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Relationship between C-reactive protein/albumin ratio and lactate/albumin ratio and mortality in traumatic brain injury

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Abstract

We aimed to evaluate whether C-Reactive Protein/Albumin Ratio (CAR) and Lactate/Albumin ratio (LAR), which include acute phase reactants that are indicators of secondary brain injury processes such as inflammatory process, cytokine release, and hypoxia that develop after primary injury, are predictors of mortality in patients with traumatic brain injury (TBI). Patients who were admitted to the emergency department with a diagnosis of traumatic brain injury and stayed in the intensive care unit for more than 24 hours were included in the study. It was conducted by scanning the retrospective records of patients diagnosed with TBI between January 2022 and December 2023 in our hospital. As a result of our study, acute physiology and chronic health assessment II (APACHE II) and Injury Severity Score (ISS) were independent risk factors for mortality in TBI, while CAR and LAR were not independent variables in terms of mortality. Similar findings were valid for Glasgow Outcome Score (GOS). In addition, in patients who died; LAR, hypertension, high APACHE II score, low Glasgow Coma Scale (GCS), high ISS, mechanical ventilation requirements vasopressor requirements at first admission to intensive care, high lactate level, and low albumin levels were meaningful of the results. APACHE II and ISS were independent risk factors for mortality in TBI, while CAR and LAR were not independent variables in terms of mortality. Similar findings were valid for GOS. In addition, LAR levels were significantly higher in patients with ex. These results show us that LAR may be a parameter in mortality prediction.

Keywords: Traumatic brain injury, lactate/albumin ratio, c-reactive protein/albumin ratio, mortality

Introduction

It is estimated that approximately 50-60 million people worldwide suffer from traumatic brain injury (TBI) each year from various causes (traffic accidents, criminal cases, etc.) [1]. Traumatic brain injury is a critical health problem that imposes a significant economic burden on society, considering its high prevalence, mortality, and morbidity rates, and the need for intensive care and palliative care [2]. Therefore, it is necessary to quickly determine the extent and severity of TBI to facilitate triage, provide appropriate treatment, and predict prognosis [3].

It has been shown that acute inflammatory reactions occur after head injuries. Neuroinflammation, caused by damage to nerve components (e.g. neurons and glial cells) due to mechanical forces, is responsible for primary and secondary brain damage. C-Reactive Protein /Albumin Ratio (CAR); It has been proven to be useful in predicting mortality in diseases where the inflammatory process is at the forefront, such as sepsis. On this basis; it can be predicted that CAR, which can be used as a marker in the inflammatory process, can also reflect the degree of tissue damage and edema in TBI, as in inflammatory processes such as sepsis [4,5].

Serum Lactate level is a routinely assessed blood gas parameter and accurately reflects the hypoperfusion status. High lactate levels are associated with increased morbidity and mortality in conditions such as trauma, sepsis, and multiple organ failure [6]. Studies have shown that the lactate/albumin ratio (LAR) predicts prognosis better than the lactate ratio alone [7]. However, further studies are needed on its effectiveness in TBI. The purpose of this study is to evaluate the relationships between CAR and LAR with prognosis and mortality in cases with TBI. There is no study in the literature that evaluates CAR and LAR together in studies on TBI. Therefore, we believe that we will make an important contribution to the literature.

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Material and Methods

Study Protocol and Patient Selection

Our retrospective cross-sectional study was initiated based on the principles stated in the Declaration of Helsinki after Samsun University Clinical Research Ethics Committee approval (date: 17/04/2024, no: 2024/8/11) was obtained. It was conducted by scanning the retrospective records of patients diagnosed with traumatic brain injury who were admitted to the 3rd level intensive care unit due to trauma between January 2022 and December 2023 in our hospital. Patients over the age of 18 who applied to the emergency department with a diagnosis of traumatic brain injury and who stayed in the intensive care unit for more than 24 hours were included in the study. Pregnant patients, those under the age of 18, patients with previous traumatic or other brain injuries, patients who were hospitalized for less than 24 hours, and patients whose patient data could not be accessed were excluded from the study.

Data Collecting

Of the 1197 patients hospitalized during the study period, 197 patients with a diagnosis of TBI and who met the inclusion criteria were included in the study. At the time of patients' admission; Demographic data (age, gender, comorbidities, etc.), laboratory values, CAR, LAR, Glasgow Coma Scale (GCS), acute physiology and chronic health assessment (APACHE II) scale, Injury Severity Score (ISS), trauma etiology, vasopressor support, mechanical ventilation support, intensive care unit stay, Glasgow Outcome Score (GOS) at discharge from the intensive care unit (ICU), mortality data in the intensive care unit were obtained from the hospital's electronic medical records and file records.

The primary outcome in our analysis was the association of CAR, and LAR rate with in-hospital 24-hour mortality in patients with TBI. The secondary outcome was the association of the CAR and LAR ratio with GOS.

Statistical Analysis

Study statistics were performed using the Statistical Package for the Social Science (SPSS) 21st program (IBM, Corp. Armonk, NY, USA). Kolmogorov-Smirnov test was used for normality tests, and Shapiro-Wilk test was used for subgroup analyses with a sample size of 30 and below. Quantitative data were presented using mean (± standard deviation) or median (interquartile range), and categorical data were presented using frequency (and percentage). Mann-Whitney U test was used for comparisons between quantitative variables, and chi-square (χ^2) or Fisher's exact test was used for comparisons of categorical data. Variables affecting mortality and GOS were evaluated by binary logistic regression analysis. For all tests, $P<0.05$ was considered statistically significant.

Results

The files of 1170 patients followed with a diagnosis of trauma were scanned retrospectively. The study was completed with 119 patients who complied with the study protocol. The mortality rate was 27.7%. Although it was seen as numerically higher in men, it was not a statistically significant increase. Patients were examined in two groups as Survivors and non-Survivors. Hypertension (HT) (P=0.007), high APACHE II score (P=0.000), low GCS (P=0.000), high ISS (P=0.000), mechanical ventilation at first admission to the ICU (P=0.000) and vasopressor requirement (P=0.000), blood lactate level (P=0.001), albumin level (P=0.032), high LAR (P=0.000) were found to be statistically different between the two groups (Table 1).

Table 1. Patient demographic and clinical characteristics

Variables		All patients	Survivors (n=86)	Non-survivors (n=33)	P
Demographics	**Age (year) median (IQR)**	46 (40)	44 (37)	58 (53.5)	0.123
	Gender (male) n (%)	103 (86.6)	76 (73.8)	27 (26.2)	0.348
Comorbidities	**Hypertension n (%)**	39 (32.8)	22 (56.4)	17 (43.6)	0.007
	Diabetes n (%)	8 (6.7)	4 (50)	4 (50)	0.145
	Coroner artery disease n (%)	13 (10.9)	8 (61.5)	5 (38.5)	0.360
	Chronic renal failure n (%)	1 (0.8)	0 (0)	1 (100)	NA
APACHE II score median (IQR)		16.5 (19)	12 (11.5)	31 (13)	0.000
GCS median (IQR)		9 (10)	12 (8)	4 (3.5)	0.000
TRISS median (IQR)		9 (17)	9 (7)	25 (13)	0.000
Mechanical ventilation n (%)		63 52.9)	32 (50.8)	31 (49.2)	0.000
Vasopressors n (%)		43 (36.1)	14 (32.6)	29 (67.4)	0.000
Laboratory test	**Lactate (mmol/L) median (IQR)**	2.8 (1.9)	2.5 (1.3)	3.7 (2.4)	0.001
	CRP (mg/dL) median (IQR)	9.7 (23.3)	9.1 (24.6)	10.7 (20.3)	0.864
	Albumin (g/dL) mean±SD	32.9±4.6	33.5±4.4	31.4±4.6	0.032
	LAR median (IQR)	0.08 (0.07)	0.07 (0.05)	0.1 (0.1)	0.000
	CAR median (IQR)	0.3 (0.7)	0.2 (0.7)	0.3 (0.6)	0.697
Other	**Surgery n (%)**	35 (29.4)	25 (71.4)	10 (28.6)	0.895
	ICU during (day) median (IQR)	4 (9)	3 (8.5)	4 (9)	0.772
	GOS median (IQR)	7 (7)	8 (1)	1 (0)	0.000
	Brain death n (%)	10 (8.4))	10 (100)	0 (0)	NA

APACHE II: acute physiology and chronic health evaluation, CRP: C-reactive protein, GCS: Glasgow Coma Scale, GOS: Glasgow Outcome Scale, ICU: intensive care unit, TRISS: trauma score and injury severity score, CAR: C-reactive protein/albumin ratio, LAR: lactate to albumin ratio; P<0.05 was considered statistically significant

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In the binary logistic regression analysis for risk factors affecting mortality; APACHE II score (odds ratio [OR], 0.808; 95% confidence interval [CI] 0.712–0.918; p=0.001) and ISS (OR, 0.865; 95% CI, 0.784–0.954; P=0.004) were determined as independent risk factors for mortality at the time of admission to ICU. However, CAR (OR, 23.69; 95% CI 0.059–951.442; P=0.301) and LAR (OR, 0.312; 95% CI 0.000–73.000; P=0.952) were not associated with mortality (Table 2). APACHE II score (OR, 0.834; 95% CI; 0.735–0.946; P=0.005) and ISS (OR, 0.887; 95% CI 0.784–0.954; P=0.022) were determined as independent risk factors for GOS at the time of admission to the ICU. C-Reactive Protein/Albumin Ratio (OR, 0.195; 95% 0.006–5.877; P=0.347) and LAR (OR, 1.689; 95% CI 0.000–1070.000; P=0.977) were not associated with GOS (Table 3).

Table 2. Effect of variables on mortality

	Beta	SE	OR (95% CI)	P
APACHE II	-0.213	0.065	0.808 (0.712–0.918)	0.001
GCS	-0.247	0.130	0.781 (0.605–1.008)	0.058
ISS	-0.145	0.050	0.865 (0.784–0.954)	0.004
Lactate	-0.010	0.699	0.990 (0.252–3.894)	0.989
CRP	-0.099	0.103	0.906 (0.740–1.109)	0.338
Albumin	0.099	0.115	1.104 (0.881–1.385)	0.390
LAR	-1.165	19.237	0.312 (0.000–73.000)	0.952
CAR	3.165	3.059	23.69 (0.059–951.442)	0.301

APACHE II: acute physiology and chronic health evaluation, GCS: Glasgow Coma Scale, ISS: injury severity score, CRP: C-reactive protein, LAR: lactate/ albumin ratio, CAR: C-reactive protein/albumin ratio; P<0.05 was considered statistically significant

Table 3. Effect of variables on GOS

	Beta	SE	OR (95% CI)	P
APACHE II	-0.182	0.064	0.834 (0.735=0.946)	0.005
GCS	0.083	0.112	1.086 (0.800=0.983)	0.459
ISS	-0.120	0.053	0.887 (0.784=0.954)	0.022
Lactate	0.032	0.710	1.033 (0.257=4.151)	0.964
CRP	0.060	0.063	1.062 (0.939=1.202)	0.339
Albumin	0.009	0.113	1.009 (0.809=1.259)	0.934
LAR	0.524	18.564	1.689 (0.000=1070.000)	0.977
CAR	-1.633	1.737	0.195 (0.006=5.877)	0.347

APACHE II: acute physiology and chronic health evaluation, GCS: Glasgow Coma Scale, GOS: Glasgow Outcome Scale, ISS: injury severity score, CRP: C-reactive protein, LAR: lactate/ albumin ratio, CAR: C-reactive protein/albumin ratio; P<0.05 was considered statistically significant

Discussion

As a result of our study, APACHE II and ISS were independent risk factors for mortality in TBI, while CAR and LAR were not independent variables in terms of mortality. Similar findings were valid for GOS. In addition, LAR levels were significantly higher in patients with ex. In patients who died; hypertension, high APACHE II score, low GCS, high ISS, mechanical ventilation requirements and vasopressor requirements at first admission to ICU (P=0.000), high lactate level, low albumin levels were meaningful of the results.

Nowadays, due to the increase in traffic accidents, wars, falls, and legal cases, etc., an increasing number of patients with TBI are applying to the emergency department. The incidence of TBI increases in infancy and early childhood (0-4 years; birth traumas, and traffic accidents, etc.), adolescence and early adulthood (15-24 years; motor vehicle accidents, legal cases, and wars, etc.), and in patients over 65 years of age (falls, etc.) [8,9]. TBI clinical; results in a spectrum from very mild findings to deep coma and death. There are two stages in the pathophysiology of brain damage in TBI. Primary damage is related to the damage caused by trauma. Secondary damage developing disruption of the blood-brain barrier, apoptosis, brain edema, and increased intracranial pressure as a result of release of neurotransmitters such as glutamate and aspartate, inflammatory processes, free radicals, and cytokine etc. Although we tried to examine primary and secondary damage in our study, it is possible to say that the main subject of our study is focused on secondary brain damage in which inflammatory, cytokine, and free radicals play a role, since it is based on parameters such as C-Reactive Protein (CRP), lactate and albumin.

One of the most important causes of mortality in TBI is the severity of trauma. GCS is a widely used parameter as the first and most easily applicable method of determining the severity of trauma. In our study, we found that mortality is associated with low GCS. In fact, most studies and articles contain similar results [10]. Hukkelhoven et al. stated in their article published in 2006 that this is more evident in geriatric patients with low GCS [11]. One of the reasons for this seems to be the increase in comorbid diseases with advanced age and the increase in

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the use of anticoagulants and antiplatelet (due to coronary artery disease, arrhythmias, previous ischemic brain damage, etc.) [12,13]. However, our clinical experience shows us that there may be a possibility of partial or complete recovery after surgery, especially in patients who have the chance of surgical intervention (less likely after pharmacological treatment), despite low GCS at first presentation. Injury Severity Score is a scoring system generally used to show the degree of injury in patients with multiple traumas and basically indicates injuries in six regions (head-neck, face, chest, abdomen and extremities). Injury Severity Score (ISS) ≥16 and above is associated with mortality. However, its predictive value on mortality is controversial. Cassidy et al.'s 2014 study showed that although it was more successful in patients with ISS<16, its mortality effectiveness was weak in patients with ISS≥16 [14]. Similarly, Colnaric et al. reported similar results and stated that GCS showed a weak correlation in trauma patients [15]. In our study, patients who died had low GCS and high ISS. However, ISS was found to be a significant independent risk factor, while GCS was not. Due to the multi-trauma patients in the study, the factor of trauma accompanying other organs and systems in addition to TBI may have been a factor in the more significant result of ISS.

In TBI patients, the intensive care process, intubation at first presentation, and the need for vasopressors are factors affecting mortality due to the effect of the primary injury. Our data in our study also support this. In addition to the possible risk of infection in the intensive care unit, concomitant pneumonia associated with ventilators (PAV); the effects of inflammatory factors, cytokines and free radicals, and hypoxia/tissue hypoxia that may occur with pneumonia are factors that increase secondary brain damage. In their research with 119 paediatric cases in 2016, Hamele et al. showed that the mortality rate was higher in patients who developed VAP [16]. The presence of hypotension depending on the magnitude of the trauma, the possibility of disruption of the blood-brain barrier, and the inability to affect blood supply and therefore the nutrition of the brain are other important factors affecting secondary brain damage. Likewise, in patients with hypertension, the possibility of an intracranial hemorrhage (ICH) after primary brain damage increases, and/or the possibility of causing a bleeding that was not initially present increases. In this case, it is an expected fact that mortality will increase.

Many clinical and laboratory markers have been tried to be determined in the prediction of mortality in TBI. CPR, Albumin, GCS, ISS, Troponin I, CAR, LAR, IL-15, and serum amyloid A, and peripheral blood carbon dioxide are some of these markers. As we mentioned before, inflammatory reactions are a part of secondary brain damage [17-19]. In general, high CRP and low Albumin are the expected effects. CRP is an acute phase reactant and an indicator of cytokine activity and inflammation. CRP provides prognostic information in cases such as trauma, sepsis, postoperative heart failure. It is also stated that it can be used as an independent marker in ICH and strokes [4,20]. In our study, we obtained CRP and albumin data in patients who died as expected. Studies have shown that in TBI, similar processes are activated as a reactive response to repair tissue damage after the primary injury (sometimes the opposite occurs, causing further damage with consequences such as edema), so CRP increases. Albumin is a negative acute phase reactant and is a parameter closely related to nutrition and liver function. With increasing age, albumin levels may also decrease due to neurodegenerative diseases. In advanced age, liver functions that can be affected by TBI due to falls, increased risk and multiple traumas lead to a decrease in albumin. In addition, VAP due to prolonged intubation due to TBI, other infections and sepsis; increased nutritional needs are other factors that cause low albumin levels. It is also one of the factors contributing to cerebral edema and increased intracranial pressure due to hypoalbuminemia and causes poor prognosis.

One of the main predictions of our study was that CAR could also be an important data in mortality prediction. Unfortunately, studies on the effect of CAR in TBI are few. In a retrospective study conducted by Wang et al. in 2020 on 151 patients, they stated that GCS and CAR could be used as a marker in mortality prediction [21]. Friedrich et al.'s study in 2024 also suggested that CAR (>0.38) could be used as a marker in terms of prognosis, similar to Wang's study [4]. However, in our study, although low CRP and albumin levels were seen as prognostic markers for mortality, the findings for CAR were in the opposite direction to the studies of Wang and Friedrich. The younger mean age of cases in our study may be the reason for the lower rate of nutritional deficiencies due to neurodegenerative diseases, which are more common in elderly patients. Therefore, although albumin levels were significantly lower in deceased patients than in surviving patients, its effect on the CAR rate may be less. The magnitude of trauma, prevalence of intubation and infection (VAP, etc.), faster mortality, etc. may also be factors affecting our results.

Lactate is considered an indicator of tissue hypoxia and is generally associated with poor prognosis (trauma, sepsis, shock etc.) [21,22]. In their study of 288 paediatric cases presenting with TBI, Fu et al. stated that high lactate levels may be an independent predictor of mortality [23]. Glucose is known to be the main energy source, especially in the brain (via oxidative phosphorylation). In traumatic brain injury, focal or diffuse circulatory disorders due to trauma cause anaerobic respiration and increase lactate. Hypoxia; due to excessive release of neurotransmitters such as aspartate, glutamate, causes disruption of sodium-calcium balance in neurons, and increases free radicals and cytokines. So, this causes cellular damage and triggers secondary brain damage (edema, cell death, etc.) [24]. Epileptic seizures, which may develop especially as a result of trauma, cause increased oxygen consumption in neurons and therefore more neuronal damage. Another reason for increased lactate in TBI due to multiple traumas are lung injury, intubation, infection, fever (cerebral or infectious), extremity injuries (arterial injury, compression syndrome, etc.). Increased hypoxia and/or tissue oxygen demand may also cause increased lactate. Indeed, in our study, lactate was significantly higher in deceased patients. However, there are also studies showing that high lactate levels (due to it can be

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used as secondary energy) increase the chance of survival in isolated TBI, especially despite low GCS, and that hypertonic sodium lactate can be used in the treatment [25-27]. Although lactate was seen as a determining factor for mortality in TBI due to multiple traumas, it was not found to be significant in isolated TBI in the studies conducted by Dübendorfer and his colleagues. This shows that the use of lactate as a secondary energy may be beneficial in brain functions, but it is a clear fact that uncertainty still continues [28].

Unfortunately, publications on the use of LAR as a prognostic marker in patients with TBI are quite insufficient. The other main hypothesis of our study was that LAR could also be used as a prognostic and mortality determinant in TBI. In the data we obtained, LAR was high in patients with ex. It was also suggested that it would be an independent variable in the regression analysis. In their study conducted by Wang et al. in 2022 on 273 patients, they found LAR, ICH (cranial, parenchymal), diffuse axonal injury, and GCS as independent variables [29]. In a retrospective analysis of 460 patients, Lee et al. showed that LAR in the first 24 hours may be a prognostic marker [7]. One of the important risk factors for mortality in patients with TBI is infections and/or sepsis during the intensive care process. Although they did not differentiate patients with TBI in their study, Çakir et al. showed in their study that LAR is a stronger marker than lactate and albumin in infections and sepsis developing during the intensive care process [30]. In our study, although LAR, lactate, and albumin values were similar to those in a small number of similar studies in non-surviving TBI patients, we did not consider LAR to be a parameter that could be evaluated as an independent variable. The inclusion of all TBI patients, including those with minor head trauma, factors in the intensive care process (length of stay, intubation, infection, etc.), the fact that the patients in our study were relatively younger and therefore had a lower probability of comorbid factors increasing with age; may have influenced our findings. However, it is a fact that studies on the role of LAR in patients with TBI are quite insufficient and this situation increases the possibility of obtaining different results.

Limitation

Since our study was discussion a single-center study, the number of patients was not at the desired level. In addition, the limitations created for the optimization of the study, especially the exclusion of pediatric cases from the study, were among the reasons for the low number of patients. These reasons cause limitations in the analysis of the obtained results.

Conclusion

As a result of our study, APACHE II and ISS were independent risk factors for mortality in TBI, while CAR and LAR were not independent variables in terms of mortality. Similar findings were valid for GOS. In addition, LAR levels were significantly higher in patients with ex. These results show us that LAR may be a parameter in mortality prediction, although it is not an independent variable. However, more studies are needed.

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

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

Ethical Approval
For our study, approval of Samsun University Clinical Research Ethics Committee dated: 17/04/2024, no: 2024/8/11as obtained.

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