ORIGINAL ARTICLE

The association between lactate to albumin ratio and outcomes at early phase in patients with traumatic brain injury

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ABSTRACT

BACKGROUND: The majority of traumatic brain injury (TBI) cases result in death in the early phase; predicting short-term prognosis of affected patients is necessary to prevent this. This study aimed to examine the association between the lactate-to-albumin ratio (LAR) on admission and outcomes in the early phase of TBI.

METHODS: This retrospective observational study included patients with TBI who visited our emergency department between January 2018 and December 2020. TBI was considered as an head abbreviated injury scale (AIS) score of 3 or higher and other AIS of 2 or lower. The primary and secondary outcomes were 24-h mortality and massive transfusion (MT), respectively.

RESULTS: : In total, 460 patients were included. The 24-h mortality was 12.6% (n=28) and MT was performed in 31 (6.7%) patients. In the multivariable analysis, LAR was associated with 24-h mortality (odds ratio [OR], 2.021; 95% confidence interval [CI], 1.301–3.139) and MT (OR, 1.898; 95% CI, 1.288–2.797). The areas under the curve of LAR for 24-h mortality and MT were 0.805 (95% CI, 0.766–0.841) and 0.735 (95% CI, 0.693–0.775), respectively.

CONCLUSION: LAR was associated with early-phase outcomes in patients with TBI, including 24-h mortality and MT. LAR may help predict these outcomes within 24 h in patients with TBI.

Keywords: Albumin; lactate; massive transfusion; mortality; prognosis; traumatic brain injury.

INTRODUCTION

Traumatic brain injury (TBI) adds considerable economic expenditure to social health and patient caregivers because of its high mortality and complications.[1,2] Deaths caused by TBI peak at 6–24 h after trauma.[3] Many effective trauma scores have been developed to quickly identify the extent of injury, perform appropriate triage, provide proper treatment, and help predict prognosis. The most commonly used tools for trauma, including TBI, are the injury severity score (ISS) and revised trauma score.[4,5] However, prognostic prediction based on these factors is not clear for TBI, which has a high mortality within 24 h.

Lactate is a simple laboratory test that can accurately reflect hypoperfusion state. Several studies have shown that it is helpful to predict mortality from critical illnesses, such as trauma and sepsis.[6,7] However, lactate level can be affected by convulsions, thiamine deficiency, medical state, and social state.[8] There were studies using the lactate-to-albumin ratio (LAR) to overcome the limitations of lactate. In studies involving sepsis and cardiac arrest, LAR was more helpful than lactate alone in predicting prognosis.[9,10] However, studies for association between LAR and the prognosis of TBI at early phase are lacking.

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Therefore, we hypothesized that LAR is associated with outcome at early phase, including 24-h mortality and massive transfusion (MT) in patients with TB. This study aimed to analyze and compare the prognostic performances of LAR and lactate levels in the early phase of TBI.

MATERIALS AND METHODS

Study Design and Population

We conducted a retrospective observational study of patients with severe trauma and TBI admitted Chonnam National University Hospital between January 2018 and December 2021. Patients with TBI were defined as those with a head injury with an abbreviated injury scale (AIS) score of 3 or higher or another AIS of 2 or lower.[11] Severe trauma was defined as an ISS 16 points or higher. Patients under the age of 18 years; those with cardiac arrest after trauma, prior to arrival at the emergency department (ED); those who underwent special trauma mechanisms, such as drowning or hanging; and those with missing data were excluded from the study. This study was approved by the Institutional Review Board of our hospital. As this was a retrospective study, the requirement for informed consent was waived.

Data Collection

The following clinical variables were obtained from each patient from electronic medical records (EMRs): age, sex, trauma mechanism, systolic blood pressure (mmHg), respiratory and pulse rate, body temperature (BT, °C), and oxygen saturation on admission, initial Glasgow Coma Scale (GCS) score, transfusion amount of packed red blood cells (PRC) during the first 24-h after admission, in-hospital mortality, and 24-h mortality. Laboratory results, including albumin and lactate levels, were obtained at the time of arrival at the ED. The L-lactate level was measured using an enzymatic method with an AU5800 automated chemistry analyzer (Beckman Coulter, Brea, CA, USA). The AIS and ISS were calculated using information from the EMRs. LAR was calculated as the ratio of lactate level to albumin level. MT was defined as transfusion of >10 units of PRCs within the first 24 h of admission or >4 units within 1 h of admission.[12] The primary outcome was 24-h mortality, and the secondary outcome was MT.

Statistical Analysis

The continuous variables that did not show a normal distribution were analyzed using the Mann–Whitney U-test. Fisher's exact test or the Chi-square test was used to compare the categorical variables. The continuous variables are presented as median values with interquartile ranges, and categorical variables are presented as frequencies and percentages. We conducted a multivariate analysis using logistic regression of relevant covariates for 24-h mortality and MT. Variables with P<0.10 in univariate comparisons were included in the multivariate regression model. We used a backward stepwise approach, sequentially eliminating variables with a threshold of P>0.10 to build a final adjusted regression model. Logistic

Table 1. Comparison of baseline characteristics of patients with traumatic brain injury according to 24-h mortality

Variables	TBI patients (n=460)	Survivors (n=432)	Nonsurvivors (n=28)	P-value
Age (years)	64.0 (51.0–74.1)	63.6 (51.0–74.1)	67.5 (52.6–77.6)	0.251
Male, n (%)	343 (74.6)	318 (73.6)	25 (89.3)	0.105
Mechanism of trauma, n (%)				
Blunt	457 (99.3)	429 (99.3)	28 (100.0)	1.000
Penetrating	3 (0.7)	3 (0.7)	0	
GCS score	13 (6–15)	13 (7–15)	3 (3–6)	<0.001
SBP (mmHg)	130 (110–150)	130 (110–150)	120 (100–160)	0.580
Respiratory rate/min	20 (20–20)	20 (20–20)	20 (20–24)	0.240
Pulse rate/min	84 (74–98)	84 (74–96)	93 (68–109)	0.154
Body temperature (°C)	36.4 (36.1–36.8)	36.4 (36.2–36.8)	36.0 (35.9–36.4)	<0.001
Lactate (mmol/L)	2.5 (1.6–4.2)	2.4 (1.6–4.0)	5.2 (3.6–8.1)	<0.001
Albumin (g/dL)	3.9 (3.5–4.1)	3.9 (3.5–4.2)	3.5 (3.0–4.0)	0.005
LAR	0.65 (0.42–1.16)	0.62 (0.41–1.06)	1.58 (0.93–2.78)	<0.001
ISS	20 (16–25)	20 (16–25)	25 (20–25)	0.016
PRC	0 (0–2)	0 (0–2)	0 (0–3)	0.304
MT, n (%)	31 (6.7)	25 (5.8)	6 (21.4)	0.005

TBI: Traumatic brain injury; GCS: Glasgow Coma Scale; SBP: Systolic blood pressure; LAR: Lactate-to-albumin ratio; PRC: Packed red blood cell; MT: Massive transfusion; ISS: Injury severity score.

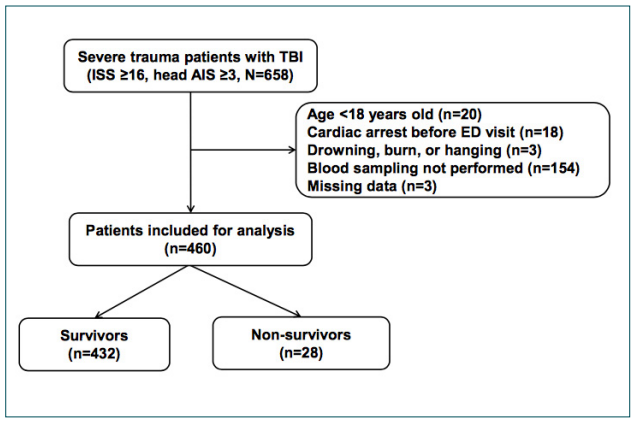

Figure 1. Schematic diagram showing the number or patients severe trauma included in the present study. TBI: traumatic brain injury, ISS: injury severity score, AIS: abbreviated injury scale, ED: emergency department

regression analysis results are presented as odds ratios (OR) and 95% confidence intervals (CI). Receiver operating characteristic (ROC) analysis was used to examine the prognostic performance of lactate and albumin levels and LAR for 24-h mortality and MT. The comparison of dependent ROC curves was performed using the DeLong et al. method.[13] All analyses were performed using PASW/SPSS™ software, version 18 (IBM Inc., Chicago, IL, USA) and MedCalc, version 19.0 (MedCalc Software, bvba, Ostend, Belgium). We performed a post-hoc power analysis using g*power. A two-sided significance level of 0.05 was defined as statistically significant.

RESULTS

Patient Selection and Baseline Characteristics

A total of 658 patients with severe trauma who met the inclusion criteria were identified during the study period. After removing data from patients based on the exclusion criteria, data from 460 patients were included in the study (Fig.1). The median age of the patients was 64.0 (51.0–74.1) years, and the median ISS was 20 (16-25). The 24-h mortality was 12.6% (n=28), and MT was performed in 31 (6.7%) patients. Non-survivors were older, had lower GCS scores and BT, and a higher ISS than survivors (Table 1). Non-survivors had higher lactate levels and LAR and lower albumin levels than survivors. A higher proportion of non-survivors underwent MT than survivors. The patients who underwent MT had higher lactate levels and LAR and lower GCS scores and albumin levels than those who did not (Table 2). Post hoc power analyses for 24-h mortality and MT had representative powers of 0.99 and 0.98, respectively.

Multivariate Analysis using Logistic Regression for 24-h Mortality and Massive Transfusion

Table 3 shows the results of the multivariate analysis for 24-h mortality and MT. GCS score (OR, 0.762; 95% CI, 0.662–0.877), BT (OR, 0.398; 95% CI, 0.182–0.869), and LAR (OR, 2.021; 95% CI, 1.301–3.139) were independently associated with 24-h mortality. GCS score (OR, 0.906; 95% CI, 0.833–0.987) and LAR (OR, 1.898; 95% CI, 1.288–2.797) were independently associated with MT.

Table 2. Comparison of baseline characteristics of patients with traumatic brain injury according to massive transfusion

Variables	No MT (n=429)	MT (n=31)	P-value
Age (years)	64.0 (52.0–74.1)	56.0 (38.0–74.1)	0.194
Male, n (%)	320 (74.6)	23 (74.2)	1.000
Mechanism of trauma, n (%)			
Blunt	426 (99.3)	31 (100.0)	1.000
Penetrating	3 (0.7)	0	
GCS score	13 (6–15)	5 (3–13)	<0.001
SBP (mmHg)	130 (110–150)	130 (100–150)	0.360
Respiratory rate/min	20 (20–20)	20 (20–24)	0.204
Pulse rate/min	84 (74–96)	88 (76–108)	0.252
Body temperature (°C)	36.4 (36.1–36.8)	36.4 (36.0–36.5)	0.044
Lactate (mmol/L)	2.4 (1.6–4.0)	4.7 (3.2–6.0)	<0.001
Albumin (g/dL)	3.9 (3.5–4.1)	3.6 (2.8–4.2)	0.023
LAR	0.63 (0.41–1.08)	1.33 (0.77–2.13)	<0.001
ISS	20 (16–25)	24 (17–25)	0.269
PRC	0 (0–1)	8 (5–11)	<0.001
24-h mortality, n (%)	22 (5.1)	6 (19.4)	0.005

MT: Massive transfusion; LAR: Lactate-to-albumin ratio; PRC: Packed red blood cell; GCS: Glasgow Coma Scale; SBP: Systolic blood pressure; ISS: Injury severity score.

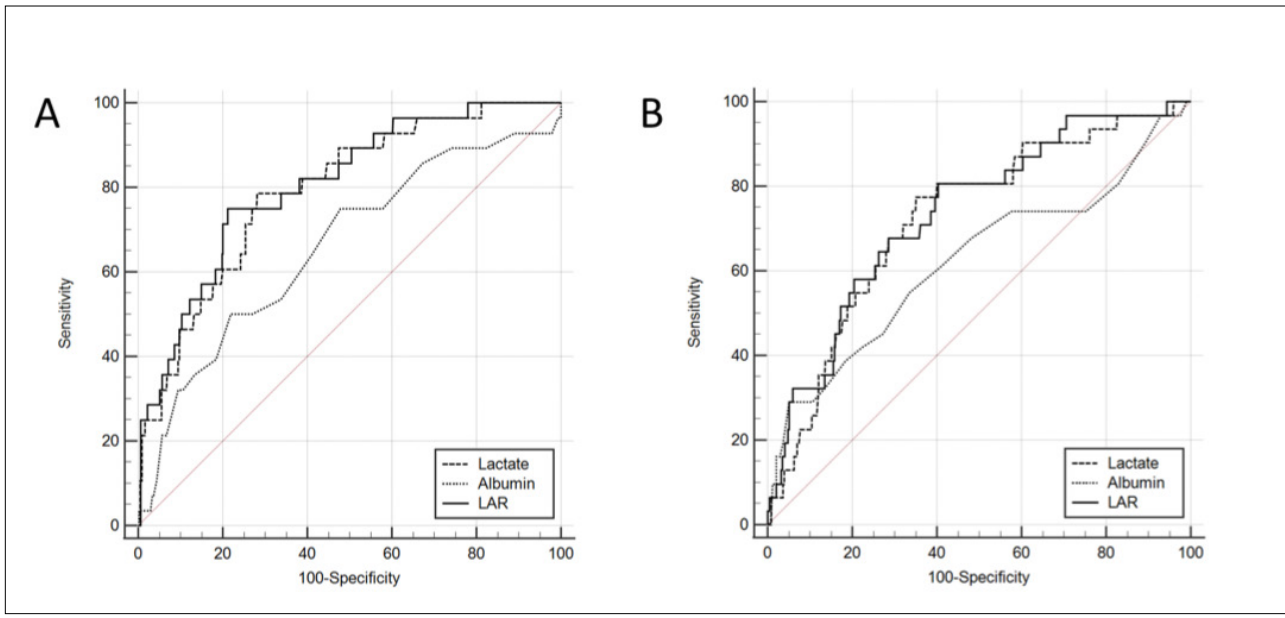

Figure 2. Receiver operating characteristics curve analyses of the lactate, albumin and LAR for predicting 24-h mortality **(a)** and massive transfusion **(b)** in patients with TBI. The AUCs of lactate, albumin, and LAR for 24-mortality were 0.791 (95% CI, 0.751–0.828), 0.659 (95% CI, 0.613–0.702), and 0.805 (95% CI, 0.766–0.841), respectively. The AUCs of lactate, albumin, and LAR for massive transfusion were 0.723 (95% CI, 0.680–0.764), 0.622 (95% CI, 0.576–0.666), and 0.735(95% CI, 0.693–0.755), respectively. LAR: lactate-to-albumin ratio, AUC: area under the curve, CI: confidence intervals.

Receiver Operating Characteristic Analysis of Lactate-to-albumin Ratio for 24-h Mortality and Massive Transfusion

The areas under the curve (AUCs) of lactate and albumin levels and LAR for 24-h mortality were 0.791 (95% CI, 0.751–0.828), 0.659 (95% CI, 0.613–0.702), and 0.805 (95% CI, 0.766–0.841), respectively (Fig. 2a). The AUC of LAR for 24-h mortality was significantly different from that of the albumin level but not from that of the lactate level. The AUCs of lactate and albumin levels and LAR for MT were 0.723 (95% CI, 0.680–0.764), 0.622 (95% CI, 0.576–0.666), and 0.735 (95% CI, 0.693–0.775), respectively (Fig. 2b). The AUC of LAR for MT was not significantly different from those of albumin or lactate levels.

Table 3. Multivariate logistic regression analysis for predicting 24-h mortality and massive transfusion

Variables	AOR (95% CI)	P-value
For 24-h mortality		
GCS score	0.762 (0.662–0.877)	<0.001
BT (°C)	0.398 (0.182–0.869)	0.021
LAR	2.021 (1.301–3.139)	0.002
ISS	1.060 (0.963–1.166)	0.233
For MT		
GCS score	0.906 (0.833–0.987)	0.023
BT (°C)	0.787 (0.404–1.531)	0.480
LAR	1.898 (1.288–2.797)	<0.001

GCS: Glasgow Coma Scale; BT: Body temperature; MT: Massive transfusion; AOR: Adjusted odds ratio; CI: Confidence interval; LAR: Lactate-to-albumin ratio; ISS: Injury severity score.

DISCUSSION

In this study, LAR was independently associated with 24-h mortality and MT, and it showed fair performance in predicting these outcomes in patients with isolated TBI. The association between lactate levels and TBI outcomes is controversial. One study showed that patients with lactate levels >5 mmol/L are more likely to have a normal cognitive status at hospital discharge than those with lactate levels ≤5 mmol/L.[14] In one randomized controlled trial, the decrease in intracranial pressure in patients who received lactate solution was more pronounced than that in patients who received mannitol.[15] Meanwhile, the lactate levels of non-survivors were higher than those of survivors in this study. In this study, the lactate level of the non-survivors was 5.2 mmol/L, which was higher than that of the survivors. We collected the lactate levels measured on admission, and it is believed that damaged neurons have a decreased ability to select and metabolize increased lactate in the early phase of severe TBI. In one experimental study, severe TBI conclusively induced a lactate storm in a failed metabolic environment.[16] A previous study reported a comparable relationship between lactate levels and mortality rates in a group of pediatric patients with TBI.[17] With a lactate level of 3.3 mmol/L as the cutoff, high lactate levels at admission showed good performance in predicting in-hospital mortality in patients with cerebral gunshot injury.[18]

Secondary injury after TBI, including neuroinflammation, can contribute to disruption of the blood–brain barrier (BBB).[19] Increased vascular permeability and BBB dysfunction after TBI induce leakage of albumin and fluids as well as the transmigration of immune cells.[19,20] Subsequently, albumin leakage degrades the basement membrane, further increases BBB permeability, and induces vasogenic edema.[19] Activation of myosin light-chain kinase in astrocytes aggravates BBB damage and deteriorates vascular barrier integrity.[21] In a retrospective study of TBI, the albumin level of patients with poor neurological outcome was found to be lower than that of patients with good neurological outcome, similar to the results of the present study.[22] In this study, the albumin level of the non-survivors was 3.5 mmol/L, which was lower than that of the survivors. High lactate and low albumin levels were associated with poor prognosis in patients with TBI in the present study; thus, LAR appears to reflect the severity of TBI. This study showed that the LAR of the non-survivors was 1.58, which was higher than that of the survivors. Moreover, the adjusted OR of the LAR for 24-h mortality was 2.201 (CI, 1.301–3.139).

Elevated lactic acid levels are associated with a variety of clinical symptoms and disease states. The causes of this lactate elevation include seizures, various medications (metformin, isoniazid, salicylates), toxins (alcohol, carbon monoxide, cyanide), thiamine deficiency, and smoking inhalation.[8] Furthermore, albumin levels are easily affected by nutritional imbalances, liver disease, and kidney disease. Given that several factors can influence both lactate and albumin levels, the LAR can be used as a reliable prognostic tool for TBI. Specifically, it has been used to measure mortality in TBI. In one study, there was a correlation between LAR and mortality at the time of admission.[23] In that study, the relationship between lactate level and in-hospital mortality was investigated; however, in our study, the acute phase prognosis was investigated in terms of 24-h mortality rate and the use of MT. In this study, the LAR of patients who received a MT was 1.33, which was higher than that of the survivors. Additionally, the adjusted OR of LAR for MT was 1.898 (CI 1.288–2.797). In severe TBI, up to 30% of patients die within the first 24 h of admission.[24] Furthermore, the average age of the survivors and non-survivors in that study was 43 years. The ages of survivors and non-survivors in our study were 63.6 and 67.5 years, respectively. This was similar to the average age of 65 years for patients requiring hospitalization for intensive care for TBI in the United States.[25]

A number of studies have shown an association of GCS with mortality in patients with TBI, which was also independently relevant in our study. In a study by Han et al., a GCS score ≤5 was associated with mortality from TBI, and in the present study, the GCS score of the non-survivors was 4 (3–9).[26] However, there are some limitations to measuring GCS. The verbal GCS scores vary and are affected by alcohol or sedative use, which may affect the state of the patient's consciousness.

The present study had some limitations. First, it had a retrospective design and was conducted at a single center. Therefore, the results cannot be immediately generalized to the overall population. Additional prospective multicenter studies are needed to confirm our results. Second, other inflammatory markers, such as cytokines and chemokines, were not investigated in this study. Further studies including these inflammatory markers are needed. Third, drugs, such as vasopressors, to improve cerebral perfusion after TBI and prevent secondary ischemic injury, and norepinephrine, to increase the production of pro-and anti-inflammatory cytokines are generally used during treatment. However, the effects of vasopressors, including norepinephrine, were not sufficiently considered in this study. Fourth, we did not investigate whether albumin was replaced according to the albumin level and whether the replaced albumin affected the clinical outcome of patients with PCAS.

Conclusion

LAR is associated with the early-phase outcomes of TBI, including 24-h mortality and MT. An early prognosis through LAR can be used to select severely ill patients, facilitate emergency surgery and blood transfusion, and eventually improve the prognosis of TBI patients.

Ethics Committee Approval: This study was approved by the Chonnam National University Hospital Clinical Research Ethics Committee (Date: 22.03.2022, Decision No: CNUH-2022080)

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept: D.H.L.; Design: J.H.L., D.H.L.; Supervision: B.K.L., Y.H.J.; Materials: Y.S.C., J.H.L., D.K.K.; Data: J.H.L., D.K.K., S.J.R.; Analysis: S.J.R., D.K.K., E.N.; Literature search: S.J.R., D.K.K., E.N.; Writing: J.H.L., D.H.L.; Critical revision: D.H.L., B.K.L., Y.H.J.

Conflict of Interest: None declared.

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ORİJİNAL ÇALIŞMA - *ÖZ*

Travmatik beyin yaralanması (TBY) hastalarında laktat/albümin oranı ile erken dönemdeki sonuçlar arasındaki ilişki

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AMAÇ: Travmatik beyin yaralanması (TBY) vakalarının çoğu erken dönemde ölümle sonuçlanır; etkilenen hastaların kısa vadeli prognozunu tahmin etmek bu durumu engellemek için gereklidir. Bu çalışma, TBY'nin erken döneminde kabul anındaki laktat/albümin oranı (LAO) ile sonuçlar arasındaki ilişkiyi incelemeyi amaçlamaktadır.

GEREÇ VE YÖNTEM: Bu retrospektif gözlemsel çalışma, Ocak 2018 ile Aralık 2020 tarihleri arasında acil servisimize başvuran TBY'li hastaları içermiştir. TBY, baş kısaltılmış yaralanma skalası (AYS) skoru 3 veya daha yüksek ve diğer AYS skoru 2 veya daha düşük olarak kabul edildi. Birincil ve ikincil sonuçlar sırasıyla 24 saatlik mortalite ve masif transfüzyon (MT) idi.

BULGULAR: Toplam 460 hasta dahil edildi. 24 saatlik mortalite %12.6 (n=28) ve MT 31 (%6.7) hastada gerçekleştirildi. Çok değişkenli analizde, LAO'nun 24 saatlik mortalite (odds oranı [OO], 2.021; %95 güven aralığı [GA], 1.301-3.139) ve MT (OO, 1.898; %95 GA, 1.288-2.797) ile ilişkili olduğu bulundu. LAO'nun 24 saatlik mortalite ve MT için eğri altında kalan alan değerleri sırasıyla 0.805 (%95 GA, 0.766-0.841) ve 0.735 (%95 GA, 0.693-0.775) idi.

TARTIŞMA: LAO, TBY'li hastalarda erken dönem sonuçlarıyla, 24 saat içinde 24 saatlik mortalite ve MT dahil olmak üzere ilişkili bulunmuştur. LAO, TBY'li hastalarda bu sonuçları tahmin etmede yardımcı olabilir.

Anahtar sözcükler: Mortalite, Masif transfüzyon, Prognoz, Laktat, Albümin, Travmatik beyin yaralanması

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