ORIGINAL ARTICLE

Is C-reactive protein-albumin ratio or neutrophil-lymphocyte ratio a better indicator to predict in-hospital mortality in traumatic brain injury?

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ABSTRACT

BACKGROUND: Neutrophil-lymphocyte ratio (NLR) and C-reactive protein-albumin ratio (CAR) are simple and objective markers of inflammatory responses. However, there are no studies in the literature evaluating these two markers together in traumatic brain injury (TBI). Therefore, this study aimed to examine whether CAR or NLR is a better biomarker for predicting in-hospital mortality in patients with TBI.

METHODS: A total of 257 consecutive patients admitted to the hospital between January 2016 and December 2021 were included in the study. The files of all patients aged >18 years with TBI were retrospectively reviewed. Clinical characteristics, Glasgow Coma Scale, and patient data during hospital stay were recorded. Definitive diagnosis was made using computed brain tomography. Routine blood tests were performed in the first 12–24 h of hospitalization. Laboratory results of patients with and without in-hospital mortality were comparatively analyzed.

RESULTS: According to the Mann–Whitney U-test, median CRP, CAR, NLR, WBC, monocyte, neutrophil, RDW-CV, RDW-SD, and platelet values were significantly higher, whereas median albumin and RBC values were significantly lower in patients with in-hospital mortality. Student's t-test showed that the mean hemoglobin level was significantly lower in patients with in-hospital mortality compared to other patients. Univariate logistics regression model revealed that age, albumin, CRP, CAR, NLR, WBC, monocyte, neutrophil, RBC, RDW-CV, RDW-SD, and hemoglobin were the factors predicting mortality. However, in the multivariate logistic regression model, only age, albumin, CAR, and WBC were the factors predicting mortality. Areas under the curve were 0.891 for CAR (95% GA, 0.847–0.935), 0.759 for WBC (95% GA, 0.696–0.823), and 0.671 for NLR (95% GA, 0.598–0.744).

CONCLUSION: The results of this study showed that CAR has better prognostic value than NLR in predicting in-hospital mortality in patients with TBI.

Keywords: C-reactive protein-albumin ratio; in-hospital mortality; neutrophil-lymphocyte ratio; traumatic brain injury.

INTRODUCTION

Traumatic brain injury (TBI) is the general term for all pathological changes in the brain caused by an external mechanical force.[1] TBI is a disease characterized as a silent epidemic. It is estimated that annually 69 million individuals are affected by TBI worldwide.[2] TBI is a critical health issue because of its prevalence, high mortality, and morbidity rates, and because it creates a heavy economic burden on society. Despite the criticality of TBI, the standard clinical instruments for predicting outcomes after brain injury are still inadequate.

Understanding the nature of TBI and accurately predicting its pathology will help doctors in assessing the current condition of patients and making appropriate treatment decisions. Neurological examinations and neuroimaging are common diagnostic strategies for TBI. The Glasgow Coma Scale

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(GCS) is widely used for assessing patients' consciousness and predicting outcomes.[3] Other evaluation methods, such as post-traumatic amnesia and loss of consciousness, are also available. However, these scoring systems can give different results during the neurological evaluation of patients according to the clinical experience of health-care professionals performing the tests. Therefore, new and objective prognostic models should be investigated. Biomarkers are objective measurements that are routinely used in patient follow-up and treatment management.[4]

Therefore, the aim of this observational study was to examine whether CAR or neutrophil-lymphocyte ratio (NLR) is a better biomarker for predicting in-hospital mortality in patients with TBI.

MATERIALS AND METHODS

The records of 746 consecutive patients with TBI admitted to Aksaray Training and Research Hospital between January 2016 and December 2021 were examined. Sample selection was not performed. All patient files that met the diagnosis of TBI between the specified dates were examined. Patient records during hospitalization were examined and recorded. Patients with missing hospital records, those who died within the first 12 h after hospitalization, those under the age of 18, and patients with chronic diseases (connective tissue, autoimmune, and inflammatory diseases; liver cirrhosis and dysfunction; malignancies; and chronic heart diseases) were excluded from the study. Patients with pregnancy were not included in the study. Patients with head trauma but no signs of injury revealed in computed brain tomography were also excluded from the study. Based on these exclusion criteria, 257 patients were included in the study. Patients were divided into two groups: Patients with in-hospital mortality (84 patients who died during hospitalization) and patients without in-hospital mortality (173 patients who did not die). Data from both groups were separately evaluated. The GCS of patients on admission to the emergency ward was recorded. Neurological examination was performed on all patients. Trauma patterns, other traumatic organs and extremity pathologies, and one or more pathological findings in CT were classified and recorded. CT results were classified according to the presence of brain shifts and ventricular hemorrhage. Laboratory results between the 12th and 24th h of patient admission were recorded for statistical analysis. NLR was obtained by dividing the neutrophil count by the lymphocyte count. CAR was obtained by dividing the C-reactive protein (CRP) level by the albumin level. Both NLR and CAR were recorded for statistical analysis.

This retrospective cross-sectional study was conducted in accordance with the 1989 Helsinki Declaration and approved by the Ethics Committee of Aksaray University Faculty of Medicine (Institutional Review Board number 62-SBKAEK, decision number 2021/16–08, date December 02, 2021).

Statistical Analysis

Results are presented as mean±standard deviation for normally distributed data, median (min-max) for abnormally distributed data and percentage (%). To investigate the distribution pattern of the data, Kolmogorov–Smirnov normality test was used. Only hemoglobin data distributed normally, thus, were compared using Student's independent samples t-test; and the other blood test parameters did not distribute normally, thus were compared using Mann–Whitney U test. Binary outcomes were investigated using Chi-square test. To investigate the predictive factors of mortality in TBI, univariate and multivariate logistic regression analyses were used. The variables with p-value of primary comparison less than 1 were included in the univariate logistic regression model. Moreover, the variables with a p-value of univariate logistic regression analysis <0.05 were included in the multivariate logistic regression model. Cox and Snell pseudo-R^2 and Nagelkerke pseudo-R^2 tests were used to assess the consistency between the variables. To assess the predictive value of variables, receiver operating characteristics (ROC) curve analysis test was used. If the area under the ROC curve is 0.5, the model does not discriminate; 0.5–0.7, the model has poor to fair discrimination; 0.7–0.8, the model has acceptable discrimination; 0.8–0.9, the model has excellent; and 0.9–1.0, the model is a very rare outcome.[5] For statistical analysis of all data, we used SPSS 23.0 software for MacOs (SPSS Inc., Chicago, IL, USA). P<0.05 was considered statistically significant.

RESULTS

In this study, 257 patients with TBI were included in the study. The patients without hospital mortality group consisted of 173 patients (155 males and 18 females, median age: 43 [19–91]) and the patients with hospital mortality group consisted of 84 patients (66 males and 18 females, median age: 53 [19–97]). The median age of the patients with hospital mortality was significantly higher compared to the patients without (p=0.013).

The comparison of blood analysis parameters between the groups is presented in Table 1. According to the Mann–Whitney U-test, the median lymphocyte, eosinophil, basophil, and platelet values did not significantly differ between the patients with and without hospital mortality (p=0.183, p=0.556, p=0.068, and p=0.835, respectively). However, the median CRP, CAR, NLR, WBC, monocyte, neutrophil, RDW-CV, RDW-SD, and platelet values were significantly higher (p<0.001 and p=0.002, respectively), and median albumin and RBC values were significantly lower (p<0.001) in patients with hospital mortality, compared with the patients without. Student's t-test revealed that the mean hemoglobin was significantly lower in patients with hospital mortality, compared with the patients without (p<0.001).

Table 1. Comparison of blood analysis parameters between the patients with and without mortality

	Patients without mortality	Patients with mortality	p–value
Age	43 (19–91)	53 (19–97)	0.013
Albumin	37 (23–51)	32 (25–45)	<0.001
C-reactive protein	19 (1–194)	78.78 (1–290)	<0.001
C-reactive protein-albumin ratio	0.5 (0.003–5.2)	2.435 (0.025–10)	<0.001
Neutrophil–lymphocyte ratio	5.88 (0.8–41)	12.5 (0.57–62.5)	<0.001
White blood cells	11.82 (4–27)	16.02 (6–36)	<0.001
Lymphocyte	1.44 (0.32–4.7)	1.05 (0.06– 9)	0.183
Monocyte	0.73 (0.07–2.6)	0.94 (0.09–2.7)	0.002
Neutrophil	9 (2–26)	13,57 (2.7–31)	<0.001
Eosinophil	0.02 (0.001–0.58)	0.012 (0.001–0.7)	0.556
Basophil	0.02 (0.001–0.34)	0.011 (0.001–0.192)	0.068
Red blood cells	4.45 (1.74–5.96)	3,94 (2–5.6)	<0.001
RBC distribution width-variation coefficient	13 (9.8–25)	13.2 (10–25.6)	<0.001
RBC distribution width-standard deviation	41 (27–78)	43 (26–61)	<0.001
Platelet	198 (56–470)	199 (70–386)	0.835
Hemoglobin	12.77±1.98	11.78±2.17	<0.001

Table 2. The distribution of the primary diagnosis of the patients with traumatic brain injury

	Patients without mortality (n=173)	Patients with mortality (n=84)
	n (%)	n (%)
Subarachnoid hemorrhage	81 (46.8)	35 (41.7)
Acute subdural hematoma	30 (17.3)	32 (38.1)
Contusio cerebri	16 (9.2)	8 (9.5)
Epidural hematoma	20 (11.6)	2 (2.4)
Brain edema	0 (0)	5 (6)
Spot bleeding	1 (0.6)	0 (0)
Depression fracture	6 (3.5)	2 (2.4)
Linear fracture	17 (9.8)	0 (0)
Pneumocephalus	2 (1.2)	0 (0)

Table 3. The distribution of the trauma type of the patients with traumatic brain injury

	Patients without mortality (n=173)	Patients with mortality (n=84)
	n (%)	n (%)
In- vehicle accient	86 (50.3)	27 (32.1)
Out of vehicle accident	12 (7.0)	9 (10.7)
Fall	36 (21.1)	18 (21.4)
Forensic case	15 (8.8)	2 (2.4)
Industrial accicent	9 (5.3)	3 (3.6)
Motorbike accident	8 (4.7)	23 (27.4)
Bicycle accident	4 (2.3)	0 (0)
Gunshot injury	1 (0.6)	2 (2.4)

Table 2 presents the distribution of the primary diagnosis and Table 3 presents the distribution of the trauma type of the patients with TBI. According to the Chi-square test, frequency of shift was significantly higher in patients with mortality (25 [29.8%] patients), compared with the patients without (14 [8.1%] patients) (X^2=20.627, p<0.001).

Table 4 presents the univariate and multivariate logistic regression analysis results. In the univariate logistic regression model, the age, albumin, CRP, CAR, NLR, WBC, monocyte, neutrophil, RBC, RDW-CV, RDW-SD, and hemoglobin were predictive factors of mortality (p=0.016, p<0.001, p=0.002, and p=0.006, respectively). However, only the age, albumin, CAR, and WBC were found to be predictive factors of mortality in the multivariate logistic regression model (p=0.008, p=0.026, and p<0.001, respectively).

Figure 1 shows the ROC curve representing the predictive value of CAR, NLR, WBC, and hemoglobin for mortality. The areas under curve were; CAR 0.891 (95%CI, 0.847–0.935), WBC 0.759 (95%CI, 0.696–0.823), and NLR 0.671 (95% CI, 0.598–0.744).

Table 4. The univariate and multivariate logistic regression analysis results

	Univariate		Multivariate	
	OR (95% CI)	**p-value**	**OR (95% CI)**	**p-value**
Age	1.016 (1.003–1.029)	0.016	1.032 (1.008–1.056)	0.008
Albumin	0.844 (0.792–0.899)	<0.001	0.883 (0.791–0.985)	0.026
C-reactive protein	1.046 (1.034–1.058)	<0.001	–	
C-reactive protein-albumin ratio	4.888 (3.208–7.448)	<0.001	3.468 (2.188–5.495)	<0.001
Neutrophil–lymphocyte ratio	1.064 (1.032–1.097)	<0.001	0.99 (0.941–1.041)	0.685
White blood cells	1.218 (1.145–1.296)	<0.001	1.307 (1.166–1.465)	<0.001
Monocyte	2.642 (1.448–4.819)	0.002	–	
Neutrophil	1.184 (1.117–1.256)	<0.001	–	
Basophil	0.007 (0–33.094)	0.25	–	
Red blood cells	0.429 (0.287–0.641)	<0.001	1.067 (0.208–5.476)	0.938
RDW- CV	1.218 (1.057–1.402)	0.006	0.857 (0.604–1.215)	0.386
RDW- SD	1.086 (1.03–1.145)	0.002	1.056 (0.944–1.182)	0.343
Hemoglobin	0.788 (0.69–0.901)	<0.001	0.875 (0.499–1.535)	0.642
			Cox & Snell pseudo-R^2=0.484	
			Nagelkerke pseudo-R^2=0.674	
			Hosmer and Lemeshow Test p=0.9	

OR: Odds ratio; CI: Confidence interval; RDW-CV: RBC distribution width-variation coefficient; RDW-SD: RBC distribution width-standard deviation.

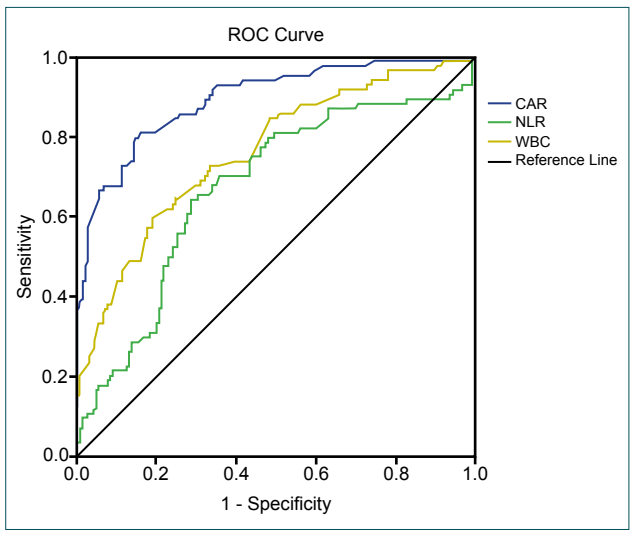

Figure 1. The ROC curve representing the predictive value of CAR, NLR, WBC, and hemoglobin for mortality.

DISCUSSION

To the best of our knowledge, this is the first study in the literature that simultaneously examines the prognostic value of CAR and NLR in TBI. The key results of this study were as follows: In the ROC curve, CAR and NLR parameters were located in the high sensitivity zone. Univariate logistics regression model revealed that age, albumin, CRP, CAR, NLR, WBC, monocyte, neutrophil, RBC, RDW-CV, RDW-SD, and hemoglobin were the factors predicting mortality. However, in the multivariate logistic regression model, only age, albumin, CAR, and WBC were the factors predicting mortality.

The inflammatory response associated with TBI begins within a few hours of trauma and reaches its peak within 12–24 h.[6] TBI has two stages. Primary damage is defined as neural tissue damage caused by a primary force. Secondary damage is the inflammatory response to injury, which can lead to the deterioration of the blood–brain barrier (BBB), neurotoxicity, neuronal death, and/or neurodegeneration.[7] Immediately after trauma, various molecules (chemokines, pro-inflammatory cytokines, and angiogenic factors) are released from damaged neuronal cells of the brain. Some of these molecules are interleukin-1alfa, interleukin-6 and -8, tumor necrosis factor-alpha, interferon-gamma, microglia colony-stimulating factor-1, and granulocyte-macrophage colony-stimulating factor. These molecules activate neutrophils, which surround the local area of the injury, and phagocytosis of the remaining damaged cells in this area occurs as a result. Neutrophils are the first and most important cells that immediately respond to injuries. They migrate to the injury site and search for pathogens for phagocytosis. Neutrophil stimulation also leads to degranulation of granules and the release of bactericidal proteinases, such as proteins that increase permeability, elastase, and metalloproteinases. Improper activation of endothelial cells may further impair the integrity of the BBB and can lead to protein fluid passage into the interstitial cavity and significant leukocyte infiltration. Increased BBB permeability is observed within hours after injury. The degradation of the

vascular wall causes more plasma and molecules to leak into the extravascular cavity, thereby increasing cerebral edema and exacerbating secondary damage.[8–10] NLR is a simple and reliable biomarker calculated from neutrophil and lymphocyte counts obtained from a complete blood count, and its use in clinical practice is growing. NLR is generally considered as an indicator of inflammation before any clinical findings can be observed. Studies have reported that a high NLR level may be a poor prognostic marker in TBI.[11–16] In the present study, a high NLR level was also identified as a poor prognostic marker, which is consistent with other studies in the literature.[11–16]

CRP is a protein synthesized by the liver, and its level rises in response to inflammation. It is a dominant protein in the acute-phase response. CRP has pro-inflammatory and anti-inflammatory properties.[17] Blood CRP level is used as an indicator of any ongoing inflammatory response, including those occurring in cancer.[18] Many recent studies have shown that CRP is a promising candidate for predicting prognosis after TBI.[19–24]

Serum albumin decreases in stress conditions due to blood loss as a result of trauma-related vascular damage. In TBI, its level decreases due to the deterioration of the BBB and subsequent passage of albumin to the nonvascular area. The literature evidence shows that a low albumin level is an independent marker of poor prognosis in TBI.[25,26] CAR is calculated based on the ratio of CRP, an indicator of inflammation, and albumin, which is roughly an indicator of nutritional status. Increased CAR indicates higher inflammation and worse nutritional status.[25]

Therefore, CAR can be a promising candidate for predicting mortality after TBI. There are limited studies in the literature on the relationship between head trauma and CAR levels. However, recent studies have shown that such a relationship exists and can be used to evaluate TBI.[19–22]

However, to the best of our knowledge, there are no studies in the literature comparing the effectiveness of NLR and CAR in TBI. According to the results of this study, CAR was more effective than NLR for predicting in-hospital mortality following TBI. In light of this finding, CAR can be used in TBI as a better prognostic marker than NLR.

The degree of TBI is categorized as mild, moderate, and severe. In the present study, all TBI types and severities were included to obtain a large patient sample. Therefore, we aimed to develop a simple, reliable, and generalizable test for predicting mortality. Indeed, studies conducted so far report that NLR is a poor prognostic indicator regardless of the degree of TBI.[16] NLR has been associated with poor prognosis even in mild TBI.[13]

Wang et al.[20] showed that increased CAR values were associated with poor prognosis in TBI. They included patients with severe and moderate TBI in their study but excluded patients with mild TBI. The authors pointed this out as a limitation of their study.

There are certain limitations of this study. Only routine laboratory parameters evaluated as part of standard care in our hospital were included in the analyses. Laboratory parameters that are not routinely used, such as cytokines, were not included in the analyses. Other limitations of this study include the fact that reference intervals for complete blood count parameters vary depending on age and gender, that differences in measurement methods can affect measurement results, and that differences among societies may have an impact on reference values. Furthermore, only patients over the age of 18 were included in the study. Therefore, the results cannot be generalized to the entire age group. Finally, the study was designed retrospectively, conducted in a single center, and the patient group was relatively small.

Conclusion

The results of this study showed that NLR and CAR obtained from peripheral blood within the 1st day of TBI are successful predictors of in-hospital mortality among TBI patients. This suggests that CAR and NLR can be useful determinants of patient outcomes in TBI as they are easy to calculate based on cost-effective blood tests. However, further studies with larger series are needed for more precise and generalizable results. The results of this study showed that CAR outperformed NLR as a predictor of in-hospital mortality in TBI. Therefore, future studies should focus on CAR instead of NLR. Further studies with large patient series investigating CAR as a predictor of mortality will surely provide valuable results.

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Ethics Committee Approval: This study was approved by the Aksaray University Clinical Research Ethics Committee (Date: 02.12.2021, Decision No: 2021/16-08).

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ORİJİNAL ÇALIŞMA - *ÖZ*

Travmatik beyin hasarında hastane içi mortaliteyi tahmin etmek için C-reaktif protein albümin oranı mı yoksa nötrofil-lenfosit oranı mı daha iyi bir göstergedir?

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AMAÇ: Nötrofil-lenfosit oranı (NLR) ve C-reaktif protein albümin oranı (CAR) enflamatuvar yanıtın basit ve nesnel bir belirtecidir. Ancak, bu iki belirteç travmatik beyin hasarında birlikte çalışılmamıştır. Bu yüzden çalışmada TBH'li hastalarda NLR ve CAR'nin hastane içi mortaliteyi öngörmede hangisinin daha iyi bir biyobelirteç olduğu çalışıldı.
GEREÇ VE YÖNTEM: Çalışmaya Ocak 2016 ile Aralık 2021 yılları arasında hastaneye kabul edilen ardışık 257 hasta dahil edildi. Tüm travmatik beyin hasarlı 18 yaş üstü hastaların dosyaları geriye dönük olarak incelendi. Hastanede kaldıkları süre içindeki verileri kaydedildi. Klinik özellikleri glaskow koma skorları (GKS) kaydedildi. Bilgisayarlı beyin tomografisi (BBT) ile tanıları sınıflandırıldı. Hastaların ilk 12–24 saat arasındaki rutin kan incelemeleri kaydedildi. Hastanede kaldıkları sürece ölenlerle ölmeyenlerin laboratuvar sonuçları karşılaştırılarak analiz edildi.
BULGULAR: Mann-Whitney U testine göre hastanede kaldıkları sürede ölen hastalarda ortanca CRP, CAR, NLR, WBC, monosit, nötrofil, RDW-CV, RDW-SD ve trombosit değerleri anlamlı derecede yüksek, ortanca albümin ve RBC değerleri ölmeyen hastalara göre anlamlı olarak daha düşüktü. Student t-testi, ölen hastalarda ortalama hemoglobinin ölmeyen hastalara göre anlamlı derecede düşük olduğunu gösterdi. Tek değişkenli lojistik regresyon modelinde yaş, albümin, CRP, CAR, NLR, WBC, monosit, nötrofil, RBC, RDW-CV, RDW-SD ve hemoglobin mortaliteyi öngören faktörlerdi. Bununla birlikte, çok değişkenli lojistik regresyon modelinde yalnızca yaş, albümin, CAR ve WBC'nin mortaliteyi öngören faktörler olduğu bulundu. Eğri altında kalan alanlar; CAR 0.891 (%95 GA, 0.847–0.935), WBC 0.759 (%95GA, 0.696–0.823) ve NLR 0.671 (%95 GA, 0.598–0.744).
TARTIŞMA: Bu çalışma, TBH'de hastane içi mortaliteyi ön görmede CAR'nin NLR ye göre daha iyi prognostik değere sahip olduğunu göstermiştir.
Anahtar sözcükler: C-reaktif protein albümin oranı; hastane içi mortalite; nötrofil-lenfosit oranı; travmatik beyin hasarı.

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