have been recalculated consistently within each disease-group column.

Table 1. Demographic and clinical characteristics of COPD and AECOPD patients (n = 300)

Characteristic	Category	COPD (n=159), f	COPD %	AECOPD (n=141), f	AECOPD %
Age	Early middle age	51	32.1%	40	28.4%
	Mid-adulthood	57	35.8%	58	41.1%
	Late middle age	51	32.1%	43	30.5%
Gender	Male	100	62.9%	140	99.3%
	Female	59	37.1%	1	0.7%
COPD severity	Mild	34	21.4%	35	24.8%
	Moderate	59	37.1%	68	48.2%
	Severe	51	32.1%	29	20.6%
	Very severe	15	9.4%	9	6.4%
Length of hospital stay	1–5 days	57	35.8%	45	31.9%
	6–10 days	85	53.5%	73	51.8%
	11–16 days	17	10.7%	23	16.3%
ICU admission	No	117	73.6%	37	26.2%
	Yes	42	26.4%	104	73.8%
Mortality	Survived	150	94.3%	128	90.8%
	Died	9	5.7%	13	9.2%
Smoking	Non-smoker	60	37.7%	46	32.6%
	Current smoker	47	29.6%	46	32.6%
	Heavy smoker	52	32.7%	49	34.8%

Note: Percentages are calculated within the COPD and AECOPD columns. f = frequency; ICU = intensive care unit.

AECOPD was strongly concentrated among male participants in this cohort. Moderate airflow limitation was the most frequent severity category in both groups. AECOPD patients also showed a higher proportion of ICU admission (73.8%) and a higher proportion of in-hospital deaths (9.2%) than the stable COPD group (26.4% and 5.7%, respectively).

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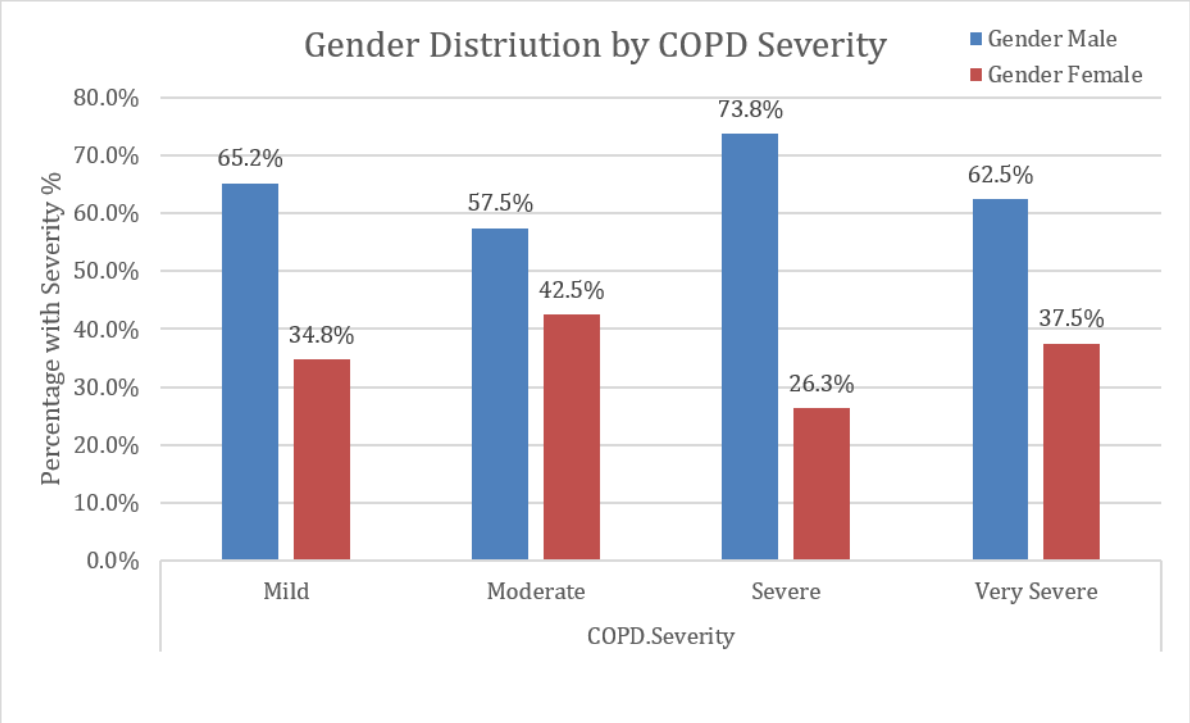

Figure 1. Gender distribution across COPD severity categories in the study population (n = 300).

Biomarker values were higher in AECOPD than in stable COPD. Mean fibrinogen was 568 ± 112 mg/dL in AECOPD compared with 378 ± 68 mg/dL in stable COPD. Mean D-dimer was 1,650 ± 850 µg/L in AECOPD compared with 450 ± 180 µg/L in stable COPD (reported $p < 0.001$), representing an approximately 3.7-fold difference. D-dimer exceeded 1,000 µg/L in 48% of AECOPD patients. Mean MPV in the AECOPD group was 10.8 ± 1.4 fL. Levene's test was interpreted only as an assessment of variance equality; group differences were interpreted from the reported between-group comparison.

Table 2. Mean differences in FEV_1, MPV, D-dimer, and fibrinogen according to mortality (n = 300)

Variable	Died (n=22)	Survived (n=278)	t value	p value	Cohen's d	Interpretation
FEV_1 (%)	38.2 ± 12.1	53.1 ± 16.4	4.52	0.001	0.98	Lower in deaths
MPV (fL)	11.6 ± 1.5	9.8 ± 1.3	4.85	0.091	1.32	Higher in deaths
D-dimer (µg/L)	2,520 ± 1,245	1,148 ± 852	7.80	0.001	1.45	Higher in deaths
Fibrinogen (mg/dL)	710 ± 148	468 ± 112	9.42	0.021	1.98	Higher in deaths

Note: Values are reported as mean ± SD. MPV = mean platelet volume; FEV_1 = forced expiratory volume in one second. Statistical values are retained from the source results supplied for the manuscript.

Patients who died had lower FEV_1 and higher MPV, D-dimer, and fibrinogen values than survivors. Based on the reported p values, differences in FEV_1, D-dimer, and fibrinogen reached statistical significance, while the MPV comparison did not ($p = 0.091$). The reported Cohen's d values nevertheless indicated large standardized differences for all four measures.

Table 3. Association between ICU admission and mortality (n = 300)

ICU status	Survived, n (%)	Died, n (%)	χ^2	df	p value	OR (95% CI)
No ICU admission (n=154)	146 (52.5%)	8 (36.4%)	5.42	1	0.022	Reference
ICU admission (n=146)	132 (47.5%)	14 (63.6%)				2.42 (0.98–5.96)
Total (n=300)	278 (100%)	22 (100%)				

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Note: Percentages in the survival columns describe the distribution of ICU status within survivors and deaths, respectively. OR = odds ratio; CI = confidence interval.

In the reported analysis, ICU admission was associated with mortality (χ^2 = 5.42, df = 1, p = 0.022), with an OR of 2.42 (95% CI 0.98–5.96). Fisher's exact test was also reported as p = 0.028. Mortality occurred in 14 of 146 ICU-admitted patients (9.6%) and 8 of 154 patients not admitted to ICU (5.2%).

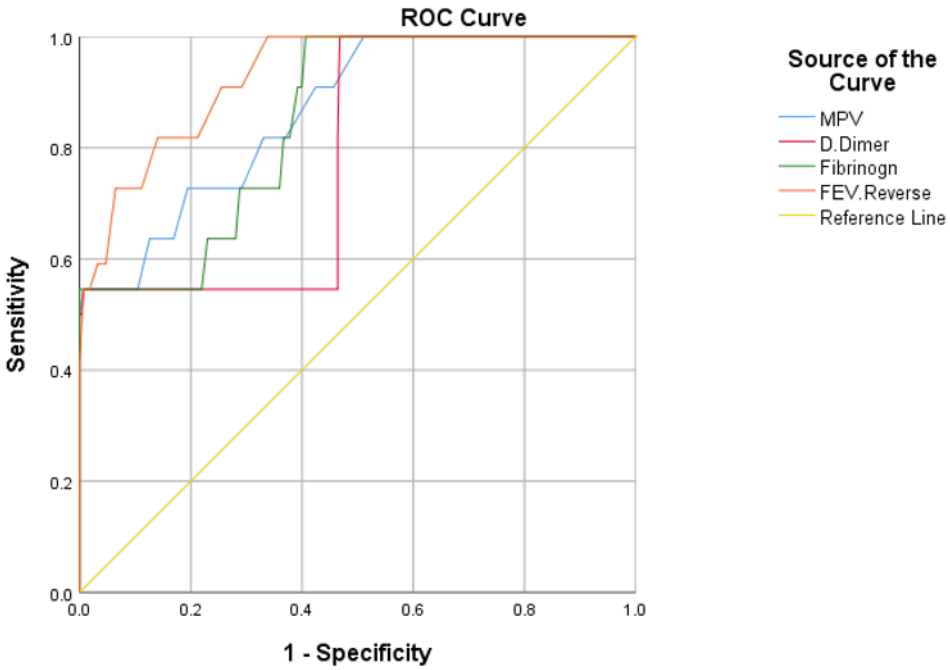

Figure 2. Receiver operating characteristic curves for FEV_1, MPV, D-dimer, and fibrinogen for in-hospital mortality.

ROC analysis demonstrated the strongest discrimination for reverse-coded FEV_1 (AUC = 0.920; 95% CI 0.872–0.986; p < 0.001). Among the hemostatic biomarkers, fibrinogen showed an AUC of 0.830 (95% CI 0.746–0.914; p < 0.001), MPV an AUC of 0.823 (95% CI 0.744–0.901; p < 0.001), and D-dimer an AUC of 0.765 (95% CI 0.667–0.864; p < 0.001). The study dataset identified exploratory thresholds of FEV_1 <42.5%, fibrinogen >685 mg/dL, MPV >11.2 fL, and D-dimer >2,150 µg/L for mortality discrimination. These thresholds should be regarded as cohort-derived and require external validation.

DISCUSSION

The present prospective cohort study evaluated readily available hemostatic biomarkers in 300 patients with COPD and found a consistent pattern of higher fibrinogen, D-dimer, and MPV values in the acute exacerbation group and in patients with adverse outcomes. The principal prognostic finding was that lower FEV_1 and higher admission hemostatic-marker concentrations characterized patients who died during hospitalization. ROC analysis further suggested useful discrimination for mortality, particularly for reverse-coded FEV_1 and fibrinogen, followed by MPV and D-dimer. The observed elevation of fibrinogen in AECOPD is biologically plausible and consistent with contemporary evidence. Fibrinogen is both a coagulation factor and an acute-phase reactant, and its concentration rises during systemic inflammatory activation. Sun et al. reported that increased fibrinogen was associated with more severe AECOPD features and predicted non-invasive ventilation failure, supporting its relevance as a marker of acute disease burden.5 More recent work examining prothrombotic biomarkers has similarly demonstrated that exacerbation-prone COPD is accompanied by increased coagulation and fibrinolytic activity (16-18).

D-dimer was markedly higher in AECOPD than in stable COPD and was also higher among patients who died. D-dimer is not disease-specific; infection, inflammation, immobility, venous thromboembolism, cardiovascular disease, and increasing age can all elevate its concentration. Nevertheless, its rise during AECOPD may identify a subgroup with a greater systemic and thrombotic burden. The AUC of 0.765 observed in this cohort suggests moderate discrimination and supports interpretation of D-dimer as an adjunctive risk marker rather than a stand-alone prognostic test (19-21). MPV was also higher in patients who died, although the reported independent-samples comparison did not reach statistical significance (p = 0.091). This distinction is important because the large reported effect size and favorable AUC should not be interpreted as proof of an independent prognostic relationship. Existing literature is mixed. Ziaei et al. found no significant association between MPV and outcome in a cohort of patients presenting with AECOPD, illustrating the uncertainty surrounding this parameter. Accordingly, MPV may be most useful as part of a multimarker approach rather than as a single decision threshold (22-24).

Lung function remained an important marker of outcome. Patients who died had substantially lower FEV_1, and reverse-coded FEV_1 achieved the largest AUC in the ROC analysis. This agrees with systematic evidence showing that impaired lung function, prior

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severe exacerbations, comorbidity, age, and markers of acute physiologic derangement contribute to mortality risk in COPD. The finding also reinforces that hemostatic biomarkers should complement, not replace, clinical and respiratory assessment (25,26). AECOPD patients in this cohort had greater ICU utilization and mortality than stable COPD patients. Of the 141 AECOPD patients, 104 (73.8%) were admitted to ICU and 13 (9.2%) died. Across the full cohort, ICU admission was associated with mortality in the reported categorical analysis. Hospitalized exacerbations are recognized as high-risk events, and recent reviews emphasize the importance of identifying patients at risk of early deterioration using clinical variables, physiology, comorbidity, and biomarkers (27,28).

The ROC-derived thresholds identified in this study—FEV_1 <42.5%, fibrinogen >685 mg/dL, MPV >11.2 fL, and D-dimer >2,150 µg/L—may help generate hypotheses for future clinical risk-stratification tools. However, they should not be regarded as treatment thresholds. In particular, these findings do not establish an indication for anticoagulation, antiplatelet therapy, or fibrinolytic therapy. Any such treatment must remain based on established clinical indications because the present observational study did not evaluate therapeutic efficacy or bleeding risk (29). From a practical perspective, fibrinogen, D-dimer, and MPV are comparatively accessible laboratory measures and could be useful in settings where advanced prognostic tools are limited. Recent reviews of AECOPD prediction models show that many published models have a high risk of bias and insufficient external validation. The most appropriate future direction is therefore not to use these biomarkers in isolation, but to test them prospectively alongside age, comorbidity, oxygenation, hypercapnia, infection markers, exacerbation history, and validated clinical scores (18,27).

The study has several strengths, including prospective data collection, simultaneous evaluation of three inexpensive hemostatic markers, inclusion of clinically relevant in-hospital outcomes, and ROC-based assessment of discriminatory performance. The comparison between stable COPD and AECOPD also provides clinical context for the biomarker elevations observed during acute illness. Several limitations should be considered. This was a single-center study using non-probability purposive sampling, which may limit generalizability and introduce selection bias. The number of deaths was relatively small (22 overall and 13 in the AECOPD group), limiting the stability of mortality estimates and the feasibility of a robust multivariable prediction model. Biomarkers were measured at a single early time point, and residual confounding from infection, cardiovascular disease, medication exposure, renal function, oxygenation, hypercapnia, and other acute physiologic factors cannot be excluded. D-dimer is nonspecific, and MPV is sensitive to laboratory and pre-analytical conditions. The cohort-derived ROC cut-offs were not externally validated. In addition, the reported unadjusted statistical results should be interpreted as associations and discriminatory estimates rather than evidence of independent causation or treatment benefit.

CONCLUSION

Fibrinogen, D-dimer, and MPV were higher in patients with AECOPD and in those experiencing adverse in-hospital outcomes, while lower FEV_1 characterized patients who died. ROC analysis indicated good discriminatory performance for reverse-coded FEV_1, fibrinogen, and MPV and moderate discrimination for D-dimer. These findings support the potential use of routinely available hemostatic biomarkers as adjuncts to clinical and spirometric risk assessment in hospitalized COPD. Larger multicenter studies with prespecified sampling, adjusted multivariable analyses, serial biomarker measurements, and external validation are required before the proposed cut-offs can be adopted in routine practice.

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