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Neutrophil-albumin ratio serves as a superior prognostic biomarker for traumatic brain injury

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Traumatic brain injury (TBI) represents a common and severe medical condition necessitating prompt risk stratification to enhance patient outcomes. Although substantial research has been conducted on the prognostic utility of various biomarkers for TBI, no single biomarker has been definitively recognized as the most precise predictor of disease outcomes. In comparison to other markers, the neutrophil-albumin ratio (NAR) has emerged as a cost-effective and reproducible inflammatory biomarker, demonstrating potential in evaluating the severity of inflammation and prognosticating outcomes in infections and cerebrovascular diseases. This study evaluated the prognostic significance of the NAR in comparison to two other readily accessible and cost-effective composite indices: the Neutrophil-Lymphocyte Ratio (NLR) and the Platelet-Lymphocyte Ratio (PLR) in individuals with TBI. We conducted a retrospective cohort analysis involving 297 hospitalized TBI patients, gathering comprehensive demographic, anthropometric, medical, clinical, laboratory, and imaging data to assess the expression changes of these biomarkers. Our findings suggest that both the NAR and the NLR possess predictive value regarding prognosis following TBI. However, receiver operating characteristic (ROC) curve analysis revealed that NAR outperformed NLR as a prognostic predictor. In conclusion, our examination of blood biochemistry composite indicators indicates that, while both NAR and NLR serve as significant prognostic markers, NAR is a more effective predictor of outcomes in patients with TBI.

Keywords Neutrophil-lymphocyte ratio, Neutrophil-albumin ratio, Traumatic brain injury, Predict, Prognosis

Traumatic brain injury (TBI) constitutes a significant public health issue, profoundly affecting patients' quality of life and imposing substantial economic burdens on society[1]. Annually, more than half a million individuals globally experience TBI, with nearly half of the world's population likely to endure one or more TBIs over their lifetime[1]. Despite the introduction of novel therapeutic strategies aimed at preventing secondary brain injury, mortality rates associated with TBI, particularly severe traumatic brain injury (STBI), remain elevated[2]. Consequently, early risk stratification of TBI patients is imperative for accurate assessment and management of their condition. Recent investigations have examined the prognostic significance of biomarkers present in serum or cerebrospinal fluid, such as glial fibrillary acidic protein (GFAP), S100 calcium-binding protein B (S100B), neurofilament light chain (NFL), ubiquitin carboxy-terminal hydrolase L1 (UCHL1), and tau protein[1]. Biomarkers originating from the central nervous system have the potential to provide more direct insights into brain injury. Nevertheless, their application in routine clinical practice is constrained by factors including cost, availability, and the time required for analysis. Conversely, non-specific biomarkers can indicate the body's inflammatory status and immune response, which are critical in the pathophysiology of TBI. For example, composite markers such as the neutrophil-lymphocyte ratio (NLR) and platelet-lymphocyte ratio (PLR) have shown prognostic value in TBI[4,5]. NLR, as a readily accessible indicator, holds significant value in evaluating systemic inflammatory status and infection risk, as well as in predicting the prognosis of community-acquired pneumonia and cerebral hemorrhagic stroke[6–8]. Furthermore, PLR, as an indicator of platelet aggregation and

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systemic inflammation, has emerged as a clinically useful tool for assessing inflammatory coagulation, severe coagulopathy, and platelet activation induced by systemic inflammation[9]. Recent research has underscored the Neutrophil-Albumin Ratio (NAR) as a significant and cost-effective biomarker for assessing the severity of inflammation and prognosticating outcomes in a range of diseases, including infectious and cardiovascular conditions[10,11]. In trauma-induced conditions, such as acute infections and severe tissue damage, elevated levels of neutrophils can lead to vascular dysfunction, disruption of the blood-brain barrier, and influence clinical prognosis[12]. Neutrophils secrete cytokines, including tumor necrosis factor (TNF), which contribute to cellular damage. Concurrently, albumin, a critical plasma protein, plays a vital role in maintaining colloidal osmotic balance, preserving microvascular integrity, and modulating inflammatory pathways. Hypoalbuminemia following TBI compromises anti-inflammatory mechanisms, thereby exacerbating cerebral edema[13]. In this context, we undertook a retrospective cohort study to examine three extensively studied, readily available, and cost-efficient composite biomarkers—namely, NAR, PLR, and NLR—in patients with TBI. The objective was to ascertain the most efficacious indicator. It is anticipated that the identification of an optimal biomarker will augment existing clinical and radiological assessments in TBI cases, thereby facilitating a more comprehensive evaluation of patient status and prognosis.

Methods

Patients

This retrospective study analyzed 330 patients admitted to the intensive care unit and neurosurgery department of West China Hospital, Sichuan University, from April 2017 to March 2020. These patients, admitted within 8 h of injury, met the criteria for TBI, with routine blood samples collected and tested within 24 h. Exclusion criteria included patients under 18 years ($n=6$), admission over 8 h post-injury ($n=10$), previous head injury ($n=1$), pre-admission immunomodulatory treatment ($n=7$), conditions like arteriovenous malformations, intracerebral hemorrhage, recent cardiovascular or cerebrovascular disease, autoimmune diseases, and stroke ($n=3$), severe compound injuries, severe organ damage ($n=2$), and incomplete laboratory results ($n=4$). Ultimately, 297 patients were included, with TBI diagnosis confirmed via computed tomography (CT) and magnetic resonance imaging (MRI). The study was approved by the Ethics Committee of West China Hospital, Sichuan University.

Data collection

We collected data on demographic characteristics, anthropometric characteristics, medical history data, clinical characteristics, laboratory findings and imaging information, including Glasgow Coma Scale (GCS, with lower scores indicating poorer level of consciousness) scores, length of intensive care unit (ICU) stay, in-hospital mortality and 90-day Glasgow Outcome Scale (GOS) scores. EDH (epidural hemorrhage), SDH (subdural hemorrhage), SAH (subarachnoid hemorrhage) and contusions were also recorded and assessed as potential predictors of poor prognosis. We calculated NLR, PLR and NAR according to the following formulae: NLR = neutrophil count/lymphocyte count; PLR = platelet count/lymphocyte count; NAR = neutrophil count/albumin count. Patients were followed up by telephone 3 months after discharge. Neurological function was assessed 3 months after TBI using the GOS. Patients were divided into a poor prognosis group with low GOS (≤ 3) and a good prognosis group with a high GOS score (>3).

Data analysis

Statistical analysis was performed using GraphPad Prism 9.0.0(121) and R version 4.2.0. Continuous variables that followed a normal distribution were expressed as means with standard deviations, and the remaining continuous variables were expressed as medians with interquartile ranges (IQR) and compared using non-parametric rank-sum tests or independent samples t-tests as appropriate. Categorical variables are expressed as frequencies and percentages and compared using chi-squared or Fisher's exact tests where appropriate. Plot the receiver operating characteristic (ROC) curve of the predictor and evaluate the predicted value using the area under the curve (AUC). The correlation between NLR, PLR, NAR was assessed by Spearman correlation. Independent predictors of TBI were determined using univariate and multivariate logistic regression; Odds ratios (OR) and 95% confidence intervals (CI) were calculated. All P values are on both sides and the significance is set to $P<0.05$.

Results

General information

A total of 330 patients were initially recruited for the study, and, following the application of exclusion criteria, 297 patients were ultimately included in the final analysis. The average age of patients was 41.28 years (ranging from 18 to 91 years), with 223 males and 74 females. The median GCS score for all patients was 5, and a total of 187 patients survived their hospitalization. Given that both the NLR and PLR include lymphocytes, and both NLR and NAR encompass neutrophils, we conducted a correlation analysis. The resulting correlation coefficients were as follows: NLR and PLR = 0.42, NLR and NAR = 0.45, and PLR and NAR = -0.045 (Fig. 1A). These values suggest that the correlations among the three ratios were not strong, thereby not affecting the subsequent multivariate analysis. Furthermore, we also examined the correlation between NLR, PLR, NAR and prognosis. The admission NAR values of TBI patients may be associated with prognosis (Fig. 1B). To predict the prognosis in TBI patients, ROC curve analysis was conducted. The results indicate that NAR demonstrates significantly enhanced predictive performance, with an AUC of 0.803, when compared to NLR and cholesterol, which exhibit AUC values of 0.708 and 0.610, respectively (Fig. 2). In addition, we analyzed the distribution of the three inflammatory composite indicators in the different groups. The mean values of NLR and NAR in the

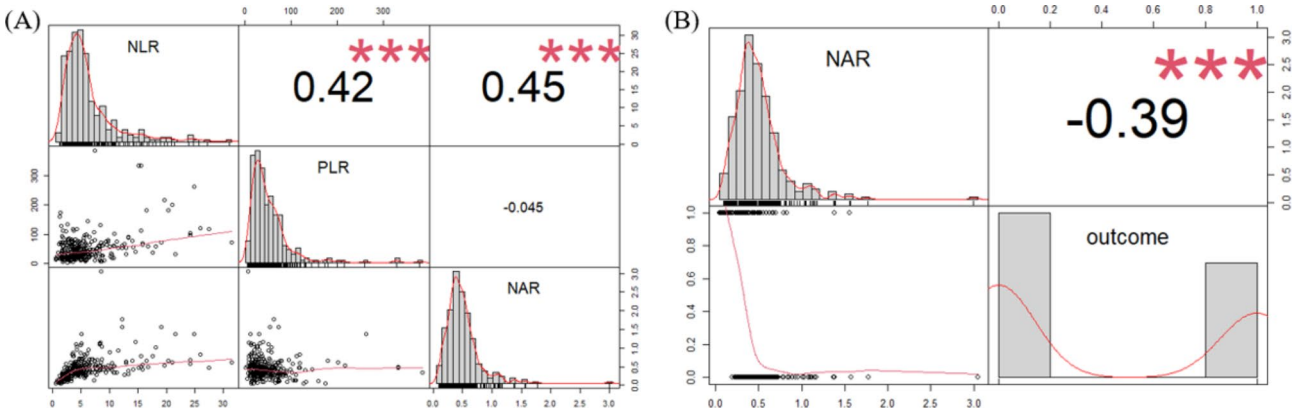

Fig. 1. The schematic diagram of correlation coefficient. (**A**): The correlation coefficient among the NLR, PLR and NAR; (**B**): The correlation coefficient between the NAR and outcome. NAR, neutrophil-albumin ratio; NLR, neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio.

non-survival group were higher than those in the survival group (Fig. 3A, B), but the mean values of PLR were lower than those in the survival group (Fig. 3C). PLR, NLR and NAR data were relatively discrete in the survival group, whereas the data in the non-survival group were relatively concentrated (Fig. 3A-C). The mean NLR and NAR were higher in the poor prognosis group than in the good prognosis group (Fig. 3D, E). The PLR, NLR and NAR data were relatively discrete in the poor prognosis group, whereas the data were relatively concentrated in the good prognosis group (Fig. 3D-F).

Baseline information of surviving versus non-surviving group

We assessed the baseline characteristics of the entire cohort and analyzed group characteristics based on survival outcomes (Table 1). Compared to patients in the survivor group, patients in the non-survivor group had significantly lower admission GCS scores ($P<0.001$), significantly higher baseline neutrophils ($P<0.001$), LDH ($P<0.001$) and blood glucose ($P<0.001$). Platelets ($P<0.$ 001), erythrocytes ($P=0.006$), lymphocytes ($P<0.001$), cholesterol ($P<0.001$), albumin ($P<0.001$) and respiratory rate ($P<0.001$) were significantly lower, with no significant differences in leukocytes, systolic blood pressure, diastolic blood pressure, body temperature or heart rate. In addition, patients in the non-survival group had significantly higher NLR ($P<0.001$) and NAR ($P<0.001$), with no significant difference in PLR.

Analysis of predictors of death in patients with TBI

Variables with p-values below 0.1 from the univariate analyses, along with gender and age, were incorporated into the multivariate logistic regression model. The results showed that respiratory rate (OR = 0.940, 95% CI, 0.889–0.994, $P=0.031$), admission GCS score (OR = 0.722, 95% CI, 0.604–0.863, $P<0.001$), blood glucose (OR = 1.125, 95% CI, 1.049–1.206, $P=0.001$), cholesterol (OR = 0.592, 95% CI, 0.451–0.777, $P<0.001$), NLR (OR 1. 116, 95% CI, 1.025–1.215, $P=0.011$) and NAR (OR = 6.208, 95% CI, 1.424–27.058, $P=0.015$), were independent risk factors for death in TBI patients (Table 2).

Baseline information of good prognosis versus poor prognosis group

As previously mentioned, patients were categorized into two groups—those with a poor prognosis and those with a good prognosis—based on their GOS scores obtained during the follow-up visit at three months (Table 3). Compared to patients with a good prognosis, those with a poor prognosis had a significantly higher rate of secondary bleeding ($P<0.001$), higher blood glucose concentration ($P=0.002$) and higher baseline neutrophil count ($P<0.001$), In contrast, patients with a poor prognosis had a significantly lower GCS score on admission ($P<0.001$), cholesterol level ($P=0.$ 002) and albumin levels ($P<0.001$). However, there were no significant differences in platelets, leukocytes, erythrocyte, systolic blood pressure, diastolic blood pressure, respiratory rate, PLR, body temperature and heart rate in the poor prognosis group compared to the good prognosis group. In addition, NLR ($P<0.001$) and NAR ($P<0.001$) were significantly higher in the poor prognosis group than in the good prognosis group. SAH and contusions occurred at a higher rate in the poor prognosis group.

Analysis of predictors of poor prognosis in patients with TBI

Similarly, In the multivariate logistic regression model, we incorporated variables that exhibited p-values less than 0.1 in the univariate analyses, in addition to including gender and age. We found that NAR (OR = 116.588, 95% CI, 17.847–761.609, $P<0.$ 001), hemorrhage (OR = 2.946, 95% CI, 1.560–5.260, $P<0.001$) and admission GCS score (OR 0.855, 95% CI, 0.757–0.966, $P=0.012$) were independent predictors of poor prognosis (Table 4).

Discussion

In this study, we explored the prognostic value of blood biochemical markers in critically ill TBI patients, with a particular focus on the significance of the NAR as an outstanding biomarker. The findings revealed a significant correlation between NAR and patient prognosis. Compared to the NLR and PLR, NAR demonstrated superior

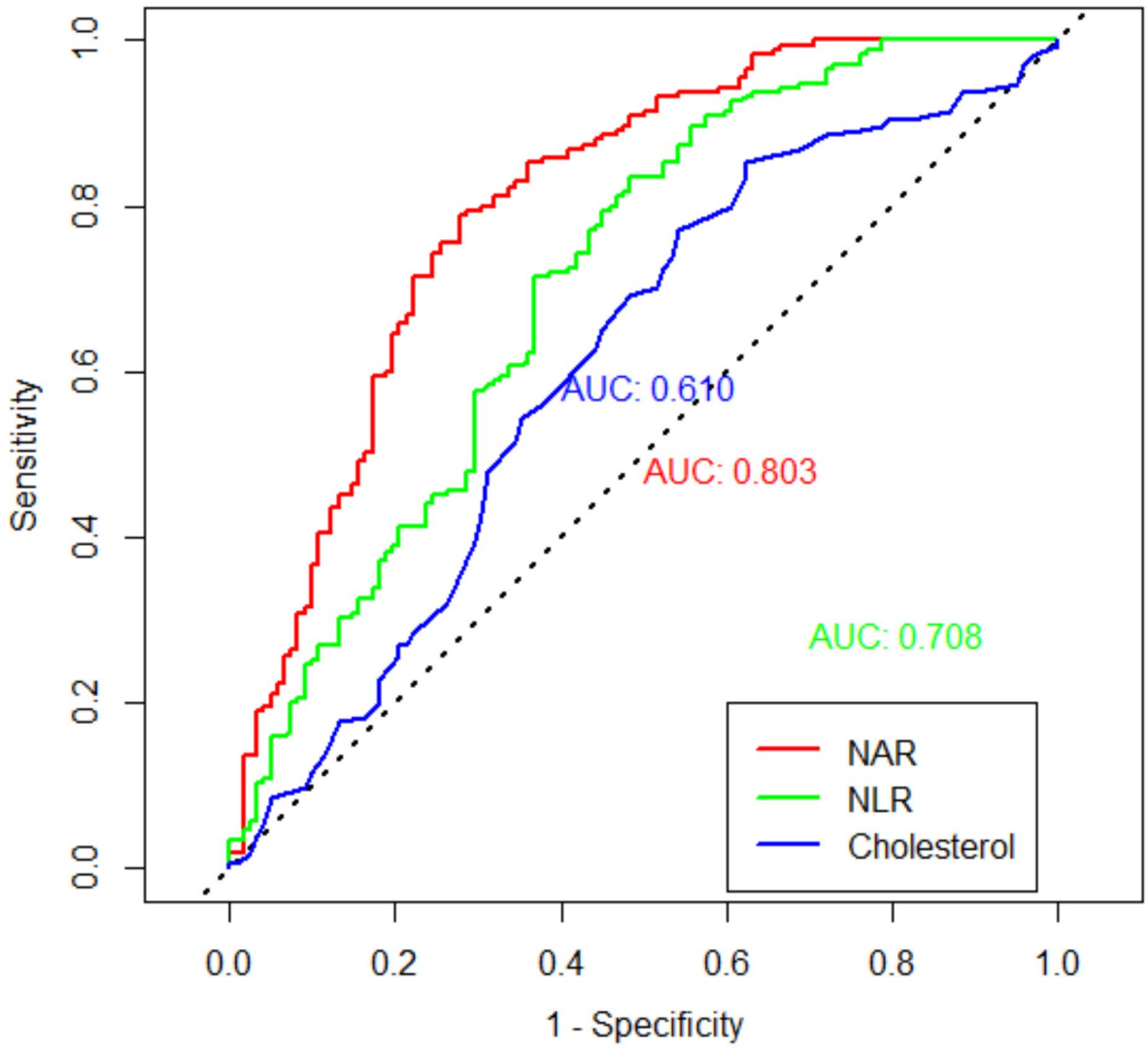

Fig. 2. ROC curve of NAR, NLR and serum cholesterol concentration at TBI patient admission. NAR, neutrophil-albumin ratio; NLR, neutrophil-lymphocyte ratio; AUC: area under curve.

performance in prognostic prediction, as evidenced by more favorable ROC curve analysis results. NAR was significantly elevated in both the non-survivors and poor prognosis groups. Furthermore, multivariate logistic regression analysis confirmed that NAR is an independent risk factor for mortality in TBI patients and a strong independent predictor of poor outcomes. Utilizing NAR may aid in the early identification of patients with poor prognoses and assist in planning aggressive treatment strategies.

It is estimated that traffic accidents and falls constitute the predominant causes of TBI.[14,15]. The condition is characterized by an initial mechanical impact injury, followed by a secondary injury driven by biochemical processes, notably inflammation[16]. This inflammatory response compromises the integrity of the blood-brain barrier and facilitates the release of neurotoxins, which may subsequently contribute to neurodegenerative processes[17]. Notably, Mediators like DAMPs, cytokines, and chemokines are quickly released post-injury, peaking within hours., during which cytokines and chemokines facilitate the recruitment of neutrophils and macrophages[19]. Consequently, neutrophils contribute to exacerbated tissue damage and increased barrier permeability, leading to acute and potentially chronic neuroinflammation[12]. Interestingly, many studies have found that elevated neutrophil levels upon admission are associated with early neurological deterioration and poor prognosis[7,19]. Additionally, albumin, a marker of nutritional status, plays a critical role in maintaining vascular integrity and scavenging free radicals[20]. Hypoalbuminemia, frequently observed in TBI patients, is correlated with poorer prognostic outcomes[21]. Notably, individuals presenting with serum albumin levels below 3.5 g/dL exhibit increased 30-day mortality rates[22]. In our study, non-survivors generally demonstrated elevated neutrophil counts and reduced albumin levels. This subset of patients exhibited a pronounced inflammatory

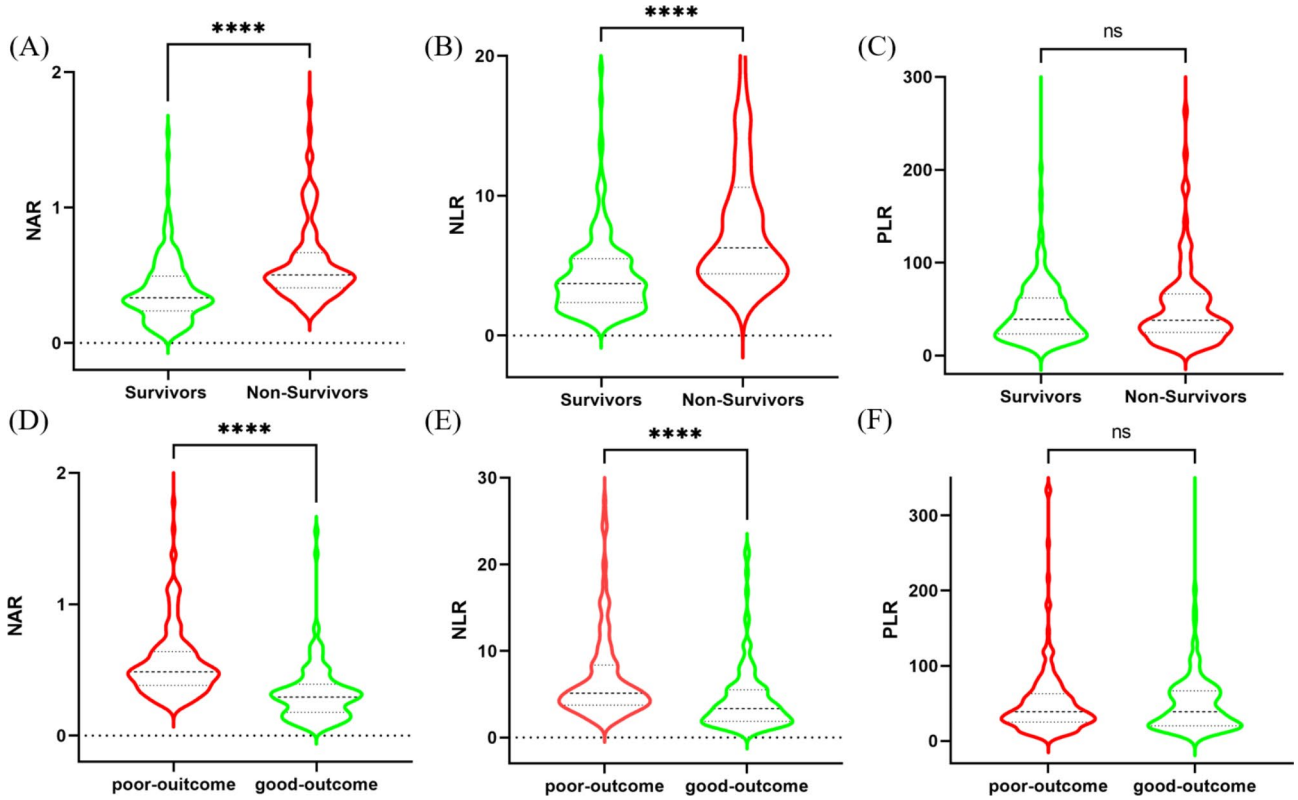

Fig. 3. The boxplots of the NAR, NLR and PLR grouped based on survival and prognosis. (**A**) The mean NAR of survivors group was higher than that in non-survivors group; (**B**) The mean NLR of survivors group was higher than that in non-survivors group; (**C**) The mean PLR of survivors group was lower than that in non-survivors group; (**D**) The mean NAR of poor-outcome group was higher than that in good-outcome group ; (**E**) The mean NLR of poor-outcome group was higher than that in good-outcome group ; (**F**) The mean PLR of poor-outcome group was lower than that in good-outcome group. NAR, neutrophil-albumin ratio; NLR, neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio.

response in the early stages of TBI. Additionally, poor nutritional status emerged as a significant adverse factor influencing patient prognosis.

Numerous clinical studies have concentrated on identifying blood biomarkers that are effective, accessible, and cost-efficient for the early prediction of TBI severity[23–25]. However, the majority of these studies necessitate the use of cerebrospinal fluid (CSF) for testing, thereby significantly complicating the clinical procedures and diagnostic processes[26]. In our study, we conducted a comparative analysis of the differential expression levels of various plasma biomarkers between the poor prognosis and good prognosis groups in TBI patients. The findings indicated that numerous blood biochemical parameters in non-survivors exhibited significant differences compared to survivors, consistent with previous research[27]. However, the results were frequently influenced by individual physiological variations. The composite index is capable of comprehensively reflecting a range of physiological and pathological processes, thereby mitigating the influence of individual variability to some extent and enhancing the stability and comparability of the index. Consequently, we selected three composite indicators—NLR, PLR, and NAR—which have been extensively studied in the context of other diseases and primarily reflect inflammatory and nutritional statuses pertinent to prognosis[6–8]. Unexpectedly, the NAR emerged as the most effective tool for predicting TBI among the three composite indicators. As a composite index integrating neutrophil and albumin levels, the NAR offers a distinctive multi-dimensional approach to prognosticating TBI. This index encapsulates the interplay between inflammatory and nutritional states, thereby providing a comprehensive assessment of the systemic physiological condition and prognosis of patients.

The NAR represents a novel composite biomarker designed to overcome the limitations inherent in prior studies that concentrated on singular inflammatory markers[28]. By integrating neutrophil counts, indicative of inflammatory response, with albumin levels, reflective of nutritional status, NAR enhances predictive accuracy for a range of clinical conditions[28–31]. In our study involving 297 patients with TBI, we identified a significant correlation between NAR and TBI outcomes. The NAR serves as an indicator of systemic inflammation and the risk of secondary brain injury. It is characterized by ease of measurement, high reproducibility, sensitivity, specificity, and the utilization of routine test components. These attributes render NAR a promising tool for improving the assessment of TBI prognosis and expediting decision-making processes in emergency settings. Consequently, NAR represents a significant advancement in the management and prognostic accuracy of TBI patients.

Demographic	Full cohort ($n=297$)	Survivors ($n=187$)	Non-survivors ($n=110$)	t/x2/z	*p*-value
Demographic					
Age, years, Mean (SD)	41.28 ± 19.85	41.61 ± 20.01	40.72 ± 19.66	0.375	0.708[a]
Gender, male, n (%)	223 (75.1%)	147 (78.6%)	76 (69.1%)	3.354	**0.067**[b]
Mechanism of injury				5.959	0.114[b]
Traffic accident, n (%)	192 (64.6%)	113 (60.4%)	79 (71.8%)	/	/
High fall, n (%)	31 (10.4%)	20 (10.7%)	11 (10%)	/	/
Stumble, n (%)	59 (19.9%)	45 (24.1%)	14 (12.7%)	/	/
Others, n (%)	15 (5.1%)	9 (4.8%)	6 (5.5%)	/	/
Clinical characteristics					
Admission SBP, mmHg, median (IQR)	120 [106–138]	121 [107–141]	119 [101–134.25]	1.569	0.117[c]
Admission DBP, mmHg, median (IQR)	71 [60–84]	72 [62–84]	69 [54.75–84]	1.345	0.179[c]
Admission GCS score, median (IQR)	5 [4–7]	6 [5–8]	5 [3–6]	6.253	**< 0.001**[c]
Respiratory rate, median (IQR)	20 [16–23]	20 [18–24]	17.5 [15–21.25]	3.568	**< 0.001**[c]
Temperature, ℃, median (IQR)	36.8 [36.5–37.1]	36.8 [36.5–37.3]	36.7 [36.2–37]	2.556	**0.011**[c]
Heart rate, median (IQR)	102 [86–120]	100 [86–116]	107 [86.25–126]	1.629	0.103[c]
Laboratory examination					
Leukocytes, 10^9/L, median (IQR)	15.14 [11.33–19.6]	15.12 [11.2–19.49]	15.42 [11.89–19.72]	0.278	0.781[c]
Platelets, 10^9/L, median (IQR)	90 [56.5–141.5]	101 [69–160]	73.5 [45–108.75]	4.224	**< 0.001**[c]
Erythrocyte, 10^9/L, Mean (SD)	88.93 ± 23.31	91.75 ± 22.30	84.13 ± 24.30	2.753	**0.006**[a]
Neutrophils, 10^9/L, Mean (SD)	11.62 ± 4.57	10.69 ± 4.60	13.20 ± 4.09	4.713	**< 0.001**[a]
Lymphocyte, 10^9/L, median (IQR)	2.5 [1.75–3.4]	2.8 [2.1–3.6]	1.95 [1.2–2.7]	6.343	**< 0.001**[c]
Cholesterol, mmol/L, Mean (SD)	3.72 ± 1.30	4.10 ± 1.32	3.10 ± 0.99	7.381	**< 0.001**[a]
LDH, U/L, median (IQR)	399 [297–587]	373 [291–504]	465 [321–760]	3.267	**< 0.001**[c]
Glu, mmol/L, median (IQR)	10.24 [7.93–14.3]	8.92 [7.37–12.6]	13.05 [9.71–16.36]	6.269	**< 0.001**[c]
Albumin, g/L, Mean (SD)	28.66 ± 8.03	30.95 ± 7.51	24.75 ± 7.37	6.919	**< 0.001**[a]
NLR, median (IQR)	4.53 [2.01–6.9]	3.71 [2.35–5.48]	6.27 [4.40–10.60]	7.836	**< 0.001**[c]
PLR, median (IQR)	38.79 [23.45–64.01]	39.02 [23.13–61.90]	38.03 [24.90–66.27]	0.462	0.644[c]
NAR, median (IQR)	0.41 [0.29–0.56]	0.33 [0.24–0.49]	0.50 [0.41–0.67]	7.064	**< 0.001**[c]
Injury types					
EDH, n (%)	26 (8.8%)	12 (6.4%)	14 (12.7%)	/	/
SDH, n (%)	66 (22.2%)	27 (14.4%)	39 (35.5%)	/	/
SAH, n (%)	165 (55.6%)	89 (47.6%)	76 (69.1%)	/	/
Contusions, n (%)	160 (53.9%)	106 (56.7%)	54 (49.1%)	/	/
Diffuse axonal injury, n (%)	102 (34.3%)	79 (42.2%)	23 (20.9%)	/	/
Length of ICU stay (day)	9 [2–23]	16 [7–31]	2 [1–6]	8.483	**< 0.001**[c]
Length of hospital stay (day)	15 [5–34]	26 [12–44]	5 [3–10.25]	9.275	< 0.001[c]

Table 1. Comparison of demographic, clinical and laboratory characteristics of patients. SD: standard deviation; SBP: systolic blood pressure; DBP: diastolic blood pressure; GCS: Glasgow Coma Scale; IQR: interquartile range; LDH: lactic dehydrogenase; Glu: blood glucose; NLR: neutrophil-lymphocyte ratio; PLR: platelet-to-lymphocyte ratio; NAR: neutrophil-albumin ratio; EDH: epidural hematoma; SDH: subdural hematoma; SAH: subarachnoid hematoma; The bold values indicated was considered statistically significant. an independent sample t-test; b $\chi 2$ test; c non-parametric rank-sum test.

While our study underscores the potential utility of the NAR in evaluating TBI prognosis, its clinical applicability warrants further validation. The limitations inherent in our research—such as its retrospective design, limited sample size, and potential data bias—underscore the need for large-scale, multicenter prospective studies to confirm the efficacy of NAR. Furthermore, it is essential to scrutinize the interactions between NAR and other prognostic factors, as well as to investigate its temporal dynamics. Comprehensive examination through both laboratory and clinical trials is critical to validate NAR as a dependable prognostic indicator for the management of TBI patients.

Conclusion

Our results suggest that PLR is not a valuable prognostic indicator among the three composite indicators of NAR, PLR and NLR in TBI patients. Although both NAR and NLR are valuable prognostic indicators, NAR is a better predictor of prognosis in patients with TBI.

Variable	Odds Ratio	95% Confidence Interval [25%, 75%]	*p*-value
Gender	1.293	[0.618–2.706]	0.495
Age, years	0.996	[0.979–1.012]	0.609
Respiratory rate	0.940	[0.889–0.994]	**0.031**
Temperature, ℃	1.039	[0.730–1.477]	0.833
Admission GCS score	0.722	[0.604–0.863]	**< 0.001**
Erythrocyte, 10^9/L	0.998	[0.984–1.012]	0.800
LDH, U/L	1.000	[0.999–1.001]	0.777
Glu, mmol/L	1.125	[1.049–1.206]	**0.001**
Cholesterol, mmol/L	0.592	[0.451–0.777]	**< 0.001**
NLR	1.116	[1.025–1.215]	**0.011**
PLR	1.003	[0.996–1.011]	0.411
NAR	6.208	[1.424–27.058]	0.015

Table 2. Multivariable logistic regression models of traumatic brain injury for predicting death. NAR: neutrophil-albumin ratio; NLR: neutrophil-lymphocyte ratio; LDH: lactic dehydrogenase; Glu: Blood glucose; The bold values indicated was considered statistically significant.

Demographic	Poor prognosis ($n = 175$)	Good prognosis ($n = 122$)	t/x2/z	*p*-value
Demographic				
Age, years, Mean (SD)	41.85 ± 19.22	40.47 ± 20.78	0.591	0.555 [a]
Gender, male, n (%)	126(72%)	97(79.5%)	2.166	0.141 [b]
Clinical characteristics				
Hemorrhage, n (%)	136(77.7%)	55(45.1%)	33.351	**< 0.001** [b]
Admission SBP, mmHg, median (IQR)	120 [104–136]	120.5 [107–142]	0.852	0.394 [c]
Admission DBP, mmHg, median (IQR)	70 [60–83]	73. [60.75–85.00]	0.686	0.493 [c]
Admission GCS score, median (IQR)	5 [4–6]	7 [5–9]	4.576	**< 0.001** [c]
Respiratory rate, median (IQR)	20 [16–22]	20 [16–24]	1.193	0.233 [c]
Temperature, ℃, median (IQR)	36.7 [36.4–37.0]	36.8 [36.5–37.3]	1.412	0.158 [c]
Heart rate, median (IQR)	102 [84–121]	101.5 [86–117]	0.161	0.872 [c]
Laboratory examination				
Leukocytes, 10^9/L, median (IQR)	15.12 [11.34–19.15]	15.83 [11.31–20.12]	0.681	0.496 [c]
Platelets, 10^9/L, median (IQR)	84 [53–132]	92 [61–159.25]	1.531	0.126 [c]
Erythrocyte, 10^9/L, Mean (SD)	87.03 ± 23.41	91.66 ± 22.99	1.688	0.092 [a]
Neutrophils, 10^9/L, median (IQR)	12.3 [10.3–15.4]	9.45 [6.0–12.2]	7.065	**< 0.001** [c]
Lymphocyte, 10^9/L, median (IQR)	2.4 [1.7–3.2]	2.65 [2.00–3.50]	1.838	0.066 [c]
Cholesterol, mmol/L, Mean (SD)	3.53 ± 1.23	4.00 ± 1.35	3.106	**0.002** [a]
LDH, U/L, median (IQR)	403 [305–616]	388 [287.25–576]	1.220	0.222
Glu, mmol/L, median (IQR)	11.7 [8.47–15.12]	9.06 [7.75–13.00]	3.075	**0.002**
Albumin, g/L, Mean (SD)	26.03 ± 7.30	32.43 ± 7.52	-7.340	**< 0.001** [a]
NLR, median (IQR)	5.12 [3.74–8.38]	3.36 [1.88–5.51]	6.111	**< 0.001** [c]
PLR, median (IQR)	38.79 [25.00–62.73]	38.83 [20.00–66.57]	0.111	0.912 [c]
NAR, median (IQR)	0.48 [0.38–0.64]	0.29[0.18–0.39]	8.895	**< 0.001** [c]
Position of hemorrhage				
EDH, n (%)	20(11.4%)	6(4.9%)	/	/
SDH, n (%)	50(28.6%)	16(13.1%)	/	/
SAH, n (%)	117(66.9%)	48(39.3%)	/	/
Contusions, n (%)	116(66.3%)	44(36.1%)	/	/

Table 3. Comparison of demographic, clinical, and laboratory characteristics between patients with and without poor prognosis. SD: standard deviation; SBP: systolic blood pressure; DBP: diastolic blood pressure; GCS: Glasgow Coma Scale; IQR: interquartile range; Glu: Blood glucose; NLR: neutrophil-lymphocyte ratio; PLR: platelet-lymphocyte ratio; NAR: neutrophil-albumin ratio; EDH: epidural hemorrhage; SDH: subdural hemorrhage; SAH: subarachnoid hemorrhage; The bold values indicated was considered statistically significant. an independent sample t-test; b $\chi 2$ test; c non-parametric rank-sum test.

Variable	Odds ratio	95% confidence interval [25%, 75%]	*p*-value
Gender	1.329	[0.675–2.616]	0.410
Age, years	1.005	[0.991–1.019]	0.460
Hemorrhage	2.946	[1.650–5.260]	**<0.001**
Admission GCS score	0.855	[0.757–0.966]	**0.012**
Erythrocyte, 10^9/L	1.004	[0.992–1.017]	0.483
Glu, mmol/L	1.024	[0.964–1.088]	0.438
Cholesterol, mmol/L	0.949	[0.758–1.189]	0.651
NLR	1.001	[0.929–1.078]	0.983
NAR	116.588	[17.847 − 761.609]	**<0.001**

Table 4. Multivariable logistic regression models of traumatic brain injury for predicting poor prognosis. NAR: neutrophil-albumin ratio; NLR: neutrophil-lymphocyte ratio; GCS: Glasgow Coma Scale; Glu: Blood glucose. The bold values indicated was considered statistically significant.

Data availability

This study strictly follows the terms of the Ethics Committee of West China Hospital of Sichuan University and respects patient privacy. The dataset used and analyzed during this study is available from the corresponding author on reasonable request.

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The authors declare no competing interests.

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Ethics approval
This study was performed in accordance with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Sichuan University (No.20231411).

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