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The Role Of Inflammatory Biomarkers NLR And PLR In Predicting Functional Outcomes Following Traumatic Brain Injury

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ABSTRACT

Background: Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality among trauma patients in developing countries. The post-traumatic inflammatory response plays a critical role in worsening patient outcomes. Routine hematological examinations such as complete blood count can provide information of neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR). Both markers have reported as simple and relatively inexpensive inflammatory markers. Identification of these biomarkers is expected to assist in predicting outcomes, particularly through assessment with the Glasgow Outcome Scale (GOS). This study was aimed to analyze the correlation between NLR and PLR with GOS in TBI patients.

Method: This research was a prospective observational analytic study using consecutive sampling technique at Dr. Soetomo General Hospital Surabaya between March and June 2025. NLR and PLR values were obtained within 24 hours after trauma. Clinical outcome (GOS) was assessed on day 14. Data analysis was performed using SPSS, with $p < 0,05$ considered statistically significant.

Results: Total of 44 patients were included in this study, with the majority of patients being male (63,6%) and median age of 32,5 years. The most common clinical outcome was GOS 5 (good recovery, 54,5%), while rate mortality was 27,3%. Median value of NLR was 8,35 and mean value of PLR was 170,45. Statistical analysis revealed no significant correlation between NLR and GOS ($p = 0,338$). In contrast, PLR showed a moderate positive correlation with GOS ($r = 0,527$, $p < 0,001$), with cut-off value of 101,24.

Conclusion: NLR was not associated with GOS in TBI patients, whereas PLR showed a significant correlation with better clinical outcomes. PLR may serve as a simple biomarker to predict prognosis in TBI patients.

KEYWORDS: Glasgow Outcome Scale, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, traumatic brain injury.

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INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of morbidity and mortality in trauma patients, especially in developing countries. The incidence rate of TBI in Indonesia reached 189 cases per 100,000 people in 2019, with mortality rate of 37,14% due to severe brain injury.1,2 Main causes of TBI are traffic accidents and falls.3 Lack of access to healthcare and rehabilitation services can worsen clinical outcomes, necessitating methods to predict the early prognosis of TBI patients.4,5 Currently, initial level of consciousness is associated with patient prognosis. While Glasgow Coma Scale (GCS) is commonly used as standard of level of consciousness measurement, it has limitations due to interobserver variability.6,7 Thus, objective biomarkers are increasingly explored for to determine the clinical outcome of TBI patients.8

Post-traumatic brain injury inflammation plays a critical role in determining outcomes. Local inflammation in injured brain tissue induce inflammatory mediators release such as proinflammatory cytokines, which subsequently induce recruitment of immune cells, including leukocytes, to injured area. Disrupted blood–brain barrier allows neutrophil infiltration, amplifying neuroinflammation.9 Neutrophil activation and decreased lymphocyte counts can lead to an imbalance in immune response, ultimately increasing the risk of secondary brain tissue damage. This dysregulation of inflammatory system contributes to cerebral edema and increased intracranial pressure, which can worsen patient outcome.10 Otherwise, platelet also contribute in hemostasis, and overreactivity of platelet aggregation worsen inflammation reaction. Report showed direct connection of low platelet count with multiple organ dysfunction on trauma patient.11 Simple biomarkers from routine hematology tests, such as neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR), can be alternative indicators of inflammation as predictors of patient outcomes.9,10,12,13

Clinical outcomes in TBI patients can be assessed using a scoring system such as Glasgow Outcome Scale (GOS). This assessment was first used to assess survival and quality of life in patients after severe brain damage. The advantages of this scoring system

are its simplicity, ease, flexibility, and short timeframe.14 Therefore, this study aims to examine the relationship between NLR and PLR with GOS in patients with TBI.

METHOD

This was a prospective cohort study. Selected patients were all degree of TBI who admitted to emergency department of Dr. Soetomo General Hospital Surabaya from March 2025 to June 2025. Inclusion criterias were : (1) There was a clear history of injury, and the time of injury was within 24 h; (2) All patients were confirmed by head CT at emergency department. Exclusion criteria were: (1) Patients also are diagnosed with heart failure, renal failure, pregnancy, tumor, blood system disease; (2) Patients taking drugs that may affect platelet and lymphocyte count and function (antiplatelet aggregation drugs, glucocorticoids, contraceptives etc.); (3) Patients whose receive blood transfusion from referral hospital.

Outcome of this study was short-term Glasgow Outcome Scale on day 14 after trauma. CT was performed immediately at emergency department. The GCS score was determined by at least two physicians after admission. Baseline characteristics of the patients included age, gender, GCS score, neurosurgery, neutrophil, platelet and lymphocyte count within 24 hours after trauma. Blood was drawn immediately after admission. GOS score was divided into five groups: 1 dead, 2 persistent vegetative state, 3 severe disability, 4 moderate disability, and 5 good recovery.

Data analysis was performed using the Statistics Package for Social Science (SPSS v. 23, IBM Corp., Armonk, NY, USA). A two-tailed Kolmogorov-Smirnov test was applied to examine whether the continuous quantitative variables. Characteristics of patients, as n (percent) or median (minimum-maximum) for categorical and continuous variables, respectively, were reported. Nominal variables were compared using a two-tailed Chi-square or Fisher's exact test, when applicable. The "Mann-Whitney U" test was used in the comparison of the measurement values of two independent groups; the "Kruskal-Wallis H" test was used in the comparison of the measurement values of three or more independent groups. The statistical significance of differences between groups was determined by the Kruskal-Wallis test followed by post hoc Mann-Whitney test. The p-value was set at <0.05 for statistical significance.

This study was approved by the ethics committee of Dr Soetomo General Hospital Surabaya and in accordance with the Declaration of Helsinki. All patients and their legally authorized representative signed written informed consent on admission.

RESULT

Forty-four TBI patients were included, with 63.6% male and a median age of 32.5 years. The most frequent mechanism was road traffic accidents. Most injuries were mild to moderate in severity. Good recovery (GOS 5) was achieved in 54.5%, with 27.3% mortality within 14 days after trauma.

Median of NLR was 8.35 (range 0.74–26.91); mean PLR was 170.45 ± 89.68. No statistically significant association was found between NLR and GOS ($p = 0.338$). In contrast, PLR was significantly correlated with GOS ($r = 0.527$, $p < 0.001$). ROC analysis yielded an AUC of 0.74 for PLR, indicating moderate prognostic value, with the optimal cut-off at 101.24 (sensitivity 93.3%, specificity 64.3%). Patients with PLR above this threshold had significantly better survival and functional outcomes.

Table 1. The baseline clinical characteristics of the study.

Characteristic	N(%) or Median (Min – Max)
Age	32,5 (18 – 64)
Sex	
Male	28 (63,6%)
Female	16 (36,4%)
Brain Injury	
Mild	15 (34,1%)
Moderate	15 (34,1%)
Severe	14 (31,8%)
GCS	11 (3-15)
Surgical Management	
Surgery	32 (72,7%)
Conservative	14 (27,3%)
GOS	
Death	12 (27,3%)
Vegetative state	2 (4,5%)
Severe disability	3 (6,8%)
Moderate disability	3 (6,8%)
Good recovery	24 (54,5%)

DISCUSSION

This study provide valuable insights into the prognostic utility of hematological inflammatory markers in traumatic brain injury, specifically predictive value of platelet-to-lymphocyte ratio (PLR) for short-term functional outcomes. Demographic profile characterized by male predominance (63.6%) and a median age of 32.5 years, is consistent with global epidemiological patterns of TBI, wherein young adult males represent the highest-risk population due to greater exposure to traffic accidents, occupational hazards, and high-risk behaviors. Road traffic accidents remain the leading etiology of TBI in low- and middle-income countries, including Indonesia, where infrastructural limitations and enforcement challenges exacerbate injury rates.15-16

Based on the severity level, the number of patients with mild traumatic brain injury (TBI) was equal to those with moderate TBI, each consisting of 15 patients (34.1%), while severe TBI cases numbered 14 patients (31.8%). These findings align with global reports on traumatic brain injury patients, where moderate and severe cases account for more than 50% of total cases.17-18 Fourteen out of fifteen mild TBI (COR) patients experienced favorable recovery, while among the 14 severe TBI (COB) patients, eight died and two remained in a vegetative state by day 14 post-trauma. This relationship is statistically significant (p=0.003). These results are consistent with a retrospective study in 2020, which found that patients with GCS <8 had higher mortality risk and worse clinical outcomes compared to those with GCS 13-15.19

There were 72.7% samples underwent surgical intervention for TBI management, consistent with Kiansantang and Tobing's (2023) findings that 54.74% of TBI patients underwent craniotomy.20 TBI management strategies are broadly categorized into conservative and surgical approaches. Conservative management includes observation, medical therapies to control intracranial pressure, seizure prophylaxis, and supportive care. Surgical management involves interventions such as decompressive craniotomy, hematoma evacuation, or intracranial pressure monitoring. Treatment decisions are tailored based on clinical condition, radiological findings, and signs of clinical deterioration, with surgery most often performed on severe TBI patients.21 Decompressive craniotomy is associated with reduced mortality, more effective intracranial pressure reduction, and shorter hospital stays in moderate to severe TBI patients compared to conservative management. Although functional outcome improvements did not significantly differ between surgical and conservative groups, surgery remains the primary treatment choice, especially when supported by worsening clinical or radiological signs.

Table 2. Distribution of demographic and clinical findings by Glasgow outcome scale.

Characteristics (N = 44)	***Glasgow Outcome Scale*** **n (%) atau median (min – max)**					**Total**	**P value**
	1	**2**	**3**	**4**	**5**		
Sex							0,491
Male	7 (25%)	1 (3,6%)	3 (10,7%)	3(10,7%)	14 (50%)	28 (100%)	
Female	5 (31,3%)	1 (6,3%)	0 (0%)	0 (0%)	10 (62,5%)	16 (100%)	
Brain Injury							**<0,001**
Mild	1 (6,7%)	0 (0%)	0 (0%)	0 (0%)	14 (93,3%)	15 (100%)	
Moderate	3 (20%)	0 (0%)	2 (13,3%)	2 (13,3%)	8 (53,3%)	15 (100%)	
Severe	8 (57,1%)	2 (14,3%)	1 (7,1%)	1 (7,1%)	2 (14,3%)	14 (100%)	
Surgical Management							0,086
Surgery	11 (34,4%)	2 (6,3%)	3 (9,4%)	3 (9,4%)	13 (40,6%)	32 (100%)	
Conservative	1 (8,3%)	0 (0%)	0 (0%)	0 (0%)	11 (91,7%)	12 (100%)	
Leukocyte ($x10^3$/µl)	19,59 (8,33 – 39,05)	25 (15,46-34,53)	13,24 (13,14 – 17,2)	18,15 (18,12 – 22,25)	15,15 (8,19 – 41,04)		0,275
Neutrophil ($x10^3$/µl)	16,38 (4,57 – 35,41)	17,57 (13,41– 21,73)	11,22 (9,45 – 15,35)	16,23 (14,7 – 18,32)	12,38 (6,87 – 33,67)		0,551
Lymphocyte ($x10^3$/µl)	2,40 (0,54 – 6,34)	6,03 (0,88 – 11,19)	1,09 (0,82 – 2,75)	1,4 (0,97 – 1,73)	1,36 (0,57 – 5,75)		0,165
Platelet ($x10^3$/µl)	280 (145 – 373)	264 (223 – 305)	208 (188 – 284)	215 (206 – 342)	238,5 (147 – 395)		0,946

Table 3. Distribution of NLR and PLR by Glasgow Outcome Scale.

Characteristics (N = 44)	***Glasgow Outcome Scale*** **n (%) atau median (min – maks)**					**P value**	**Death + PVS**	**Alive**	**P value**
	1	**2**	**3**	**4**	**5**				

NLR	7,15 (0,74 - 26,57)	8,59 (1,94 – 15,24)	10,29 (3,44 – 18,72)	13,08 (8,5 – 16,73)	8,4 (2,97 – 26,91)	0,674	7,15 (0,74 – 26,57)	8,81 (2,97 – 26,91)	0,338
PLR	93,94 (49,21 – 327,78)	140,34 (27,26 – 253,41)	172,48 (103,27- 253,66)	221,65 (119,08- 244,29)	177,32 (50,27 – 385,96)	0,287	126,56 ± 88,27	190,93 ± 84,08	**0,025**

Keterangan : Group 1 death, 2 persistent vegetative state, 3 severe disability, 4 moderate disability, 5 good recovery. Group Death + PVS = GOS 1+2, Group alive = GOS 3+4+5.

This study found elevated leukocyte counts (average 17.43 x 10^3/µl) among all patient groups compared to normal populations. The highest leukocyte counts were found among patients with poor clinical outcomes (GOS 1 and 2); deceased patients had an average of 19.59 x 10^3/µl, and vegetative patients averaged 25 x 10^3/µl. No significant leukocyte count differences were statistically found between groups. Leukocytosis within 24 hours post-injury is driven by increased leukocyte activity and pro-inflammatory cytokines.23 Inflammatory mediators released from injured brain cells recruit leukocytes, contributing to neuroinflammation. Leukocyte counts above 17,500/µl have been linked to worse clinical outcomes and longer hospital stays.24-25

Mean platelet counts across all GOS groups remained within normal ranges (208 x 10^3/µl – 280 x 10^3/µl), with the highest counts observed in deceased patients (280 x 10^3/µl, range 145–373 x 10^3/µl). No significant differences were found among GOS groups for leukocytes, neutrophils, lymphocytes, or platelets, consistent with Baroto26, who found no platelet count differences among five GOS patient groups.

The lowest neutrophil to lymphocyte ratio (NLR) was observed in deceased patients (7.15) compared to other groups. Median NLR in patients with favorable outcomes was higher (8.81). However, statistically, no significant differences were found among groups (p=0.338). NLR values were elevated relative to the normal range (typically 1-2), with pathological values below 0.7 or above 3.10,27 Global standardization of NLR reference values remains lacking, but healthy adult means average around 1.65 (range 0.78–3.53).27-28

These findings align with Cine et al. (2023)30, who reported no significant correlation between NLR and GOS scores at one year in pediatric TBI patients. Initial NLR values, despite reflecting acute inflammation, may not independently predict long-term clinical outcomes. Some prior studies, however, found significant associations between high NLR and poor outcomes.30-31

Leukocytosis due to trauma-induced hypovolemia mobilizes leukocytes (lymphocytes, monocytes, neutrophils) as part of systemic inflammatory response. Elevated lymphocytes may indicate hypovolemia-associated hypotension and cerebral hypoperfusion, while neutrophils relate to inflammation severity.32

NLR reflects the balance between innate and adaptive immunity, influenced by physiologic, pathologic, demographic, pharmacologic, and lifestyle factors.10 This study found no association between NLR and clinical outcome at day 14 post-trauma, potentially due to influencing factors like age, comorbidities, treatments, and trauma type.33 Differences in timing of NLR measurements also affect findings since peak neutrophil recruitment typically occurs 2–4 days after injury.34

There was significant association was found between platelet to lymphocyte ratio (PLR) and clinical outcome (p=0.025). Lower PLR values correlated with poorer outcomes, while moderate positive correlation with GOS at day 14 was observed (r=0.527). Li and Deng reported PLR as an independent biomarker for 30-day mortality in moderate to severe TBI, with low PLR reflecting thrombocytopenia and lymphocytosis due to vascular injury, platelet adhesion, and coagulopathy.12 PLR cut-off value of 101.24 in this study aligns with other findings: Andari et al. (2024)35 reported lower PLR in severe TBI linked to higher mortality risk, while other studies found cut-offs ranging from 65.35 to 142.9 associated with mortality in trauma.32, 36-38 Clinical utility of PLR lies in its simplicity and prognostic relevance, suggesting integration into emergency and intensive care screening protocols to guide early management of TBI patients.

Several limitations of the present study warrant acknowledgment. First, the single-center design and modest sample size (n = 44) may limit generalizability and statistical power, particularly for detecting associations with NLR given its higher intra-individual variability. Second, the short follow-up period (14 days) precludes assessment of long-term functional outcomes, cognitive sequelae, and quality of life, which are critical endpoints for TBI patients and their families. Third, the heterogeneity of intracranial pathology (e.g., diffuse axonal injury, contusions, subdural hematomas) within the cohort may have obscured subgroup-specific associations between biomarkers and outcomes. Fourth, the lack of repeated biomarker measurements limits the ability to capture temporal dynamics and identify peak or nadir values that may have stronger prognostic significance. Future studies should adopt multicenter, prospective designs with larger cohorts, longitudinal biomarker assessments, comprehensive adjustment for confounders, and extended follow-up to validate and refine the prognostic utility of PLR and NLR in diverse TBI populations.

CONCLUSION

NLR was not associated with GOS in TBI patients, whereas PLR showed a significant correlation with better clinical outcomes. PLR may serve as a simple biomarker to predict prognosis in TBI patients.

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