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Predictors of clinical outcomes in traumatic intracerebral haemorrhage: Development of a prognostic scoring model

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Original Article

ABSTRACT

Background: Traumatic intracerebral haemorrhage (tICH) is frequently observed as a form of intracranial bleeding that follows traumatic brain injury (TBI).
Objectives: This study aims to analyse the Glasgow Coma Scale (GCS) score, haemorrhage volume, leukocyte count, and sodium and potassium levels as predictors of clinical outcomes in patients with traumatic intracerebral haemorrhage and to develop a predictive scoring model based on these variables.
Methods: This observational analytic study used a retrospective cohort design in the neurology inpatient room at RSUDZA Banda Aceh between January and March 2025. The study included 80 patients with tICH who met the inclusion criteria. The GCS score, bleeding volume, leukocyte count, and sodium and potassium levels were assessed as predictors. The Glasgow Outcome at Discharge Scale (GODS) was used to evaluate clinical outcomes. A multiple linear regression analysis was used to evaluate the effects of independent variables on clinical outcomes. The model was validated using the Hosmer and Lemeshow tests ($p>0.05$), showing a good fit between the predicted and observed values.
Results: A bivariate analysis indicated significant associations between GCS score, bleeding volume, leukocyte count, sodium, potassium levels, and outcomes ($p<0.05$). A multivariate analysis identified only GCS score ($p=0.010$; OR=10.102) and bleeding volume ($p=0.034$; OR=0.129) as independent predictors. A predictive scoring model was developed from the regression coefficients, with scores ranging from 0 to 7. Higher scores indicate a greater probability of poor outcomes, with a score of 7 corresponding to a 98.7% probability.
Conclusion: This study highlights the GCS score and bleeding volume as key predictors of clinical outcomes in patients with tICH, while leukocyte count and sodium and potassium levels remain relevant supportive factors in the clinical context. Further validation in larger, diverse populations is warranted to assess its applicability in various clinical settings.

INTRODUCTION

Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality in children worldwide.[1] Head injury is a global health problem that has a significant impact on morbidity and mortality rates, especially in young and productive age groups. According to the World Health Organization (WHO), head injury or TBI has become one of the leading causes of long-term disability and high mortality worldwide.[2] In Indonesia, head injury cases still dominate emergency department visits, mainly due to traffic accidents.[3] Head injuries have a wide range of clinical manifestations, ranging from mild to severe, depending on the mechanism of trauma and the location of the brain injury.[4,5]

A severe case of head injury that often leads to serious neurological impairment is traumatic intracerebral haemorrhage (tICH), which is bleeding that occurs within the brain tissue

due to trauma.[6] This condition has the potential to cause increased intracranial pressure, secondary brain tissue damage, and the risk of herniation, which is closely associated with high mortality and disability in both the acute and chronic phases.[4]

Clinical outcomes in patients with tICH are highly varied and difficult to predict from initial symptoms alone; thus, early prognostic assessment is important to prioritize treatment and follow-up interventions.[7] One instrument used is the Glasgow Outcome at Discharge Scale (GODS), which assesses patient outcomes from death to complete recovery. In practice, this scale is often simplified into two categories: favourable outcomes (score 5-8) and unfavourable outcomes (score 1-4), to facilitate analysis and clinical decision-making.[8]

Various clinical, radiological, and laboratory parameters have been investigated to predict the outcomes of patients with severe head injury. The GCS score ≤8 is associated with poor outcomes and increased mortality. Bleeding volumes ≥30 mL are also associated with a high risk of mortality due to increased intracranial pressure and decreased cerebral perfusion.[9] Leucocytosis is found in 40-70% of patients with head injury and reflects systemic inflammation that exacerbates secondary brain injury.[10] Electrolyte disturbances, such as hyponatremia (±30% of cases) and hypokalaemia, also play a role in worsening neurological function and increasing treatment duration.[1] The combination of these variables has been used in multifactorial prediction models and shown to be effective for classifying the risk of poor outcomes in patients with severe TBI.[11]

Numerous clinical, radiological, and laboratory parameters have been investigated to predict patient outcomes in severe head injury cases. A GCS score of ≤8 is strongly associated with adverse outcomes and elevated mortality.[1] The integration of these variables into a multifactorial prediction model has demonstrated efficacy in classifying the risk of adverse outcomes in patients with severe TBI.[9–11] This study aims to determine the predictors of clinical outcomes and develop a model to predict clinical outcomes in patients with tICH.

METHODS

Study design

This observational analytic study used a retrospective cohort design and was conducted in the neurology inpatient room of RSUDZA Banda Aceh from January to March 2025. This study was conducted in the Emergency Department and Inpatient Ward of Dr. Zainoel Abidin General Hospital (RSUDZA) in Banda Aceh.

Population and sample

The study population included all patients with tICH. The sample in this study comprised patients with tICH who met the inclusion and exclusion criteria and were recruited using a consecutive sampling. The inclusion criteria for this study were as follows: (1) age ≥ 18 years, (2) onset of head injury ≤ 24 h, and (3) diagnosis of severe head injury by head CT scan. The tICH was diagnosed by the medical doctor in charge (DPJP).

The exclusion criteria were as follows: (1) patients with multiple traumas; (2) patients with non-traumatic disorders (cardiovascular disorders, systemic infections, autoimmune diseases, malignancy, and kidney disease), and (3) incomplete radiology and laboratory data.

Data collection

Data on demographic characteristics were obtained from the patients' medical records. Data on the diagnosis of tICH were obtained from the results of history taking, physical examination, and supporting examination of head CT without contrast. A neurologist diagnosed the tICH. Data on serum leukocyte, sodium, and potassium levels were obtained from routine blood tests performed within 24 h. Blood sample examination was conducted at the clinical pathology laboratory of RSUDZA and verified by a clinical pathologist. A non-contrast head CT scan was performed within ≤24 h after injury and assessed by a radiologist. Intracerebral haemorrhage volume was measured using a non-contrast head computed tomography (CT) scan. The Broderick formula (ABC/2) was used to assess the haemorrhage volume, where A is the

greatest diameter of the haemorrhage as observed on a CT scan, B is the diameter perpendicular to A, and C is calculated by multiplying the approximate number of CT slices showing the haemorrhage by the thickness of each slice.[12] The GCS data were used to assess the level of consciousness by calculating the eye (E), motor (M), and verbal (V) components with a score range of 3-15. Clinical outcomes for patients with tICH were collected through structured interviews using the Glasgow Outcome Discharge Scale (GODS) with either the patients or their caregivers at the patients' discharge. The degree of outcome was grouped into eight levels: death =1, vegetative state=2, lower severe disability=3, upper severe disability=4, lower moderate disability=5, upper moderate disability=6, lower good recovery=7, and upper good recovery=8.[8,13]

Data analysis

The obtained data were analysed using SPSS statistical software version 25.0. Categorical data were presented as numbers, percentages, tables, and graphs. Univariate analysis was performed to describe the demographic data of the patients. Bivariate analysis was performed using chi-square tests. Diagnostic tests were conducted using 2×2 contingency tables to determine the sensitivity, specificity, cut-off values, and area under the curve (AUC) for each variable. Multiple linear regression analysis was used to assess the simultaneous effects of independent variables on clinical outcomes. Then, statistical significance was set at $p < 0.05$.

A multivariate analysis was conducted to develop a model exhibiting a stronger association with the dependent variable. Models identified as independent predictors were incorporated as components of the clinical outcome predictor scale for head injury. The development of this scoring model was based on findings from a multivariate logistic regression analysis of five key variables: GCS, bleeding volume, leukocytes, sodium, and potassium. Each of these variables was transformed into a categorical format and assigned a score reflecting the magnitude of the regression coefficient. A score of 7, the highest possible, indicates a severe clinical condition and correlates with a 98.7% probability of an adverse outcome. Conversely, a score of 0 represented the lowest risk, with a 3.4% probability of an adverse outcome. An increase in the cumulative score indicates a heightened likelihood of poor clinical outcomes. The model was validated using the Hosmer and Lemeshow tests, which produced a significance value of $p > 0.05$.

Ethical statement

Approval for study eligibility was obtained from the Ethics Committee of Dr. Zainoel Abidin Hospital Banda Aceh (No. 007/ETIK-RSUDZA/285). Written informed consent was obtained from all patients, their parents, or their guardians.

RESULTS

Eighty patients met the study's inclusion and exclusion criteria. The baseline characteristics of the study participants are shown in Table 1. Traumatic intracerebral haemorrhage predominantly affects males, with 46 cases (57.5%) reported in this study. An analysis by age group revealed that the highest incidence occurred in individuals aged 18-40 years, accounting for 35 cases (43.8%). This was followed by those aged 60 years or older (25 cases, 31.3%) and the 40-60 age group (20 cases, 25%). Upon admission, the GCS scores indicated that 70 patients (87.5%) had a GCS score exceeding 8, whereas 10 patients (12.5%) had a score of 8 or below. Regarding haemorrhage volume, 56 (70%) had a volume less than 20.5 mL, while 24 (30%) had a volume of 20.5 mL or more. Regarding leukocyte counts, 57 cases (71.3%) exhibited counts of 12,540 or higher, in contrast to 23 cases (28.7%) with counts below this threshold.

Table 1. Baseline characteristics of patients with tICH (n=80)

Variable	Category	n (%)
Gender	Male	46(57.5)
	Female	34(42.5)
Age group (year)	18-40	35(43.8)
	41-60	20(25.0)
	>60	25(31.3)
GCS Score on admission	≤ 8	10(87.5)
	>8	70(2.3)
Haemorrhage volume (mL)	Low (<20.5)	56(70.0)
	High (≥20.5)	24(30.0)
Leukocyte count (cells/µL)	Low (<12,540)	23(28.7)
	High (≥12,540)	57(71.3)
Sodium levels (mmol/L)	Normal (135–145)	43(53.8)
	Hyponatremia (<135)	36(45.0)
	Hypernatremia (>45)	1(1.3)
Potassium levels (mmol/L)	Normal (3.5–5.0)	58(72.5)
	Hypokalaemia (<3.5)	21(26.3)
	Hyperkalemia (>5.0)	1(1.3)
GODS score	Poor (1-4)	32(40.0)
	Good (5-8)	48(60.0)

tICH: traumatic intracerebral haemorrhage; GCS: Glasgow coma scale; GODS: Glasgow outcome at discharge scale

The AUC values of the independent variables as predictors of clinical outcomes in patients with tICH are shown in Table 2. The AUC for bleeding volume as a predictor of clinical outcomes in patients with tICH was 0.922, with a cut-off point of 20.5 mL, demonstrating a sensitivity and specificity of 68.8% and 95.8%, respectively. In contrast, the leukocyte AUC as a predictor of clinical outcomes in the same patient population was 0.838, with a cut-off point of 12,540 cells/µ, yielding a sensitivity of 93.8% and specificity of 54.2%. The AUCs for sodium and potassium as predictors of clinical outcomes in these patients were 0.636 and 0.750, respectively. The cut-off point for sodium was 134.5 mmol/L, with a sensitivity of 91.7% and a specificity of 40.6%, whereas the cut-off for potassium was 3.45 mmol/L, with a sensitivity of 70.8% and a specificity of 68.7%.

Table 2. AUC values of the independent variables of patients with tICH

Variable	Area	Cut-off	Sensitivity	Specificity	p-value
Haemorrhage volume (mL)	0.922	20.5	68.8	95.8	0.000*
Leukocyte count (cells/µL)	0.838	12,540	93.8	54.2	0.000*
Sodium levels (mmol/L)	0.636	134.5	91.7	40.6	0.040
Potassium levels (mmol/L)	0.750	3.45	0.708	0.687	0.000*

*p<0.05

The findings of the bivariate analysis of the independent variables and clinical outcomes are presented in Table 3. A significant correlation was observed between the GCS and clinical outcomes (p=0.000). The prevalence ratio of 3.182 suggests that patients with a low GCS score are three times more likely to experience adverse outcomes. Additionally, bleeding volume was

significantly associated with clinical outcomes (p=0.000; OR=50.6), with a prevalence ratio of 5.12. This indicates that patients with bleeding volumes ≥ 20.5 mL are 50 times more likely to encounter poor outcomes than those with lesser bleeding volumes.

Table 3 shows the correlations among several variables for predicting clinical outcomes in patients with tICH. In the groups with leukocyte counts ≥ 12,540/µL, 52.6% of patients experienced adverse outcomes, a significantly higher proportion than those in the group with leukocyte counts < 12,000/µL (8.7%; p=0.000; OR=0.086). This indicates a strong correlation between elevated leukocyte counts and adverse clinical outcomes. Furthermore, patients with hyponatremia exhibited a 76.5% incidence of poor outcomes, whereas the normal sodium group demonstrated a lower incidence of 30.6% (p=0.002; OR=7.35), suggesting that hyponatremia increases the risk of adverse outcomes by seven-fold. Additionally, a significant association was observed between potassium levels and clinical outcomes in patients with tICH (p=0.001), with hypokalaemia increasing the risk of poor outcomes by fivefold (OR=5.72) (Table 3).

Table 3. Correlation of Independent variables and clinical outcomes of patients with tICH

Variables	Clinical Outcomes (%)		p-value	Prevalence Ratio	OR
	Good	Poor			
GCS Score					
≤ 8	0	100.0	0.000	3.182	-
>8	68.6	31.4			
Haemorrhage volume (mL)			0.000		
<20.5	82.1	91.7		5.12	50.6
≥20.5	8.3	8.7			
Leukocyte count (cell/µL)					
<12,540	91.3	8.7	0.000	6.05	0.086
≥12,540	47.4	52.6			
Sodium levels (mmol/L)					
<135	23.5	76.5	0.002	2.5	7.35
135-145	69.4	30.6			
>145	100	0			
Potassium levels (mmol/L)					
<3.5	42.9	57.1	0.001	3.02	5.72
3.5-4.5	81.1	18.9			
>4.5	0	1			

Chi-square test. *p<0.05 statistically significant. GCS: Glasgow coma scale; OR: odds ratio.

The multivariate analysis revealed that both GCS score and bleeding volume significantly influenced the clinical outcomes, with p-values of 0.010 and 0.034, respectively. The odds ratio (OR) for the GCS was 10.102, indicating that each one-point increase in the GCS enhanced the likelihood of a favourable clinical outcome by more than tenfold. Conversely, bleeding volume exhibited a negative regression coefficient (B = -2.045) with an OR of 0.129, suggesting that an increase in bleeding volume substantially reduced the probability of a favourable outcome by nearly 87% for each unit in volume. However, leukocyte, sodium, and potassium levels did not demonstrate statistically significant associations with the clinical outcomes (p > 0.05).

Table 4. Analysis of Independent variables and clinical outcomes of patients with tICH

Variable	B	p-value	OR
GCS score	2.313	0.010	10.102
Haemorrhage volume	-2.045	0.034	0.129
Leukocyte count	-0.873	0.460	0.418
Sodium levels	1.267	0.246	3.549
Potassium levels	1.184	0.135	3.267

Multivariate analysis. *p<0.05 statistically significant. GCS: Glasgow coma scale; OR: odds ratio.

Table 5 provides a comprehensive overview of the results of the logistic regression analysis. When the GCS score, bleeding volume, leukocyte count, and potassium and sodium levels were identified as risk factors, the predicted likelihood of a poor clinical outcome was 98.7%. In cases where GCS score, bleeding volume, leukocyte count, and potassium level were considered risk factors, but sodium level was not, the probability of mortality was estimated to be 95.6%. Alternatively, when bleeding volume, leukocytes, potassium, and sodium were considered risk factors, excluding GCS, the predicted poor clinical outcome was 88.4%. Furthermore, if bleeding volume, potassium, and sodium were risk factors, whereas GCS and leukocytes were not, the prediction of a poor clinical outcome was 76.2%. Finally, when neither bleeding volume nor sodium was considered a risk factor, the predicted clinical outcome was 49.4%.

In cases where the GCS, bleeding volume, and leukocyte count were not considered risk factors, but sodium and potassium levels were identified as such, the probability of a poor clinical outcome was 29.2%. Alternatively, when GCS score, bleeding volume, leukocyte count, and potassium level were not considered risk factors, but sodium level was, the likelihood of a poor clinical outcome was 11.2%. Additionally, if none of the factors—GCS score, bleeding volume, leukocyte count, potassium, and sodium levels—were identified as risk factors, the predicted probability of poor clinical outcomes was 3.4%. The combined scores for the GCS, leukocyte count, bleeding volume, potassium, and sodium levels ranged from 0 to 7 (Table 5).

Table 5. Logistic regression modelling analysis of independent variables on clinical outcomes of patients with tICH

GCS ≤ 8	Haemorrhage volume >20.5	Leukocyte count >12,540	Sodium levels <135	Potassium levels <3.5	Probability
1	1	1	1	1	0.987
1	1	1	0	1	0.956
0	1	1	1	1	0.884
0	1	0	1	1	0.762
0	1	0	1	0	0.494
0	0	1	1	0	0.292
0	0	0	1	0	0.112
0	0	0	0	0	0.034

Note: 1 = risk; 0 = no risk

Five variables were identified as significantly correlated with the clinical outcomes of patients with tICH: GCS score, bleeding volume, leukocyte count, sodium level, and potassium level. A predictive scoring system was developed using the regression coefficient values for each variable to enhance the interpretability of the results and facilitate clinical applications. Scoring was conducted in proportion to the value of the regression coefficient (β), such that a greater contribution of a variable to adverse outcomes corresponded to a higher score.

Based on the modelling results, the value of GCS at risk was 2, the value of bleeding volume at risk was 2, and the value of leukocytes, sodium, and potassium at risk was 1. This scoring model aims to simplify the identification of the risk of poor outcomes in patients with head injuries. With a maximum total score of 7, the following risk stratification can be determined: Score 0-2=low risk; score 3-4=moderate risk; score 6-7 = high risk.

Clinical outcome prediction score based on GCS, bleeding volume, leukocytes, sodium, and potassium in patients with tICH was presented in Table 6.

Table 6. Clinical outcome prediction score in patients with tICH

Variables	Score
GCS Score	
≤ 8	2
>8	0
Haemorrhage volume (mL)	
<20.5	0
≥20.5	2
Leukocyte count (cell/μL)	
<12,540	0
≥12,540	2
Sodium levels (mmol/L)	
<135 and 145	1
135-145	0
Potassium levels (mmol/L)	
<3.5. and 5.0	1
3.5-4.5	0
Total score	0-7

This outcome indicates a robust alignment between the predicted and observed values, indicating a satisfactory model fit. Additionally, the Hosmer and Lemeshow contingency table revealed a consistent distribution across score strata and predicted outcome categories, particularly at higher scores (5–7), in which the proportion of patients with poor outcomes was predominant. The proportion of favorable outcomes increased progressively with higher scores. For example, patients with a total score of 7 demonstrated a predicted probability of poor outcome of 98.7%, whereas those with a score of 0 had a corresponding probability of only 3.4%.

DISCUSSION

The current study revealed significant correlations between the GCS variable, bleeding volume, leukocyte count, potassium, and sodium, and clinical outcome in patients with tICH. However, in the multivariate test, the significant value was only in the GCS and bleeding volume. The study results showed that GCS had a significant relationship with the clinical outcomes of the patients. All patients with GCS ≤8 had poor outcomes, whereas 68.6% of patients with GCS >8 had good outcomes ($p<0.001$). In the multivariate analysis, the GCS was the single strongest independent predictor of clinical outcomes, with an OR of 10.102 ($p=0.010$). This finding is consistent with the basic neurology theory, which states that the GCS reflects the patient's level of consciousness and neurological function. A low GCS reflects cortical and brainstem dysfunction due to primary trauma or increased intracranial pressure; therefore, when neurologic function is significantly impaired, the clinical prognosis is generally poor. This study is consistent with the systematic review by Siegel and Trovato, which states that the GCS is still the primary measurement tool for assessing the severity of traumatic brain injury and has a strong correlation with clinical outcomes, particularly mortality.[7]

In the bivariate analysis, 91.7% of patients with bleeding ≥20.5 ml had poor outcomes. Bleeding volume remained significant in the multivariate analysis, with an OR of 0.129 ($p = 0.034$), indicating that the greater the bleeding, the lower the chance of a good outcome. Physiologically, intracranial haemorrhage leads to increased intracranial pressure (ICP), decreased cerebral perfusion pressure (CPP), and ultimately ischemia and death of the brain tissue. Rosnati et al. stated that hematoma volume >20 ml is a critical point associated with poor prognosis in head injury patients.[14]

Leukocyte levels showed a significant association in the bivariate test, which 91.3% of patients with leukocyte counts <12,540/μL had a good outcome ($p<0.001$). However, in the multivariate analysis, leukocytes were not significant ($p=0.460$), indicating that although leucocytosis was associated with poor outcomes, its effect was not an independent factor. Following head trauma, leucocytosis often occurs as part of the systemic inflammatory response

syndrome (SIRS), which triggers several thrombogenic mechanisms and inflammatory cytokines.[15] Experimental studies in animals by Bele et al. showed that interventions such as quetiapine and propranolol significantly reduced leukocyte migration to the lesion area and cerebral oedema, supporting leukocytes as the primary inflammatory mediators after trauma.[16] A large retrospective cohort analysis using MIMIC-III data by Wang et al identified three phenotypes of head injury patients based on initial leukocyte patterns within 24 hours, where the group with the highest leukocyte elevation had a mortality of 25.6%, versus 13.3% in the low group further corroborating the role of leukocytes as an inflammatory indicator and predictor of mortality, but not an independent variable after correction for other clinical factors.[17]

In the bivariate test, hyponatremia (<135 mmol/L) was significantly associated with poor outcomes (76.5%) (p=0.002), but the multivariate test results were not significant (p=0.246). This indicates that sodium imbalance contributes to outcomes but does not stand alone as a strong predictor after controlling for other variables. Sodium disturbance in head injury is often caused by SIADH (syndrome of inappropriate antidiuretic hormone secretion) or cerebral salt-wasting syndrome (CSWS), both of which can exacerbate cerebral oedema. Rajagopal et al. reported that hyponatremia is the most common electrolyte disorder in head injury and acts as an independent predictor of poor neurological outcome. In contrast, SIADH is a common cause of hyponatremia after head injury and is associated with neurological symptoms such as seizures and decreased consciousness.[18]

The CSWS, though less common than SIADH, requires diagnostic differences due to different therapies. A review of fluid and electrolyte disorders in TBI explains that CSWS causes hypovolemia and natriuresis and increases cerebral oedema risk, which can worsen prognosis if untreated.[19] Potassium levels showed a significant association, where patients with hypokalaemia <3.5 mmol/L were more likely to have poor outcomes (57.1%) (p=0.001). However, in multivariate analysis, potassium was not a significant independent predictor (p=0.135). This finding was supported by a systematic review that concluded hypokalaemia in TBI patients was associated with increased mortality. A longitudinal study by Mwachaka et al. revealed that hypokalaemia detected on admission and after 48 hours of care showed increased mortality risk (OR 4.12 and 5.12, respectively).[20]

In this study, bivariate analysis showed that leukocyte, sodium, and potassium levels were significantly associated with the clinical outcomes of patients with severe head injury. However, in the multivariate test, they did not show any statistical significance. This suggests that the influence of these three variables on clinical outcomes is not an independent predictor, but may play a supporting or secondary role, and that their influence can be compensated for by more dominant variables.[21] A substantial increase in hematoma volume is associated with poor prognostic outcomes in patients with intracerebral haemorrhage (ICH).[22] A previous study identified specific clinical factors that can predict hematoma expansion, including initial systolic blood pressure, anticoagulant use, and liver disease, underscoring their significance in the early assessment and management of ICH.[23] Furthermore, the significance of blood pressure variability, particularly during the acute phase following an intracerebral haemorrhage (ICH) event, has been increasingly recognized as a critical predictive factor. Variability in systolic blood pressure shortly after a haemorrhagic incident has been consistently associated with both early neurological deterioration and subsequent poor functional outcomes, thereby reinforcing the necessity for rigorous blood pressure monitoring and management. [24,25]

The importance of hematoma characteristics, including size and the presence of intraventricular haemorrhage (IVH), has been emphasized in numerous studies. A large initial hematoma volume is widely recognized as a significant predictor of adverse outcomes following tICH.[22,26]. Metabolic indices associated with blood glucose levels have been investigated in various studies. Elevated glucose levels, particularly in stress hyperglycaemia, may be correlated with adverse clinical outcomes because of their potential to exacerbate cerebral ischemia during haemorrhagic events.[27,28]

Statistically, this can be explained by the confounding effect of other more powerful variables, namely GCS and haemorrhage volume, which remained significant in the multivariate

analysis. The GCS directly reflects neurological integrity, and evidence suggests that lower GCS scores are associated with increased risk of adverse outcomes, including higher intracranial pressure (ICP) and a higher likelihood of structural damage.[29,30] Bleeding volume is an indicator of structural brain damage and intracranial pressure.[31] These two variables have been widely confirmed as major predictors of outcomes in patients with head injury.[32,33] In contrast, elevated leukocyte counts generally reflect a systemic inflammatory response due to trauma or a secondary infectious process.[34] Although leucocytosis may worsen the prognosis by contributing to cerebral oedema and metabolic instability, it is more of a marker of secondary processes. Therefore, in multivariate models that account for the interaction of all variables, the influence of leukocytes is not significant.[35]

Similarly, sodium and potassium disorders are common in patients with severe brain injury due to the SIADH, CSWS, or therapeutic interventions such as hypotonic fluid administration or diuretics.[36] Hyponatremia and hypokalaemia may worsen the clinical status, but are often sequelae of the severity of the injury itself. In addition, the high variability of laboratory values and the influence of external factors such as medical therapy, comorbidities, and infections can lead to a reduction in the predictive power of these variables in multivariate models and the possible correlation between variables (multicollinearity).[37,38] For example, the correlation between low GCS and electrolyte disturbances can cause the independent effect of the variable to be statistically insignificant because most of its effect is already explained by other variables.[39,40] Consequently, this predictive score serves as an effective risk-stratification tool for patients with head injury in clinical settings.

Thus, the non-significance of leukocyte, sodium, and potassium variables in the multivariate analysis does not necessarily exclude their clinical relevance; rather, it reflects that their contribution to head injury patient outcomes is secondary and contextual, and shows a relatively weaker influence than the primary neurologic variables that have a more direct etiologic association. A limitation of this study is the small sample size; therefore, further research involving a larger population and sample size is warranted.

CONCLUSION

This study revealed that GCS score and haemorrhage volume were significant, independent predictors of clinical outcomes. Lower GCS scores and larger bleeding volumes were strongly associated with poor outcomes in this study. Although leukocyte count, sodium levels, and potassium levels showed significant associations in the bivariate analysis, they did not emerge as independent predictors in the multivariate analysis. This scoring system offers a practical tool for risk stratification in clinical settings, potentially aiding rapid decision-making for patients with tICH. However, further research is needed to validate this model in larger and more diverse populations and to explore its applicability across various clinical contexts.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest associated with the materials presented in this paper.

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None

DATA AVAILABILITY

The authors confirm that the data supporting the findings of this study are available within the article.

SUPPLEMENTAL DATA

No additional supplemental data are provided for this study. All relevant data supporting the findings of this research are included within the main article.

AUTHOR CONTRIBUTIONS

AR and SS initiated the study, formulated the research objectives, coordinated the overall study process, and made significant contributions to the data interpretation and manuscript preparation. IZ played an active role in refining the methodology, supervising the recruitment and data collection phases, and contributed to data analysis and manuscript revision. SS and IZ provided expert input on the theoretical frameworks. IH and NM guided the interpretation of the complex findings and conducted a thorough critical review to enhance the clarity and robustness of the manuscript. All authors have read and agreed to the published version of this manuscript.

DECLARATION OF USING AI IN THE WRITING PROCESS

We hereby confirm that no artificial intelligence (AI) tools or methodologies were utilized at any stage of this study, including during data collection, analysis, visualization, or manuscript preparation. All work presented in this study was conducted manually by the authors without the assistance of AI-based tools or systems.

LIST OF ABBREVIATIONS

TBI: Traumatic Brain Injury; tICH: Traumatic Intracerebral Haemorrhage; GCS: Glasgow Coma Scale; GODS: Glasgow Outcome at Discharge Scale. WHO: World Health Organization, RSUDZA: Rumah Sakit Umum Daerah dr Zainoel Abidin, CT: Computed Tomography, E: Eyes, M: Move, V: Verbal, AUC: Area Under the Curve, OR: Odds Ratio, CPP: Cerebral Perfusion Pressure, ICP: Intracranial Pressure, SIRS: Systemic Inflammatory Response Syndrome, MIMIC-III: Medical Information Mart for Intensive Care III, CSWS: Cerebral Salt-Wasting Syndrome, SIADH: Syndrome of Inappropriate Antidiuretic Hormone Secretion; DPJP: Dokter Penanggung Jawab Pelayanan.

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