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Unveiling the prognostic potential: Naples score for predicting in-hospital mortality in intensive care unit patients with acute exacerbation of COPD

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Abstract

To estimate in-hospital mortality and identify its independent predictors among patients with acute exacerbation of chronic obstructive pulmonary disease (AE-COPD) admitted to the general intensive care unit (ICU), focusing on the Naples Prognostic Score (NPS). This retrospective analysis was conducted in the general ICU of a tertiary university hospital between 2023 and 2024, including patients with AE-COPD. Demographic data, comorbidities, pulmonary function test results, arterial blood gas levels, and laboratory markers (including albumin, total cholesterol, neutrophil-to-lymphocyte ratio [NLR], lymphocyte-to-monocyte ratio [LMR], and lactate levels) were extracted. NLR and LMR were evaluated individually and as components of the Naples Prognostic Score (NPS) to comprehensively assess the combined impact of systemic inflammation and nutritional status on patient outcomes. The NPS was calculated as the sum of points (0–4), with 1 point assigned for each of the following: albumin level <4.0 g/dL, total cholesterol level ≤180 mg/dL, NLR≥2.96, and LMR≤4.44. We confirmed that albumin values were converted from g/L to g/dL for the calculation. Associations with in-hospital mortality were examined using univariate analyses and multivariate regression, and NPS discrimination was assessed using receiver operating characteristic (ROC) analysis (area under the curve [AUC], sensitivity, and specificity). We analyzed data of 153 patients with AE-COPD who were admitted to the ICU and had an in-hospital mortality rate of 8.05%. Compared with survivors, non-survivors were older, had higher lactate levels, more frequent invasive mechanical ventilation, and higher NPS scores. In multivariable models, NPS (odds ratio [OR], 4.851; 95% confidence interval [CI], 1.934–12.164; p=0.001), age (OR, 1.062; 95% CI, 1.017–1.109; p=0.007), and lactate levels (OR, 1.553; 95% CI, 1.117–2.103; p=0.008) independently predicted in-hospital mortality. NPS showed good discrimination (area under the curve [AUC] = 0.822; sensitivity = 84.1%; specificity = 77.1%). In patients with AE-COPD, higher NPS, older age, and elevated lactate levels independently predicted in-hospital mortality. Because NPS components are routinely available, incorporating this composite nutrition-inflammatory index alongside basic clinical variables may facilitate early risk stratification and guide monitoring and support in the ICU setting.

Keywords: Pulmonary disease, chronic obstructive, acute disease, intensive care units, prognosis, inflammation, serum albumin, cholesterol

Introduction

Acute exacerbations of COPD (AE-COPD) that necessitate intensive care are critical events that impose a considerable burden on both patients and healthcare providers, significantly influencing quality of life, prognosis, and survival outcomes. [1,2]. Over the years, many studies have sought to identify the main factors linked to mortality during COPD exacerbations. Several variables, including cardiac impairment, mechanical ventilation requirement, older age, comorbid conditions, serum albumin, baseline arterial blood gases, lactate levels, and nutritional indices, have been linked to mortality, although their impact differs between studies. [3–7]. In clinical practice, there is a growing need for simple indicators that capture the combined impact of systemic inflammation and nutritional status, enabling early risk stratification and individualized treatment plans.

The management of AE-COPD is complex and multifactorial.

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Successful outcomes depend not only on adequate respiratory support but also on the timely recognition of comorbidities and metabolic inflammatory disturbances that affect disease progression. In this retrospective analysis, we evaluated in-hospital mortality and determined its predictors among patients with AE-COPD admitted to the intensive care unit (ICU). We evaluated a wide range of demographic and clinical variables, including age, body mass index (BMI), smoking status, pulmonary function test results, arterial blood gas levels, and easily measurable inflammatory and biochemical markers, such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and total cholesterol level. Furthermore, the Naples Prognostic Score (NPS), an established composite index integrating nutritional and inflammatory information, was included to assess mortality trends and its ability to predict outcomes in critically ill patients with AE-COPD. Through these parameters, we aimed to describe the mortality patterns in this critically ill population and assess how effectively the NPS can predict clinical outcomes for AE-COPD patients requiring intensive care.

Material and Methods

This investigation was a single-center, retrospective cohort study conducted at a tertiary university hospital ICU, including consecutive patients aged ≥ 40 years who were admitted with a diagnosis of acute exacerbation of COPD (AE-COPD) during 2023-2024. The study was approved by the Ethics Committee of Kafkas University Faculty of Medicine (approval number: 80576357-050-99/436), and the requirement for informed consent was waived due to the use of de-identified routine care data. Patients were considered to have AE-COPD when their respiratory symptoms acutely worsened and required therapeutic escalation. [8]. Primary and secondary diagnoses were coded according to the ICD-10 classification system. We excluded patients admitted for conditions other than AE-COPD, those with a hospital stay <24 h, and those with readmissions within 1 month (only the first eligible admission was retained). Throughout the study period, the ICU management protocols for AE-COPD remained unchanged. Laboratory data collected within the first day of ICU admission were included in the analyses. Information was retrieved from the hospital information system. It included demographics (age, sex, body mass index, smoking status), comorbidities (respiratory failure, hypertension, coronary heart disease, diabetes), pulmonary function tests, and arterial blood gas (ABG) values. Laboratory analyses included hematologic and biochemical indices: red and white blood cell counts, NLR, LMR, lipid profile, hemoglobin, albumin, and creatinine. The primary outcome was all-cause mortality during hospitalization, and to ensure reproducibility, we specified operational definitions. Acidosis and hypercapnia were defined from admission arterial blood gas using standard thresholds routinely reported in the ICU. Radiographic consolidation on admission was extracted from the radiologist's report. The Naples Prognostic Score (NPS) was calculated from the same-day laboratory panel using albumin, total cholesterol, NLR, and LMR according to their conventional formulation; higher scores indicated a worse combined nutritional–inflammatory status. For patients with multiple eligible laboratory results on day 1, the earliest measurement was selected. When duplicate ICU stays occurred within 30 days, only the index admission was analyzed.

Missing data were handled by complete-case analysis for the primary models; variable-wise denominators are reported in the tables when applicable. Continuous variables were inspected for distributional assumptions. Summary statistics are expressed as mean ± SD or median (IQR), as appropriate, and categorical variables as counts (percentages). Between-group comparisons (survivors vs. non-survivors) were performed using the t-test or Mann–Whitney U test for continuous variables and χ^2 or Fisher's exact test for categorical variables. The association with in-hospital mortality was first explored using univariate models, followed by multivariable regression with stepwise selection. The model type (logistic or Cox regression) was aligned with the primary outcome definitions and time structure. Proportional hazards assumptions were verified when Cox models were applied, and multicollinearity was evaluated using the variance inflation factors. The discrimination of the NPS for in-hospital mortality was assessed using receiver operating characteristic (ROC) analysis with area under the curve (AUC) and 95% confidence intervals; the optimal cut-off was determined by the Youden index, with corresponding sensitivity and specificity. All analyses were performed using standard statistical software, and a two-sided p-value<0.05 was considered statistically significant.

Result

Between 2023 and 2024, 153 patients diagnosed with AE-COPD were admitted to the intensive care unit of our hospital. Nineteen patients (8.05%) died during hospitalization. Individuals were classified as survivors or non-survivors, and their baseline features are presented in Table 1. The cohort's mean age was 66±14 years, with a predominance of males. Body mass index and smoking history did not differ significantly between the groups. However, coronary heart disease and hypertension were more prevalent among non-survivors, whereas diabetes was more common in survivors. Pulmonary function was better in survivors, and radiographic consolidation rates were comparable between the groups.

Initial arterial blood gas analysis showed that acidosis was more frequent among survivors, whereas the frequency of hypercapnia was comparable between the groups. Serum lactate concentrations were significantly higher in the non-survivor group (p=0.016). During intensive care follow-up, invasive mechanical ventilation was required more frequently in patients who did not survive (p<0.001).

There was a significant difference in Naples Prognostic Score values between patients who survived and those who did not, with median scores of 2.65 and 3.79, respectively (p<0.001). The ROC curve analysis confirmed that the score effectively

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differentiated patients according to in-hospital survival outcomes. (AUC=0.822, p<0.001). Using the optimal cut-off, the sensitivity and specificity reached 84.1% and 77.1%, respectively.

In the univariate models, the Naples score, age, and lactate level were each associated with in-hospital mortality, with effect estimates of OR 5.131 (95% CI 2.004–13.139; p=0.001), OR 1.061 (95% CI 1.017–1.106; p=0.006), and OR 1.388 (95% CI 1.080–1.783; p=0.010), respectively. In the multivariate stepwise Cox regression, these variables remained independent predictors: Naples score, OR 4.851 (95% CI 1.934–12.164; p=0.001); age, OR 1.062 (95% CI 1.017–1.109; p=0.007); and lactate, OR 1.553 (95% CI 1.117–2.103; p=0.008) (Figure 1).

Table 1. The baseline demographic characteristics and the laboratory of the study population

Variables	MORTALITY						
	Total	n=153	Survivors	n=134	Non-survivors	n=19	p value
Age, years	66	±13.6	65	±13.5	72	±11.9	0.039
Female, n (%)	40	26.1%	37	27.6%	3	15.8%	0.274
BMI, kg/m2	26	3	26.2	±3	25	±2.6	0.055
Diabetes, n (%)	53	34.6%	48	35.8%	5	26.3%	0.417
Hypertension, n (%)	76	49.7%	66	49.3%	10	52.6%	0.784
CAD, n (%)	41	26.8%	35	26.1%	6	31.6%	0.616
Smoking status, n (%)	115	75.2%	101	75.4%	14	73.7%	0.874
Smoking exposure, years	34.4	±12.1	34.1	±11.8	36.5	±14.4	0.703
Creatinine, mg/dl	1.0	0.7-2.0	1.0	0.7-2.0	1.0	0.7-2.3	0.776
Albumin, g/L	3.5	±0.6	3.5	±0.6	3.5	±0.4	0.846
Hemoglobin, g/dL	15.7	±1.8	15.7	±1.8	15.4	±1.3	0.338
White blood cell count, 109/L	12.1	±4.6	12.1	±4.8	11.5	±3.7	0.672
Neutrophil count, 109/L	9.0	±4.3	9.1	±4.4	8.6	±3.0	0.881
Lymphocyte count, 109/L	1.8	1.2-2.5	1.8	1.1-2.6	1.8	1.6-2.2	0.851
Monocyte, 109/L	0.7	0.5-0.9	0.8	0.5-0.9	0.6	0.5-0.9	0.274
NLR	4.5	2.7-7.2	4.9	2.5-7.8	3.9	3.5-5.5	0.761
LMR	2.5	1.7-4.1	2.5	1.6-4.1	3.0	2.1-3.7	0.387
Total cholesterol, mg/dl	175.5	±39.1	178.6	±39.0	153.7	±32.7	0.016
LDL-C, mg/dl	112.5	±37.3	114.9	±37.1	95.5	±34.4	0.103
HDL-C, mg/dl	40.0	±12.4	40.2	±12.6	38.2	±11.4	0.417
Triglycerides, mg/dl	114	78-150	114	79-156	116	65-135	0.517
NAPLES score	2.8	±1.1	2.6	±1.1	3.8	±0.5	0.000
GOLD stage	3.0	2.0-3.0	3.0	2.0-3.0	3.0	2.0-3.0	0.851
FEV1, %	45.5	31.0-59.4	45.6	30.3-59.4	43.1	32.2-60.5	0.908
Consolidation, %	58	37.9%	52	38.8%	6	31.6%	0.545
Lactate, mmol/L	1.5	1.2-2.3	1.5	1.2-2.2	2.0	1.4-3.1	0.016
Acidemia, %	29	19.1%	26	19.5%	3	15.8%	0.697
Hypercapnia, %	55	35.9%	48	35.8%	7	36.8%	0.931
IMV, %	18	11.8%	10	7.5%	8	42.1%	0.000
ICU, stay/days	7.0	3.0-12.0	6.0	3.0-11.0	9.0	5.0-16.0	0.041

BMI: Body mass index, CAD: Coronary artery disease, NLR: Neutrophil/ lymphocyte ratio, LMR: Lymphocyte/ monocyte ratio, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, GOLD stage: Global initiative for chronic obstructive lung disease, FEV1: Forced expiratory volume, IMV: Invazif mechanical ventilation, ICU: Intensive care unit). CHD: conjestive heart dieases, DM: Diabetes mellites, HT: hypertension, CAD: Coronary Artery Disease, BSA: Body surface area, WBC: White blood cell, Hgb: Hemoglobin, Na: Sodium, K: Potassium, CPR: C-reactive protein, TSH: Thyroid stimulating hormone, PM: Pace-maker, ICD: Implantable cardioverter-defibrillator, CRT: Cardiac resynchronization therapy) IVM: Invazif mechannical ventilation

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Table 2. Univariate and multivariate analyses of risk factors for mortality in patients' with acute exacerbation of chronic obstructive pulmonary diseases

Variable	Univariate			Multivariate		
	HR	95% CI	p value	HR	%95 CI	p value
Age, years	1.061	1.017-1.106	0.006	1.062	1.017-1.109	0.007
Lactate	1.388	1.080-1.783	0.01	1.553	1.117-2.103	0.008
NAPLES	5.131	2.004-13.139	0.001	4.851	1.934-12.164	0.001

Non-survivors had higher Naples scores, higher lactate levels, and a greater need for invasive mechanical ventilation; in addition, this group was older than the survivors. No significant group differences were detected in body mass index, smoking habits, radiographic consolidation, or hypercapnia.

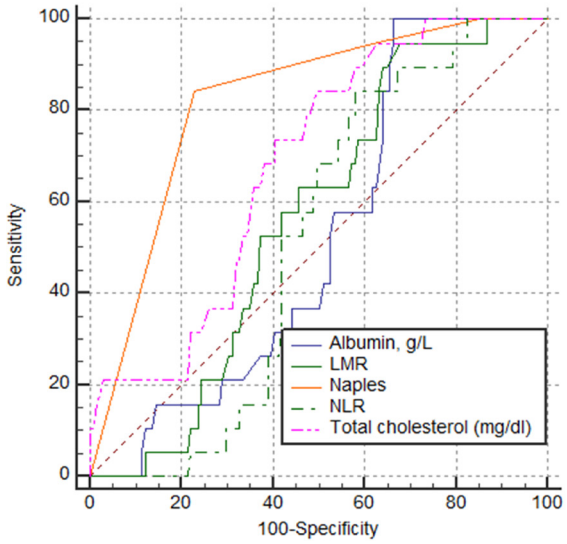

Figure 1. Receiver operating characteristic (ROC) curve analysis for Albumin, Total cholesterol, LMR, NLR, and Naples Score for all-cause mortality patients with AE-COPD (LMR: lymphocyte-to-monocyte ratio, NLR: neutrophil-to-lymphocyte ratio)

Discussion

The present analysis demonstrated that the Naples Prognostic Score, together with age and lactate concentration, independently predicted mortality in patients with AE-COPD requiring intensive care. The score achieved notable predictive accuracy, with robust sensitivity and specificity. The overall mortality rate of 8.05% observed in this study corresponds with previously reported figures, suggesting that our cohort reflects a typical ICU population with moderate disease severity.

Understanding the prognosis of AE-COPD and identifying factors that predict poor outcomes are crucial for physicians when counseling patients about expected disease trajectories and tailoring early management. Previous studies have found several markers—including the neutrophil-to-lymphocyte ratio, lymphocyte-to-monocyte ratio, requirement for invasive mechanical ventilation, age, lactate level, and cholesterol—to be associated with increased in-hospital mortality rates [10–17]. These variables are attractive because they are readily obtainable at the bedside and can be integrated into routine assessments without additional resource burden, thereby facilitating timely risk stratification in the ICU setting.

Chronic obstructive pulmonary disease involves both airway and systemic inflammation, which is markedly intensified during acute exacerbations [18]. This inflammatory surge alters immune cell ratios, such as the NLR and LMR, reflecting systemic immune activation. Several investigations have linked elevated NLR and decreased LMR with higher mortality and more severe disease courses [11,19–21]. Combining these hematologic markers with clinical parameters allows for a more objective assessment of short-term prognosis. Although NLR and LMR were not individually associated with mortality in our cohort, their integration within the Naples Prognostic Score offers a more comprehensive representation of the inflammatory–nutritional burden. Prior studies have similarly shown that these ratios may lose their independent predictive power in heterogeneous ICU populations. In contrast, composite indices, such as the NPS, magnify their prognostic relevance by incorporating albumin and cholesterol alongside immune cell ratios [31,32]. Therefore, the significant association observed between the NPS and mortality in our study likely reflects the synergistic contribution of these parameters rather than the isolated effects of NLR or LMR.

The relationship between albumin levels and outcomes in AE-COPD is complex and influenced by multiple factors. In addition to serving as a marker of systemic inflammation, decreased albumin levels also indicate underlying malnutrition and concurrent health issues. Moreover, as part of the acute-phase protein response, low serum albumin levels may indicate clinical deterioration or sustained inflammation during AE-COPD episodes [22,23]. Through its antioxidative and anti-inflammatory effects, albumin may modulate systemic inflammation and, consequently, affect the progression of COPD exacerbations. Thus, integrating albumin monitoring with traditional inflammatory markers can provide clinicians with additional insights into disease severity and prognosis [24,25], especially when decisions regarding monitoring intensity or escalation of care need to be made quickly.

Alterations in lipid metabolism may contribute to the pathophysiology of COPD by influencing airway inflammation and immune regulation [26]. Although the relationship between

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lipid levels and outcomes remains debated, population-based studies have reported that lower LDL cholesterol levels are associated with higher rates of severe exacerbations and mortality [17,27–29]. Evaluating lipid profiles during patient follow-up could help identify individuals at greater risk and tailor preventive or therapeutic strategies.

The NPS, first described by Galizia et al. in colorectal cancer research, combines four accessible laboratory indicators—albumin, cholesterol, NLR, and LMR—to reflect the overall nutritional and inflammatory balance [30]. More recently, COPD-focused cohorts have shown that a higher NPS is associated with adverse outcomes, supporting its prognostic utility beyond oncology [30–32]. In our study, we found that the Naples score, a well-established and validated prognostic tool for predicting mortality in AE-COPD, was a robust predictor of mortality. In both univariate and multivariate analyses, we observed that the Naples score, age, and lactate levels independently contributed to predicting mortality. This suggests that the Naples score, along with age and lactate levels, serves as a valuable prognostic indicator for mortality risk assessment in patients with AE-COPD (Figure 1). Furthermore, our analysis of the ROC curve reaffirmed the utility of the Naples score in mortality prediction, demonstrating its high specificity and sensitivity. Collectively, our findings suggest that integrating nutritional and inflammatory indicators with routine clinical parameters can help make earlier, more informed decisions about monitoring and supportive management.

This study has several limitations. Although the overall findings are consistent with previous literature, the relatively small number of deaths limits the statistical power of our analysis. The retrospective, single-center design limits generalizability, and pre-admission nutritional and functional data were unavailable, potentially affecting the interpretation of the NPS. Treatment approaches for respiratory failure vary among clinicians, possibly introducing bias. Additionally, unmeasured confounding factors, such as nutritional support during ICU stay, corticosteroid or statin use, and concomitant infections or sepsis, may have influenced inflammatory and biochemical parameters, potentially affecting the Naples Prognostic Score and mortality outcomes. However, these variables were not consistently available and could not be fully controlled in our analysis. Moreover, post-discharge outcomes were not captured, preventing the assessment of long-term survival. Future multicenter prospective studies are needed to confirm these associations and enhance their external validity.

Conclusion

This study identified the Naples Prognostic Score, age, and lactate level as independent determinants of in-hospital mortality among ICU patients with acute exacerbations of COPD. These results emphasize the usefulness of combining routinely available laboratory indicators for early prognostic evaluation and patient stratification. Implementing NPS within clinical workflows may help guide timely interventions and optimize care in critical settings, such as the ICU. Further multicenter prospective investigations are warranted to validate these findings and establish standardized applications of the NPS in respiratory intensive care.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
The study was approved by the Ethics Committee of Kafkas University Faculty of Medicine (approval number: 80576357-050-99/436)

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