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# Systemic inflammatory biomarkers for predicting clinical deterioration in traumatic brain injury

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### *Abstract*

*Background:* Traumatic brain injury (TBI) is a major cause of morbidity and mortality worldwide. Identifying patients at risk of early clinical deterioration is essential for timely intervention. *Methods:* We retrospectively analyzed adult patients with mild to moderate TBI admitted to a tertiary care center. Three biomarkers were calculated from admission labs: red cell distribution width-to-platelet ratio (RPR), C-reactive protein-to-albumin ratio (CAR), and neutrophil-to-lymphocyte ratio × RPR (NLTRP). The primary outcome was deterioration within 5 days, defined as a ≥2-point decline in Glasgow Coma Scale or need for mechanical ventilation. The secondary outcome was 28-day mortality. Results: Of 378 patients, 67 (17.7%) deteriorated and 30 (10.3%) died within 28 days. RPR (AUC 0.614, p=0.003) and CAR (AUC 0.609, p=0.005) predicted early deterioration. RPR (AUC 0.719, p<0.001), CAR (AUC 0.650, p=0.006), and NLTRP (AUC 0.645, p=0.008) predicted mortality. In multivariate analysis, CAR independently predicted early deterioration (OR 1.715, 95% CI 1.310–2.246).
*Conclusion:* RPR, CAR, and NLTRP are inexpensive, routinely available markers that may assist in early risk stratification of mild to moderate TBI. Validation in prospective multicenter studies is warranted.

*Keywords:* Biomarkers, brain injuries, traumatic, emergency medicine, inflammation, C-reactive protein, prognosis

## INTRODUCTION

Traumatic brain injury (TBI) affects nearly 70 million people each year and remains a leading cause of morbidity and mortality worldwide.[1] Early recognition of patients at risk for clinical deterioration is crucial for guiding emergency department decisions, optimizing monitoring strategies, and improving outcomes.[2,3]

TBI prognosis is influenced not only by unmodifiable factors such as age and primary injury severity but also by secondary mechanisms including inflammation and oxidative stress.[2,4] Systemic inflammatory activation in both central and peripheral compartments contributes to blood–brain barrier disruption, microvascular dysfunction, and neuronal damage, ultimately worsening neurological outcomes.[2,5]

In recent years, several inflammatory biomarkers have been proposed to quantify these systemic responses. Among them, the C-reactive protein-to-albumin ratio (CAR) reflects hepatic acute-phase reactivity: during the inflammatory cascade, CRP, a positive acute-phase protein induced by interleukin-6 and interleukin-1β, rises rapidly, whereas albumin, a negative acute-phase reactant, decreases due to reduced hepatic synthesis.[6,7] The ratio amplifies these opposite shifts, providing a sensitive indicator of systemic inflammatory intensity. The red cell distribution width-to-platelet ratio (RPR), on the other hand, captures hematologic alterations associated with inflammation: elevated RDW reflects stress erythropoiesis, while decreased platelet counts indicate consumption and impaired thrombopoiesis.[3,8]

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By representing complementary dimensions of systemic inflammation (hepatic acute-phase and hematologic responses) CAR and RPR together may improve early prognostic assessment after injury. The neutrophil-to-lymphocyte ratio multiplied by RPR (NLTRP) further integrates immune cell dynamics with erythrocyte–platelet indices, potentially offering a broader view of systemic inflammatory burden.[3,9]

Although most patients with mild to moderate TBI recover without complications, a clinically relevant subset deteriorates unexpectedly. Current clinical and imaging findings are not always sufficient to identify these patients early, making triage and escalation of care difficult. While previous studies have examined inflammatory indices mainly in relation to mortality or long-term outcomes, evidence on their role in predicting early deterioration is limited. Our study therefore aimed to evaluate the prognostic value of CAR, RPR, and NLTRP for deterioration within five days of admission and for 28-day mortality. Demonstrating their utility may support the use of simple, inexpensive laboratory indices as adjuncts to early decision-making in emergency care.

## METHODS

### *Study design*

This retrospective cohort study was conducted at the Emergency Medicine Department of Health Sciences University Ankara Training and Research Hospital, a tertiary care center. The study was conducted in accordance with the principles of the Declaration of Helsinki (2024 revision) and approved by the Clinical Research Ethics Committee of Ankara Training and Research Hospital. The requirement for informed consent was waived due to the retrospective design of the study.

### *Study population*

*Inclusion criteria:* Patients aged 18 years and older who presented to the emergency department with head trauma between January 2019, and June 2024, diagnosed with mild to moderate TBI, and had positive brain computed tomography (CT) findings were included.

*Exclusion criteria:* Pregnancy; Presentation to the emergency department more than 24 hours after trauma; Severe extracranial injuries (Abbreviated Injury Scale > 3); Incomplete medical records; Evidence of intoxication; Known coagulopathy or regular warfarin use; Clinical deterioration prior to hospital admission (during the emergency department stay, defined as a ≥2-point drop in GCS score or need for mechanical ventilation)

### *Study parameters*

Patients were identified using ICD-10 (International Classification of Diseases, 10th Revision) diagnostic codes related to TBI in the hospital electronic medical record system. TBI severity was determined based on the Glasgow Coma Scale (GCS), and patients with a GCS score of 9 or higher were selected. Patients who met the inclusion and exclusion criteria were recorded in the data collection form.

Demographic characteristics, comorbidities, antiplatelet or anticoagulant use, trauma mechanisms, and GCS scores at presentation were documented. Initial brain CT scans were reviewed to classify pathological findings, and Rotterdam CT scores were calculated. Laboratory tests obtained at the time of emergency department admission (typically within the first few hours after arrival, and within 24 hours of trauma onset), including hemoglobin, neutrophil, lymphocyte, platelet, RDW (Red Cell Distribution Width), glucose, calcium, albumin, and CRP (C-Reactive Protein) levels, were recorded. The CAR, RPR, and NLTRP indices were calculated.

Patient records were reviewed to identify patients with clinical deterioration within the first five days of follow-up. Clinical deterioration and 28-day mortality outcomes were recorded.

### *Biomarkers calculation*

Three inflammatory indices were calculated using laboratory data obtained at the time of emergency department admission: CAR (C-reactive protein-to-albumin ratio) = CRP (mg/L) ÷ Albumin (g/L); RPR (red cell distribution width-to-platelet ratio) = RDW (%) ÷ Platelet count ($\times 10^9$/L); NLTRP (neutrophil-to-lymphocyte ratio × RPR) = (Neutrophil count ÷ Lymphocyte count) × RPR

### *Study outcomes*

The primary outcome was clinical deterioration within the first five days of hospitalization, defined as a ≥2-point decrease in GCS score or the need for mechanical ventilation.[6] The secondary outcome was 28-day mortality following trauma.

*Statistical analysis*

All statistical analyses were conducted using SPSS version 27 (IBM Corp., Armonk, NY, USA) and Jamovi version 2.5.7 software. Receiver operating characteristic (ROC) curves were generated and visualized using SPSS version 27. Descriptive statistics for categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using the Shapiro-Wilk test and histogram plots. Continuous variables were expressed as medians and interquartile ranges (IQR; 25th–75th percentiles). Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. For continuous variables, normally distributed data were compared using the Student's t-test, while homogeneity of variances was assessed with Levene's test. Non-normally distributed variables were compared using the Mann-Whitney U test.

Receiver operating characteristic (ROC) curve analysis was used to evaluate the diagnostic performance of inflammatory biomarkers in predicting clinical deterioration and 28-day mortality. The area under the curve (AUC) was reported as a measure of diagnostic accuracy. Cut-off values, sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and positive and negative likelihood ratios were calculated. Optimal cut-off points were determined using the Youden Index. Logistic regression analysis was performed to identify independent predictors of clinical deterioration and mortality, including variables with $p < 0.05$ in univariate analysis. Variables with high multicollinearity or low clinical relevance were excluded from the multivariate model. A two-tailed p-value of $<0.05$ was considered statistically significant.

*Data availability*

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## RESULTS

*Study population and characteristics*

A total of 588 patients with TBI were screened, and 378 met the inclusion criteria (Figure 1). Of these, 67 (17.7%) experienced clinical deterioration within 5 days and 30 (10.3%) died within 28 days. Baseline demographic, clinical, and radiological characteristics are summarized in Table 1. No missing laboratory data required imputation; all analyses were performed on complete cases.

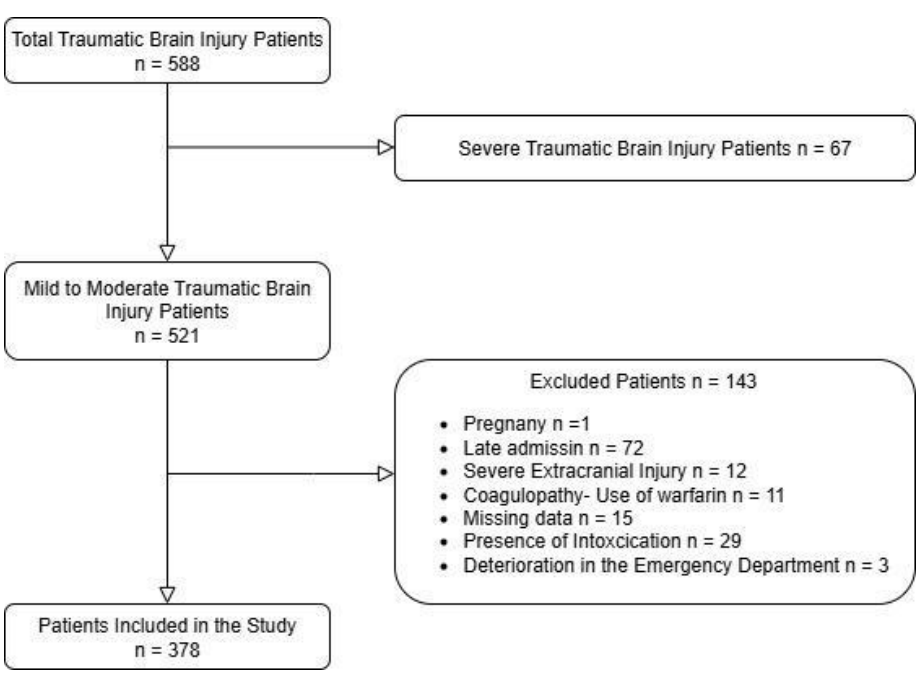


Figure 1. Patient flow diagram.

**Table 1: Baseline characteristics of the patients**

| Variable | Total (n = 378) |
|---|---|
| Age, years, median (IQR 25–75) | 53 (34-70) |
| Sex, n (%) | |
| Female | 78 (20.6) |
| Male | 300 (79.4) |
| Comorbidities, n (%) | |
| Coronary artery disease | 56 (14.8) |
| Hypertension | 90 (23.8) |
| Diabetes mellitus | 60 (15.9) |
| Degenerative CNS diseases | 21 (5.6) |
| Medication use, n (%) | |
| NOAC use | 5 (1.3) |
| Antiplatelet use | 56 (14.8) |
| GCS at admission, median (IQR 25–75) | 15 (14-15) |
| Mechanism of injury, n (%) | |
| Falls | 205 (54.2) |
| Traffic accidents | 94 (24.9) |
| Assault | 71 (18.8) |
| Other | 8 (2.1) |
| CT Findings, n (%) | |
| Subarachnoid hemorrhage | 221 (58.5) |
| Subdural hematoma | 157 (41.5) |
| Contusion | 94 (24.6) |
| Intraparenchymal hematoma | 64 (16.9) |
| Epidural hematoma | 63 (16.7) |
| Intraventricular hemorrhage | 11 (2.9) |
| Midline shift | 42 (11.1) |
| Hemorrhage volume | |
| < 25 cm3 | 330 (87.3) |
| ≥25 cm3 | 48 (12.7) |
| Rotterdam CT score, median (IQR 25–75) | 3 (2-3) |
| Laboratory Parameters, median (IQR 25-75) | |
| - Hemoglobin (g/dL) | 14.3 (12.6-15.5) |
| - Neutrophils (×10⁹/L) | 8.3 (5.7-11.9) |
| - Lymphocytes (×10⁹/L) | 1.77 (1.10-2.81) |
| - RDW (%) | 13.1 (12.5-13.8) |
| - Platelets (×10⁹/L) | 226 (182-269) |
| - Albumin (g/L) | 43 (40-46) |
| - Glucose (mg/dL): | 126 (108-155) |
| -CRP (mg/L) | 7.3 (1.9-30.5) |
| -INR | 1.08 (1.02-1.18) |
| -aPTT | 26.9 (24.7-29.6) |
| -PT | 14.2 (12.9-15.3) |
| Inflammatory Indices, median (IQR 25-75) | |
| RPR | 0.058 (0.048-0.076) |
| NLTRP | 0.289 (0.139-0.659) |
| CAR | 0.161 (0.041-0.739) |

aPTT: Activated Partial Thromboplastin Time, CAR: CRP-to-Albumin Ratio, CNS: Central Nervous System, CRP: C-Reactive Protein, CT: Computed Tomography, GCS: Glasgow Coma Scale, INR: International Normalized Ratio, NLTRP: Neutrophil-to-Lymphocyte Ratio × RPR, NOAC: Novel Oral Anticoagulant Drugs, PT: Prothrombin Time, RDW: Red Cell Distribution Width, RPR: RDW-to-Platelet Ratio

**Table 2: Association of inflammatory indices with clinical deterioration and 28-day mortality**

| Inflammatory Indices, median (IQR 25-75) | Clinical Deterioration | | | 28 Day Mortality | | |
|---|---|---|---|---|---|---|
| | No (n:311) | Yes (n:67) | p value | No (n=348) | Yes (n=30) | p value |
| RPR | 0.056 (0.047-0.073) | 0.064 (0.051-0.095) | **0.003** | 0.056 (0.047-0.073) | 0.081 (0.060-0.108) | **<0.001** |
| NLTRP | 0.279 (0.142-0.584) | 0.481 (0.131-0.879) | 0.066 | 0.281 (0.132-0.603) | 0.589 (0.201-1.21) | **0.008** |
| CAR | 0.155 (0.042-0.577) | 0.500 (0.057-1.46) | **0.005** | 0.157 (0.042-0.660) | 0.785 (0.090-2.08) | **0.006** |

CAR: CRP-to-Albumin Ratio, CRP: C-Reactive Protein, NLTRP: Neutrophil-to-Lymphocyte Ratio × RPR, RPR: RDW-to-Platelet Ratio
Mann Whitney U testi kullanılmıştır.

*Inflammatory indices and clinical deterioration*

Patients with deterioration had higher RPR (p=0.003) and CAR (p=0.005) values compared with those without deterioration, whereas NLTRP did not differ significantly (p=0.066) (Table 2). In ROC analysis, RPR (AUC 0.614) and CAR (AUC 0.609) showed modest predictive ability for deterioration (Figure 2). In multivariate analysis, CAR (OR 1.715, 95% CI 1.310–2.246), age, and intraventricular hemorrhage independently predicted early deterioration (Table 3).

*Inflammatory indices and 28-day mortality*

At 28-day follow-up, when patients with and without mortality were compared, statistically significant differences were observed in terms of RPR, NLTRP and CAR ($p < 0.001$; $p = 0.008$; $p = 0.006$, respectively) (Table 2). Among these, RPR showed the highest predictive performance for mortality (AUC 0.719, 95% CI 0.621–0.816) (Figure 2).

## DISCUSSION

Early identification of patients at risk of deterioration after TBI is crucial for triage decisions and intensive care admission. Although clinical and radiological findings guide management, additional tools are needed to improve early risk prediction. There is therefore growing interest in inexpensive and widely available biomarkers that may assist emergency physicians in early risk stratification.[3,9]

In this study, higher RPR and CAR values were associated with both early deterioration and 28-day mortality, while NLTRP was associated with mortality alone. Among these indices, CAR emerged as an independent predictor of deterioration in multivariate analysis. Although

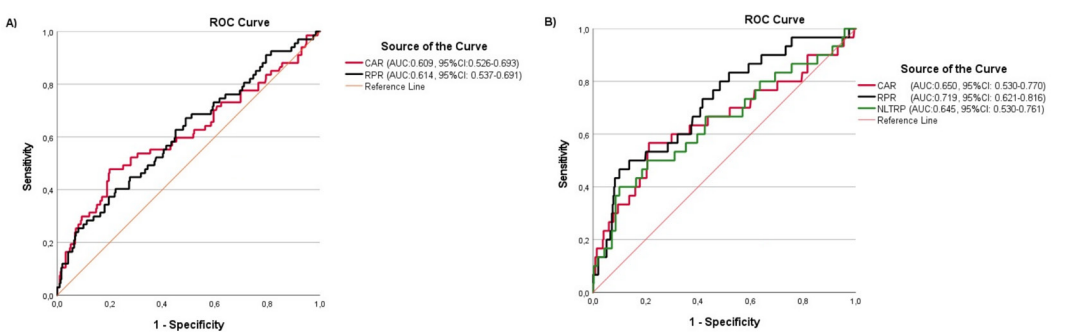


Figure 2. Receiver operating characteristic (ROC) curves of systemic inflammatory indices. AUC (95% CI) values are indicated for each biomarker.
(A) CAR and RPR for predicting early deterioration.
(B) RPR, CAR, and NLTRP for predicting 28-day mortality.

**Table 3: Logistic regression analysis of risk factors for predicting clinical deterioration in patients**

| | Univariate Analysis OR (%95 CI) | p value | Multivariate Analysis OR (%95 CI) | p value |
|---|---|---|---|---|
| **Age, year** | 1.036 (1.022-1.051) | <0.001 | **1.038 (1.022-1.054)** | **<0.001** |
| **Intraventricular Hematoma** | 8.954 (2.542-31.543) | <0.001 | **12.225 (3.114-47.997)** | **<0.001** |
| **Midline Shift** | 2.341 (1.144-4.790) | 0.020 | 2.170 (0.768-6.130) | 0.144 |
| **CAR** | 1.589 (1.254-2.015) | <0.001 | **1.715 (1.310-2.246)** | **<0.001** |
| **PT** | 1.110 (1.025-1.202) | 0.010 | 1.074 (0.986-1.169) | 0.101 |
| **INR** | 0.978 (0.891-1.073) | 0.640 | | |

CAR: CRP-to-Albumin Ratio, CI: Confidence Interval, CRP: C-Reactive Protein, INR: International Normalized Ratio, OR: Odds Ratio, PT: Prothrombin Time

prior studies have examined these markers in relation to in-hospital or long-term mortality, data on early clinical deterioration are limited. To our knowledge, this is the first study to demonstrate that CAR independently predicts early deterioration in mild to moderate TBI. By including a relatively large cohort and focusing on both early deterioration and short-term mortality, our findings strengthen the existing evidence and highlight the potential value of these indices in emergency care.

Our findings regarding RPR align with and extend previous research. RPR reflects the ratio of red cell distribution width (RDW) to platelet count, both of which are influenced by systemic inflammation. Elevated RDW indicates impaired erythropoiesis and increased release of immature reticulocytes, while reduced platelet counts often result from increased consumption and destruction during the inflammatory response.[3,8] Beyond TBI, RPR has been investigated in cardiovascular, metabolic, and cerebrovascular conditions, including ischemic stroke, where it demonstrated consistent prognostic utility.[10-12] Evidence specific to TBI, however, remains scarce. Earlier studies have reported significant associations between elevated RPR and in-hospital mortality, and one study also identified RPR as a predictor of early clinical deterioration in patients with mild TBI.[3,8,13] Notably, the present study is the first to demonstrate a significant relationship between RPR and 28-day mortality, extending its relevance beyond immediate outcomes. By simultaneously confirming the association of RPR with both early deterioration and 28-day mortality in a mild-to-moderate TBI cohort, our results complement previous studies and highlight the potential role of RPR as a simple, inexpensive, and widely accessible prognostic marker in emergency settings.

NLTRP, a composite marker integrating the neutrophil-to-lymphocyte ratio (NLR) and RPR, was recently introduced to enhance prognostic accuracy in TBI.[3] While our study did not demonstrate a significant association between NLTRP and early deterioration, higher values were observed among non-survivors at 28 days. This pattern may be explained by the time-dependent behavior of NLR, which has been shown in multiple studies to rise progressively after trauma and to peak days later, remaining elevated during the subacute phase.[4,14-16] Early NLR measurements (such as those obtained on admission) may therefore underestimate the overall inflammatory burden, reducing the discriminative value of NLTRP for early events but preserving its association with later outcomes.

Physiologically, neutrophil activation occurs rapidly and drives acute blood–brain barrier disruption and oxidative stress, whereas lymphocyte and erythropoietic responses evolve more slowly, reflecting sustained systemic inflammation.[4] Accordingly, NLTRP may better represent prolonged inflammatory stress that contributes to late complications or mortality rather than immediate neurological decline. Variations in the definition of clinical deterioration across studies may also explain differing results, as some have used broader or longer follow-up criteria, while our study defined deterioration strictly as a ≥2-point fall in GCS or need for mechanical ventilation within five days. Overall, our results emphasize that NLTRP's prognostic accuracy may be influenced by the timing of assessment and that prospective studies investigating its longitudinal kinetics are needed

to clarify its optimal application in TBI.

CAR combines C-reactive protein, an acute-phase reactant that rises rapidly after tissue injury and systemic inflammation, with albumin, a negative acute-phase protein that decreases under stress and catabolic conditions.[7] The CRP-to-Albumin Ratio has been investigated as a prognostic marker in various conditions, including sepsis, stroke, cancer, and cardiovascular diseases, and has been identified as a useful marker.[17-20] In 2020, Wang *et al*. were the first to report an independent association between elevated CAR levels and in-hospital mortality among TBI patients.[21] Subsequent studies have reinforced the prognostic value of CAR in this population.[22,23] However, some conflicting results exist; for example, Jung *et al.* did not observe a significant association between CAR and six-month mortality, although elevated CAR in conjunction with fever was linked to poorer long-term neurological outcome.[24] In our cohort, CAR values were significantly higher in patients with both clinical deterioration and mortality, and multivariate regression confirmed CAR as an independent predictor of early deterioration. To our knowledge, this is among the first studies to demonstrate that CAR independently predicts early deterioration in mild to moderate TBI. Given its routine availability, CAR represents a practical and feasible adjunct to clinical and imaging assessments in the early triage of TBI, particularly in centers where advanced monitoring is limited. By evaluating a relatively large and well-defined cohort and focusing on both early deterioration and short-term mortality, our study strengthens the current evidence base and highlights the potential contribution of CAR and related indices to emergency care decision-making.

From a clinical standpoint, incorporating simple inflammatory indices such as CAR and RPR into the initial assessment of patients with TBI may offer a rapid and cost-effective adjunct to existing decision-making processes. These routinely available parameters could assist clinicians in identifying patients who require closer monitoring, repeat imaging, or early transfer to higher-level care, particularly in resource-limited emergency settings. While such biomarkers are not intended to replace clinical judgment or neuroimaging, they may provide additional support for triage and follow-up decisions in the acute phase of TBI management.

Our study has several limitations. First, due to its retrospective design, data validation and longitudinal assessment of biomarker kinetics were not possible, which limits interpretation of their dynamic changes over time. Second, being a single-center study with a relatively small sample size restricts the generalizability of the findings. Third, variations in the time from trauma to emergency department admission resulted in differences in blood sampling time, which may have influenced biomarker levels. Future studies incorporating serial measurements and external validation in multi-center cohorts representing diverse populations are warranted to confirm these findings and establish standardized cut-off values for clinical use.

In conclusion, this study demonstrates that RPR and CAR are reliable predictors of both early clinical deterioration and 28-day mortality in patients with mild to moderate TBI, whereas NLTRP is associated only with mortality. The availability, simplicity, and low cost of these biomarkers make them attractive tools for early risk stratification in emergency settings. Incorporating such indices into clinical assessment may facilitate timely interventions and optimize resource allocation. Nevertheless, validation through prospective, multicenter studies is required before these biomarkers can be routinely integrated into clinical practice.

## DISCLOSURE

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Conflict of interest: None

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