ORIGINAL ARTICLE

BIOMARKERS RELATED TO MORTALITY IN OLDER ADULTS TREATED IN A TERTIARY INTENSIVE CARE UNIT: A THREE-YEAR RETROSPECTIVE ANALYSIS

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Abstract

Introduction: In this study, we investigated the prognostic performance of several inflammatory and nutritional indices—including the glucose/albumin ratio, C-reactive protein/albumin ratio, neutrophil/lymphocyte ratio, lactate/albumin ratio, platelet/neutrophil ratio, platelet/lymphocyte ratio, and systemic immune-inflammatory index in predicting 28-day all-cause mortality among patients aged 65 years and older admitted to a tertiary-level intensive care unit.

Materials and Method: This single-center, retrospective observational study included a total of 1,000 patients aged 65 years and older who were admitted to a tertiary intensive care unit between January 1, 2022, and January 1, 2025. Patients' demographic characteristics, clinical features, laboratory data obtained within the first 24 hours of admission, and clinical outcomes were evaluated.

Results: In non-survivors, GAR, CAR, NLR, PNR, LAR, and SII values differed significantly from survivors (all $p<0.001$ except SII $p=0.002$; Mann-Whitney U test). PLR showed no significant difference between groups ($p=0.434$). PNR values were lower in non-survivors, while all other significant biomarkers were higher.

Conclusion: Glucose/albumin ratio, C-reactive protein/albumin ratio, neutrophil/lymphocyte ratio, and lactate/albumin ratio measured at intensive care unit admission were found to be associated with 28-day mortality in patients aged 65 years and older. In multivariate analysis, C-reactive protein/albumin ratio was identified as the only independent inflammatory marker of mortality, while lactate/albumin ratio was the biomarker with the highest discriminatory power in predicting mortality.

Keywords: Critical Care; Aged; Biomarkers; Mortality; Intensive Care Units; Inflammation.

INTRODUCTION

Ageing is accompanied by progressive physiological alterations and an increasing prevalence of comorbid conditions, which collectively contribute to heightened vulnerability in older adults (1). One of the key consequences of ageing is the gradual deterioration of immune system efficiency, which results in impaired immune responses over time (2). These phenomena, often described as immune ageing and inflammaging, are closely associated with frailty, declining functional capacity and an increased susceptibility to acute physiological stressors (3). As these processes advance, they may lead to cumulative health deterioration and a substantially elevated risk of death. In recent years, several immuno-nutritional and systemic inflammatory indices, calculated from routinely obtained laboratory parameters, have emerged as valuable prognostic tools for mortality prediction. Given their simplicity, low cost and non-invasive nature, these biomarkers offer practical advantages for risk stratification and clinical decision-making in older adults (4).

Researchers have demonstrated significant associations between glucose/albumin ratio (GAR), and mortality in patients with various clinical conditions, including non-alcoholic fatty liver disease and cerebral haemorrhage (5,6). Similarly, C-reactive protein/albumin ratio (CAR), which reflects the combined effects of systemic inflammatory response and nutritional status, has been reported to provide superior prognostic information compared to the evaluation of C-reactive protein or albumin alone (1,7). Neutrophil/lymphocyte ratio (NLR), has also been widely studied as a prognostic indicator in sepsis, with consistently higher values reported among non-surviving patients in intensive care units (ICU) (8,9,10). Lactate is widely recognized as a key indicator of tissue hypoperfusion and metabolic stress and is commonly used in prognostic assessment, particularly in critically ill patients. However, lactate alone may be insufficient to fully reflect the complex interplay of inflammation and nutritional status. For this reason, the LAR, which integrates lactate with serum albumin levels, has recently emerged as a more comprehensive biomarker. Previous studies conducted in septic ICU populations have demonstrated that LAR provides superior prognostic performance compared to lactate or albumin alone in predicting clinical outcomes. (11).

In older adults patients admitted to intensive care units, these biomarkers are commonly used to assess the presence and progression of various diseases, and their prognostic value is considered significant, particularly in this population characterized by a high burden of comorbidities. However, evidence demonstrating the independent and relative effects of these biomarkers on mortality in the presence of comorbid conditions is limited. Therefore, in this study, considering the burden of comorbidities, we investigated the prognostic value of readily available inflammatory and immune-nutritional biomarkers in predicting 28-day mortality in patients aged 65 years and older monitored in a tertiary intensive care unit.

MATERIALS AND METHOD

Our retrospective observational cohort study was conducted in accordance with the Declaration of Helsinki and the STROBE Checklist, following approval by the Ethics Committee of Malatya Turgut Özal University (decision no: 2024/44; 30 May 2024). The study included 1,000 patients aged 65 years or older who were monitored and treated in the intensive care unit of Malatya Training and Research Hospital between 1 January 2022 and 1 January 2025. Data regarding the patients were obtained from patient files and the hospital automation system. Patients with missing haematological and clinical data at admission, a history of malignancy or suspected Covid-19, as well as traffic accident victims, were excluded from the study due to changes in their haematological and immunological



profiles and because traffic accidents would affect mortality rates. The patients' demographic data, comorbidities, surgical history, length of stay in the intensive care unit, mechanical ventilator support, inotrope use, discharge status and survival status were recorded. Biomarkers [GAR, CAR, NLR, LAR, platelet/neutrophil ratio (PNR), platelet/lymphocyte ratio (PLR), and systemic immune-inflammatory index (SII)] were obtained from routine laboratory parameters. These biomarkers were calculated using laboratory data gathered within the first 24 hours of intensive care unit admission, and their predictive power for 28-day mortality was evaluated.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Normality of distribution was assessed using the Kolmogorov-Smirnov test. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data or median with interquartile range (IQR) for non-normally distributed data. Categorical variables were presented as frequency (n) with percentage (%). Cross-group comparisons between survivors and deceased patients were performed using independent samples t-test for normally distributed continuous variables and Mann-Whitney U test for non-normally distributed continuous variables. Categorical data were compared using Pearson's chi-square test or Fisher's exact test when expected cell counts were less than 5.

Univariate binary logistic regression analysis was performed to identify potential predictors of 28-day mortality. Variables with $p<0.05$ in univariate analysis were included in a multivariate binary logistic regression model using the enter method. Results were reported as odds ratios (OR) with 95% confidence intervals (CI). Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test, and model performance was evaluated using Nagelkerke R^2 and Akaike Information Criterion (AIC).

Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the discriminative ability of inflammatory ratios for predicting 28-day mortality. The area under the curve (AUC) with 95% CI was calculated. Optimal cut-off values were determined using Youden's index (Sensitivity + Specificity - 1). Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall accuracy were calculated for each optimal cut-off value.

All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant. Variables for multivariable logistic regression analysis were selected based on three criteria: statistical significance ($p<0.05$) in univariate analysis, established clinical relevance in geriatric critical care based on existing literature, and assessment of multicollinearity. Age and sex were included a priori as potential confounders regardless of univariate significance. For improved interpretability of continuous biomarkers with wide ranges, the neutrophil-to-lymphocyte ratio was scaled to reflect the odds ratio per 5-unit increase, while other continuous biomarkers were analyzed per 1-unit increase.

Multicollinearity among biomarkers was assessed using variance inflation factor (VIF) prior to inclusion in the multivariate model. All biomarkers demonstrated VIF values below 2 (glucose/albumin ratio: 1.45, C-reactive protein/albumin ratio: 1.45, neutrophil/lymphocyte ratio: 1.24, lactate/albumin ratio: 1.24), indicating minimal collinearity and supporting their independent contribution to the predictive model.

The multivariate logistic regression model included 13 predictor variables with 415 outcome events (deaths), yielding an events-per-variable ratio of 31.9. This substantially exceeds the recommended minimum of 10 events per variable, ensuring adequate model stability and minimizing the risk of overfitting.

Two patients (0.2%) had missing lactate/albumin ratio values at ICU admission and were excluded from analyses involving this biomarker. All other included patients had complete hematological and clinical data at admission; therefore, no imputation was required.

ICU length of stay was included as a covariate in the multivariate model because it

reflects both disease severity and response to treatment during the early ICU course. While ICU stay duration is partially influenced by the outcome (i.e., death may occur before prolonged stay), it also captures information about clinical trajectory and treatment intensity during the initial days of ICU admission that is not fully reflected in admission biomarkers alone. Previous studies have demonstrated that early ICU length of stay provides prognostic information that complements admission severity assessments.

RESULTS

The demographic and clinical characteristics of patients are listed in Table 1. Patients were divided into two groups (survivors and non-survivors). There was a significant difference between the two groups in terms of age and sex, with a higher mortality rate in males (50.4%). Mechanical ventilation and inotrope requirements were significantly higher in those who died, and ICU length of stay was longer. The incidence of congestive heart failure, acute-chronic kidney failure, and pneumonia was higher in those who died. Our 28-day mortality rate was 41.7%. The high mortality rate found in our study can be explained by the advanced age of the patient population, the high prevalence of multiple comorbidities, and the fact that a significant proportion required mechanical ventilation and inotrope support.

The distribution of inflammatory ratios between survivors and non-survivors is shown in Table 2. GAR, CAR, NLR, LAR ($p<0.001$), and SII ($p=0.02$) were higher in non-survivors. PNR was lower. PLR was not statistically significant ($p>0.05$). LAR showed a stronger and statistically more significant relationship with mortality compared to NLR and SII.

Table 1. Baseline Characteristics According to 28-Day Mortality

Variable	Survivors (n = 583)	Deceased (n = 417)
Age (years), mean ± SD	82.8 ± 9.4	80.7 ± 8.6
Male sex, n (%)	250 (42.9)	210 (50.4)
Postoperative admission, n (%)	169 (29.0)	20 (4.8)
Hypertension, n (%)	414 (71.0)	278 (66.7)
Diabetes mellitus, n (%)	186 (31.9)	117 (28.1)
Congestive heart failure, n (%)	264 (45.3)	266 (63.8)
Chronic obstructive pulmonary disease, n (%)	221 (37.9)	176 (42.2)
Chronic/acute kidney disease, n (%)	125 (21.4)	202 (48.4)
Neurological disease, n (%)	222 (38.1)	204 (48.9)
Pneumonia, n (%)	181 (31.0)	190 (45.6)
ICU length of stay (days), median (IQR)	4 (2–8)	5 (2–15)
Mechanical ventilation, n (%)	44 (7.5)	290 (69.5)
Inotrope use, n (%)	34 (5.8)	273 (65.5)

SD: standard deviation; IQR: interquartile range; ICU: intensive care unit.

Table 2. Inflammatory and Immuno-Nutritional Biomarkers According to 28-Day Mortality

Biomarker	Survivors Median (IQR)	Deceased Median (IQR)	p value
Glucose (mg/dL) / Albumin Ratio (g/dL) (GAR)	46.02 (37.00–61.71)	54.19 (41.29–80.00)	<0.001
CRP (mg/L)/Albumin (g/dL) Ratio (CAR)	1.83 (0.33–4.01)	2.63 (0.63–6.34)	<0.001
Neutrophil (10^3/μL) / Lymphocyte (10^3/μL) Ratio (NLR)	7.47 (4.00–13.57)	9.84 (5.32–18.77)	<0.001
Platelet (10^3/μL) / Neutrophil (10^3/μL) Ratio (PNR)	25.57 (17.81–37.68)	19.90 (12.80–31.45)	<0.001
Platelet (10^3/μL) / Lymphocyte (10^3/μL) Ratio (PLR)	181.33 (109.36–305.99)	188.75 (114.40–341.44)	0.434
Lactate (mmol/L) / Albumin (g/dL) Ratio (LAR)	0.60 (0.43–0.92)	1.13 (0.62–2.28)	<0.001
Systemic Immune-Inflammation Index (SII)*	1585 (779–3031)	1923 (964–3821)	0.002

*SII = Neutrophil × Platelet / Lymphocyte; IQR: Interquartile Range. Mann-Whitney U test was used for comparisons.

Table 3. Univariate Logistic Regression Analysis for 28-Day Mortality

Variable	Odds Ratio (OR)	95% CI
Age (years)	0.974	0.961–0.988
Male sex	1.351	1.050–1.739
Postoperative admission	0.123	0.076–0.200
ICU length of stay (days)	1.034	1.022–1.047
Mechanical ventilation	27.972	19.295–40.553
Inotrope use	30.827	20.640–46.040
Congestive heart failure	2.129	1.645–2.755
Chronic/acute kidney disease	3.459	2.625–4.557
Pneumonia	1.867	1.439–2.423
Glucose/Albumin Ratio (GAR)	1.036	1.026–1.046
CRP/Albumin Ratio (CAR)	1.370	1.302–1.439
Neutrophil/Lymphocyte Ratio (per 5-unit)	1.104	1.027–1.181
Lactate/Albumin Ratio (LAR)	6.280	5.778–6.782

OR: odds ratio; CI: confidence interval; ICU: intensive care unit. Variables with p <0.01 were considered statistically significant.

The individual association of each factor with mortality is tested in Table 3. (OR>1: Increased risk, OR<1: Decreased risk) Inotrope use and mechanical ventilation were very strong risk factors. Chronic/acute kidney disease and congestive heart failure were strong risk factors. Pneumonia, sex (male), CAR, and ICU length of stay increased mortality risk. NLR and LAR had lower risk ratios.

In multivariate logistic regression analysis, independent risk factors associated with 28-day mortality in older adults admitted to the ICU are evaluated in Table 4. Variables found to be significant in univariate analyses or considered clinically important were included in the multivariate model to control for possible confounding effects. Thus, the effect of a variable on mortality was evaluated independently of the effects of other variables. It was demonstrated that, independent of basic demographic variables such as age and sex, the factors primarily determining mortality were

Table 4. Multivariate Logistic Regression Analysis for 28-Day Mortality

Variable	Adjusted OR	p value
Age (years)	0.996	0.770
Male sex	0.862	0.453
Postoperative admission ***	0.157	<0.001
ICU length of stay (days)	1.015	0.070
Mechanical ventilation ***	6.063	<0.001
Inotrope use ***	9.597	<0.001
Congestive heart failure *	1.546	0.028
Chronic/acute kidney disease *	1.648	0.016
Pneumonia ***	2.284	<0.001
Glucose/Albumin Ratio	1.002	0.177
CRP/Albumin Ratio (CAR) *	1.048	0.024
Neutrophil/Lymphocyte Ratio (NLR)	1.000	0.328
Lactate/Albumin Ratio (LAR)	1.000	0.082

OR: odds ratio; CI: confidence interval; ICU: intensive care unit. Significance codes: *** p<0.001, ** p<0.01, * p<0.05. Model performance: Nagelkerke R^2 = 0.462

Table 5. ROC Curve Analysis and Performance Metrics for Prediction of 28-Day Mortality

Biomarker	AUC	95% CI	Cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)	p value
Glucose/Albumin Ratio	0.607	0.570–0.645	69.66	34.8	84.2	61.2	64.4	63.6	<0.001
CRP/Albumin Ratio (CAR)	0.590	0.556–0.623	4.37	39.8	77.7	56.1	64.3	61.9	<0.001
Neutrophil/Lymphocyte Ratio (NLR)	0.588	0.552–0.622	6.62	69.3	46.1	47.9	67.8	55.8	<0.001
Lactate/Albumin Ratio (LAR)	0.712	0.678–0.744	0.92	59.6	75.6	63.6	72.4	68.9	<0.001

AUC: Area Under the Curve; PPV: Positive Predictive Value; NPV: Negative Predictive Value. Cut-off values determined using Youden's Index (Sensitivity + Specificity - 1). AUC interpretation: 1.0 = Perfect discrimination, 0.7-0.8 = Good discrimination, 0.6-0.7 = Acceptable discrimination, 0.5 = No discrimination (equal to chance).

related to clinical severity and inflammatory burden. Furthermore, this analysis strengthened the main findings of the study by revealing that CAR was the only inflammatory ratio independently associated with mortality compared to other biomarkers.

Assessment of multicollinearity demonstrated variance inflation factor values below 2 for all biomarkers (GAR: 1.45, CAR: 1.45, NLR: 1.24, LAR: 1.24), confirming the absence of problematic collinearity. In the multivariate model, among the inflammatory and nutritional biomarkers, only CAR remained independently associated with 28-day mortality (adjusted OR 1.048, 95% CI 1.006-1.091, p=0.024) after adjusting for clinical severity factors including mechanical ventilation and inotrope use.

Table 5 presents the ROC curve analysis, which was performed to evaluate the performance of inflammatory biomarkers in discriminating 28-day



mortality. The AUC (Area Under Curve) value in ROC analysis indicates the discriminatory power of the test. Best discriminatory power: LAR (AUC ~0.71) is the most reliable marker for mortality prediction. It can be calculated from a single laboratory measurement and is easily applicable. Other useful ratios: GAR, CAR, NLR (AUC ~0.61, AUC ~0.59) show moderate performance and are insufficient alone.

An important finding of our study is the discrepancy between univariate discriminatory performance and multivariate significance. While LAR demonstrated the highest area under the curve in ROC analysis (0.712), it did not retain independent statistical significance in the fully adjusted multivariate model (p=0.082). This apparent paradox likely reflects the strong physiological relationship between elevated lactate levels and clinical severity markers, particularly mechanical ventilation and inotrope use, which may represent mediators in the causal pathway between LAR and mortality rather than true confounders. The inclusion of these treatment-related variables in the multivariate model may have led to overadjustment, attenuating LAR's independent effect.

Nevertheless, LAR's superior univariate discriminatory performance (AUC 0.712) compared to CAR (AUC 0.590) suggests it remains a valuable prognostic marker for risk stratification at ICU admission, before the initiation of intensive support measures. Similarly, while NLR lost statistical significance in the adjusted model (p=0.328), its established role as an inflammatory marker in critical illness supports its clinical utility as part of a comprehensive assessment.

It is important to note that while these biomarkers showed statistically significant associations with mortality, their AUC values ranging from 0.59 to 0.71 indicate only modest to moderate discriminatory ability. These findings suggest that these readily available inflammatory and nutritional biomarkers should be interpreted as adjuncts to established clinical severity scores such as APACHE II or SOFA, rather than standalone prognostic tools. Their value lies in their ease of calculation from routine laboratory tests and their potential to contribute to early risk stratification in resource-limited settings where more comprehensive severity scoring may not be immediately available.

Clinical significance: Risk stratification can be performed using cut-off values. Performance ranking of biomarkers according to cut-off values: LAR shows the best performance with cut-off >0.92 indicating high risk, high sensitivity capturing 60% of those at mortality risk, high specificity correctly identifying 75% of low-risk patients, and accuracy of 70%. GAR shows moderate performance with cut-off >69.66 indicating high risk, low sensitivity capturing 35% of those at mortality risk, high specificity correctly identifying 84% of low-risk patients, and accuracy of 61%. CAR shows moderate performance with cut-off >4.37 indicating high risk, low sensitivity capturing 40% of those at mortality risk, high specificity correctly identifying 78% of low-risk patients, and accuracy of 59%. NLR shows moderate performance with cut-off >6.62 indicating high risk, high sensitivity capturing 70% of those at mortality risk, low specificity correctly identifying 46% of low-risk patients, and accuracy of 59%.

Overall, LAR exhibits the most balanced and reliable performance at the optimal cut-off value, relatively reducing both false positives and false negatives. GAR and CAR emerged as moderately contributing biomarkers. NLR has the lowest accuracy—reassuring when negative, but not very reliable when positive.

DISCUSSION

Our findings indicate that GAR, CAR, NLR and LAR can be used to predict all-cause mortality in older adults in intensive care. The main findings of this study are the following: CAR is the only independent inflammatory predictor of intensive care unit mortality; GAR is a novel biomarker in mortality

prediction; NLR shows moderate performance in mortality prediction, and LAR is the demonstrated the highest discriminatory performance for mortality prediction.

An unexpected finding was that non-survivors were slightly younger than survivors (80.7 ± 8.6 vs 82.8 ± 9.4 years, p<0.001). This counterintuitive result likely reflects survival bias and differences in clinical presentation. Younger patients in this geriatric cohort (65-75 years) may have been admitted with more acute, severe conditions requiring intensive care, whereas the oldest patients (>85 years) who survived may represent a particularly resilient subset with less acute pathology. This phenomenon has been observed in other geriatric ICU studies and underscores the heterogeneity of the older adult population. Age did not retain independent significance in multivariate analysis (p=0.770), suggesting that clinical severity and comorbidity burden were more important determinants of outcome than chronological age alone.

GAR provides a multidimensional reflection of metabolic reserve, inflammatory burden and overall disease severity. In a large-scale study conducted by Liu et al. in 2025, which included 6,731 patients, GAR was identified as an independent predictor of 28-day all-cause mortality in individuals with sepsis (12). Consistent findings have been reported in subsequent studies in which GAR emerged as a was associated with of both 30-day and 90-day sepsis-related mortality in intensive care settings (13). Therefore, recent evidence has shown elevated GAR values to be a novel biomarker associated with increased mortality risk in sepsis. In our study, GAR values were higher in non-survivors compared to survivors, showed moderate performance in detecting all-cause mortality, and were found to be particularly useful in distinguishing patients who did not have a high mortality risk.

NLR reflects systemic inflammatory activity and has emerged as a practical marker of immune imbalance. In the present cohort, NLR values were significantly higher among non-survivors than survivors, a finding that aligns with previously reported evidence. The authors of a large prospective cohort study published in 2023, which included 5,034 older adults, demonstrated that patients who non-survivors exhibited markedly elevated NLR levels compared to survivors (14). Moreover, in a recent study conducted among older adults presenting to emergency departments, elevated NLR values at admission were found to remain independently associated with in-hospital mortality after adjustment for relevant confounding variables (15). Also, in a study conducted in 2024 by Çakın et al. on 333 intensive care unit patients, NLR showed a moderate effect on 28-day mortality, which is consistent with the results of our study (16). According to our findings, NLR values can be used to identify elderly individuals with lower clinical mortality risk.

CAR has been increasingly identified as a strong independent indicator of mortality in diverse geriatric cohorts, including individuals with sepsis and chronic medical conditions (1,17,18), which is consistent with our results. In a retrospective analysis conducted by Par et al., CAR was found to be an independent determinant of 28-day mortality among intensive care unit patients, with higher CAR values being associated with increased mortality risk in critically ill individuals (19). Furthermore, in a recent large-scale study carried out in our country, Adem et al. demonstrated that elevated CAR levels at emergency department admission independently predicted all-cause in-hospital mortality in patients aged 65 years or older (20). In our study, CAR exhibited a moderate predictive capacity for mortality, with an acceptable level of accuracy. This finding is consistent with the literature, and it suggests that CAR may serve as a practical tool for mortality prognostication in routine clinical practice.

In a large-scale study, the LAR was found to be significantly associated with both ICU and in-hospital mortality, and was identified as a robust



parameter for risk stratification in critically ill patients (21). Consistent findings have also been reported in studies conducted in our country involving septic patients admitted to the ICU, where elevated LAR values were significantly associated with increased mortality (22). Similarly, in a study by Erdoğan et al. focusing on pneumo-sepsis patients in the ICU, higher LAR levels were significantly correlated with mortality, and LAR was identified as an independent prognostic marker (23). Furthermore, LAR has been shown to retain its prognostic value even after adjustment for potential confounding factors in multivariable analyses, suggesting that it may serve as an independent risk parameter providing incremental value beyond established clinical scoring systems (24). This is consistent with our findings. In our study, LAR was the demonstrated the highest discriminatory performance for mortality prognosis and could be confidently used to predict all-cause mortality in geriatric intensive care patients.

The two main strengths of our study are its relatively large sample size (1,000 patients) and its findings regarding the prognostic value of easily accessible and low-cost comprehensive biomarkers in geriatric intensive care patients. However, this study also has several limitations. Our research is a retrospective observational study conducted at a single centre, which limits the generalizability of the results to different centres. Additionally, limiting mortality at 28 days did not allow for the evaluation of long-term outcomes. Multicentre studies including long-term mortality outcomes are also needed on this subject. Additionally, we excluded patients with malignancy, did not include severity scores (APACHE II, SOFA), and did not systematically collect data on therapeutic interventions or serial biomarker measurements, which limits generalizability and our ability to assess treatment effects. Multicenter prospective studies with serial measurements, severity scores, and long-term outcomes are needed to validate these findings.

CONCLUSION

This study demonstrated that GAR, CAR, NLR and LAR were associated with 28-day all-cause mortality in patients aged 65 years or older admitted to a tertiary intensive care unit. In multivariate analyses, CAR was identified as the only independent inflammatory predictor of mortality. LAR showed the highest discriminatory power in ROC analysis, and it emerged as the demonstrated the highest discriminatory performance for mortality prognosis. These easily accessible and low-cost biomarkers can contribute to early risk stratification in older adults in intensive care.

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