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Original Article

Influencing factors on neurological prognosis after traumatic brain injury and the role of brain tissue oxygen pressure ($PbtO_2$) monitoring

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Abstract: Objective: To identify factors influencing neurological prognosis following traumatic brain injury (TBI) and to analyze the role of brain tissue oxygen pressure ($PbtO_2$) monitoring in prognostication. Methods: In this case-control study, medical records of 412 individuals diagnosed with TBI were thoroughly examined and analyzed. The patients were divided into two groups based on their prognosis at three months post-injury: Good Prognosis (n = 321) and Poor Prognosis (n = 91). Demographic and clinical characteristics, brain tissue oxygen partial pressure, radiological and laboratory findings, treatment interventions, and complications were compared between the two groups. Logistic regression analysis was conducted to identify the risk factors for neurological prognosis, and the predictive value of these factors was evaluated using receiver operating characteristic (ROC) curve analysis. Results: The study identified associations between Injury Severity Score (ISS), Glasgow Coma Scale (GCS), $PbtO_2$ levels, radiological findings (diffuse axonal injury and subarachnoid hemorrhage), and laboratory parameters (platelet count and arterial oxygen partial pressure (PaO_2)) with neurological prognosis following TBI. Initial $PbtO_2$ levels demonstrated independent predictive value for poor neurological outcomes (Area Under the Curve (AUC) = 0.804). Conclusion: The study highlights the prognostic significance of injury severity, brain tissue oxygenation, radiological findings, and laboratory parameters in determining neurological outcomes following TBI. Furthermore, the findings emphasize the potential of $PbtO_2$ monitoring as a valuable tool in prognostic assessment.

Keywords: Traumatic brain injury, neurological prognosis, influence factors, brain tissue oxygen pressure

Introduction

Traumatic brain injury (TBI) is a significant public health concern due to its pervasive impact on individuals across all age groups and its substantial socioeconomic burden. TBI encompasses a broad spectrum of injuries resulting from external forces, such as blunt trauma or penetrating head injuries, often resulting from motor vehicle accidents, falls, sports-related incidents, and assaults [1, 2]. Globally, TBI affects millions of individuals each year, imposing a considerable burden on healthcare systems and society [3, 4]. TBI is particularly concerning due to its association with significant morbidity and mortality, exerting a substantial toll on affected individuals and their families [5-7]. While TBI affects individuals across the lifespan, it is notably prevalent among young individuals, with peak incidence observed in adolescents as well as young adults. The long-term consequences of TBI can be profound, leading to a range of physical, emotional, cognitive, and behavioral impairments that can profoundly impact an individual's quality of life and functional independence [8, 9].

Over the past few years, considerable progress has been made in understanding the pathological mechanisms and prognostic factors associated with TBI [10]. Factors such as the Injury Severity Score (ISS), Glasgow Coma Scale (GCS), and radiological findings like diffuse axonal injury and subarachnoid hemorrhage have been consistently identified as critical determinants of neurological outcomes post-TBI [11]. These elements reflect the extent of primary and secondary brain injuries, influencing recov-

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ery trajectories and long-term functional outcomes [12]. Among these, physiological monitoring parameters have gained particular interest, specifically brain tissue oxygen partial pressure ($PbtO_2$) monitoring [13]. $PbtO_2$, a measure of cerebral oxygenation, offers real-time insights into the brain's metabolic state and serves as a sensitive marker of cerebral hypoxia-a known factor exacerbating secondary brain injury [13]. Its role in prognostication is increasingly recognized, as it potentially bridges the gap between the global and regional assessments of brain oxygenation, providing a more nuanced understanding of cerebral pathology in TBI patients [14]. Despite these insights, integrating $PbtO_2$ monitoring into routine clinical practice requires robust evidence of its prognostic value, highlighting the necessity to systematically investigate its utility [15].

The multifaceted nature of TBI pathophysiology necessitates a comprehensive approach to prognostication and management, considering the diverse factors involved in the intricate interplay of primary and secondary injury mechanisms [16-18]. This underscores the need for a multidimensional understanding of TBI pathophysiology to inform the development of tailored therapeutic strategies and prognostic models aimed at improving patient outcomes. Despite advances in acute management strategies, predicting neurological prognosis following TBI remains challenging, necessitating a comprehensive understanding of the diverse factors that influence patient outcomes. This study aims to elucidate the influencing factors of neurological prognosis after TBI and to perform an analysis role of $PbtO_2$ monitoring in this context.

Materials and methods

Study design

This retrospective case-control study analyzed data from 412 TBI patients admitted to Baotou Central Hospital from January 2023 to December 2023. The patients were divided into two groups based on their prognosis three months post-injury: Good prognosis group (n = 321) and Poor prognosis group (n = 91). This study was approved by the Ethics Committee of Baotou Central Hospital and informed consent was waived.

Inclusion and exclusion criteria

Inclusion criteria [19, 20]: 1. Definitive diagnosis of acute TBI based on medical history and auxiliary examinations; 2. GCS score of 3-14; 3. Follow-up duration of more than three months. Exclusion criteria: 1. Age under 14 years; 2. Pregnant women; 3. History of autoimmune disease or gout.

The inclusion and exclusion criteria were carefully selected to ensure a homogeneity of the study population and strengthen the validity of the findings. Patients under 14 years of age were excluded due to the physiological and anatomical differences in pediatric populations compared to adults, which could impact the generalizability of brain oxygenation data. Children and adolescents exhibit distinct patterns of injury, recovery, and neurodevelopment that differ significantly from adults, potentially introducing variability that could obscure the analysis of $PbtO_2$'s prognostic value. Additionally, individuals with a history of autoimmune disease or gout were excluded to minimize factors such as chronic inflammation and metabolic disturbances. Autoimmune diseases and gout can independently affect systemic oxygenation and cerebral metabolism, complicating the interpretation of $PbtO_2$ measurements as purely reflective of traumatic injury effects. By excluding these patients, the study aims to focus more precisely on the pathophysiology and prognostic outcomes of TBI in a controlled adult population.

Grouping method

The Glasgow Outcome Score (GOS) was used to evaluate the prognosis of the two patient groups at 3 months post-injury, with 1 point for death, 2 for vegetative state, 3 for severe disability, 4 for moderate disability, and 5 for good recovery. A GOS score > 3 is defined as a favorable prognosis. Patients were grouped based on their prognosis score, with GOS scores > 3 categorized into the Good Prognosis group, and patients with GOS scores ≤ 3 categorized into the Poor Prognosis group.

Clinical characteristics

ISS scores (injury severity): The severity of injuries across nine body regions (face, head, neck, abdomen, thorax, spine, lower extremities, upper extremities, and external) was assessed

using the Abbreviated Injury Scale (AIS), which ranges from 0 to 51. The scale classifies injuries by severity, where 0 for no injury, 1 - a minor injury, 2 - a moderate injury, 3 - a serious injury, 4 - a severe injury, and 5 - a critical injury.

To compute the ISS, these nine body areas were consolidated into six broader categories: R1 for head or neck injuries, R2 for face, R3 for chest, R4 for abdominal or pelvic parts, R5 for extremities or the pelvic girdle, and R6 for external injuries. The ISS was determined by summing the squares of the AIS scores for the three most severe injuries, producing a scale range of 0 to 75. The ISS demonstrated a high reliability, with a Cronbach's alpha of 0.93 [21].

Admission glasgow coma scale (GCS) score: The GCS is a clinical tool used to assess the level of consciousness in patients by evaluating three aspects: eye response, verbal response, and motor response. The scores for each component are summed, with higher scores indicating better consciousness levels. The maximum score is 15, indicating clear consciousness. Scores of 13-15 indicate mild consciousness impairment, 9-12 indicate moderate impairment, and below 8 indicate coma. Within the scale, a score of 13-15 was categorized as mild, 9-12 as moderate, and 3-8 as severe. The GCS demonstrated a Cronbach's alpha of 0.78 [22].

Brain tissue oxygen partial pressure detection

Three months post-injury, $PbtO_2$ was measured using the LICOX-II $PbtO_2$ monitoring system (GMS, Germany) with a Clark-type microelectrode probe. The $PbtO_2$ monitoring probe was placed at the junction of the necrotic brain tissue (hematoma) and the penumbra of contused brain tissue. The levels of $PbtO_2$ (mmHg) were recorded, with $PbtO_2$ < 15/20/25/30 mmHg (%) representing the percentage of time at each measurement.

Radiological findings

Three months post-injury, patients underwent brain Computed Tomography (CT) examinations using the Philips Brilliance 64-slice/128-layer spiral CT scanner. The scan parameters included a slice thickness and reconstruction interval of 0.45 mm, a scanning speed of 0.75 r/s, a tube voltage of 120 kV, and a tube current of 250 mA, applying an adaptive iterative dose reduction algorithm. The scan range extended from the inferior margin of the second cervical vertebra to the outer table of the skull. Via a median cubital vein, iodixanol (320 mgI/mL) of a dose of 50-60 mL and 30 mL of normal saline was injected at a flow rate of 4.5-5.5 mL/s. Arterial-phase images were acquired at 2-second intervals, starting 10 seconds post-injection, to capture cross-sectional views. For suspected aneurysms in the circle of Willis, an enlarged scan technique was employed whenever possible. The original images were transferred to the Philips IntelliSpace Portal workstation for post-processing, including volume rendering, multiplanar reformation, curved planar reformation, and maximum intensity projection. Two senior radiologists and one associate chief neurosurgeon jointly reviewed the original axial CTA images and performed image post-processing, documenting midline shift (mm) and the presence of intracranial hemorrhage, diffuse axonal injury, skull fracture, and brain edema.

Laboratory findings

Three months after the injury, a 5 ml blood sample was collected from the antecubital vein early in the morning while the patient was fasting. The levels of hemoglobin (g/dL), hematocrit (%), white blood cell count ($\times 10^9$/L), and platelet count ($\times 10^9$/L) were measured. The analyses were conducted using the STA Compact, China, a fully automated coagulation analyzer (model HC00608166). Additionally, arterial blood was sampled under fasting conditions and analyzed using the Roche automated biochemical analyzer with accompanying reagents, along with an automated blood gas analyzer to assess the patient's biochemical indicators and perform arterial blood gas analysis, examining lactate, pH, and PaO_2.

Treatment methods

Upon admission, patients received individualized treatment tailored to their specific conditions, and the interventions were duly documented. Over the three months post-admission, any complications experienced by the patients were meticulously recorded.

Statistical analysis

Data analysis was conducted using SPSS 29.0 statistical software (SPSS Inc., Chicago, IL, USA). Categorical data were presented as [n (%)] and analyzed using the chi-square test.

Table 1. Comparison of demographic and clinical characteristics between the two groups

Parameter	Good prognosis (n = 321)	Poor prognosis (n = 91)	t/χ^2	p
Age (years)	39.14 ± 10.23	40.67 ± 12.85	1.186	0.236
Gender (M/F)	195 (60.75%)/126 (39.25%)	57 (62.64%)/34 (37.36%)	0.107	0.744
BMI (kg/m^2)	23.98 ± 2.14	24.26 ± 1.92	1.126	0.261
Smoking history (%)	44 (13.71%)	10 (10.99%)	0.460	0.498
Drinking history (%)	26 (8.10%)	10 (10.99%)	0.742	0.389
Hypertension (%)	57 (17.76%)	15 (16.48%	0.080	0.778
Diabetes (%)	70 (21.81%)	18 (19.78%)	0.173	0.677
Hyperlipidemia (%)	48 (14.95%)	12 (13.19%)	0.178	0.673
ISS	26.89 ± 5.67	30.45 ± 7.32	4.938	< 0.001
Admission GCS	11.02 ± 2.80	8.80 ± 3.20	6.462	< 0.001

BMI: Body mass index; ISS: Injury severity score; GCS: Glasgow coma scale.

Table 2. Comparison of $PbtO_2$ between the two groups

Parameter	Good prognosis (n = 321)	Poor prognosis (n = 91)	t/χ^2	P
Initial $PbtO_2$ (mmHg)	26.85 ± 4.82	20.34 ± 5.67	10.922	< 0.001
$PbtO_2$ < 20 mmHg (%)	29 (9.03%)	21 (23.08%)	13.112	< 0.001
$PbtO_2$ < 25 mmHg (%)	57 (17.76%)	29 (31.87%)	8.548	0.003
$PbtO_2$ < 30 mmHg (%)	165 (51.40%)	55 (60.44%)	2.327	0.127
$PbtO_2$ < 35 mmHg (%)	195 (60.75%)	58 (63.74%)	0.267	0.605

$PbtO_2$: Brain tissue oxygen pressure.

Normality of continuous variables was assessed using the Shapiro-Wilk method. For normally distributed continuous variables, data were presented in the form of (mean ± SD) and analyzed using the t-test with corrected variance. A two-tailed $P < 0.05$ was considered statistically significant. The relationship between continuous variables, such as platelet count and PaO_2 (mmHg), and prognostic outcomes was assessed using Pearson correlation analysis, while the relationship between categorical variables, such as the presence of subarachnoid hemorrhage and prognostic outcomes, was evaluated using Spearman correlation analysis. The diagnostic accuracy of $PbtO_2$ differences in brain injury was assessed using the area under the receiver operating characteristic (ROC) curve (AUC). Variables showing significant differences in both difference and correlation analyses were included as covariates in logistic regression analysis.

Results

Demographic and clinical characteristics

The demographic and clinical characteristics of the study population are summarized in **Table 1**. There were 321 patients in the good prognosis group and 91 patients in the poor prognosis group. Age, gender, body mass index (BMI), smoking history, drinking history, hypertension, diabetes, and hyperlipidemia showed no significant differences between the two prognosis groups ($P > 0.05$ for all). However, the ISS and Admission GCS demonstrated statistically significant differences. Specifically, the poor prognosis group exhibited a significantly higher ISS (30.45 ± 7.32 vs. 26.89 ± 5.67, $t = 4.938$, $P < 0.001$) and lower GCS on admission (8.80 ± 3.20 vs. 11.02 ± 2.80, $t = 6.462$, $P < 0.001$) compared to the good prognosis group. These results suggest that ISS and GCS may play a pivotal role in predicting the neurological prognosis after TBI.

$PbtO_2$ monitoring

A comparison of $PbtO_2$ levels between the two groups revealed that initial $PbtO_2$ levels were significantly higher in the good prognosis group than in the poor prognosis group (26.85 ± 4.82 mmHg vs. 20.34 ± 5.67 mmHg, $t = 10.922$, $P < 0.001$) (**Table 2**). Additionally, the proportion of patients with $PbtO_2$ below 20 mmHg and 25 mmHg were significantly different between the

Table 3. Comparison of radiological findings between the two groups

Parameter	Good prognosis (n = 321)	Poor prognosis (n = 91)	t/χ^2	P
Diffuse axonal injury	1.72 ± 0.50	2.41 ± 1.20	8.126	< 0.001
Subarachnoid hemorrhage	138 (42.99%)	53 (58.24%)	6.632	0.010
Intraventricular hemorrhage	44 (13.71%)	16 (17.58%)	0.856	0.355
Base-of-skull fracture	80 (24.92%)	27 (29.67%)	0.831	0.362
Epidural hematoma	110 (34.27%)	35 (38.46%)	0.547	0.460

two prognosis groups ($PbtO_2$ < 20 mmHg: 9.03% vs. 23.08%, t = 13.112, P < 0.001; $PbtO_2$ < 25 mmHg: 17.76% vs. 31.87%, t = 8.548, P = 0.003). However, there were no significant differences in the percentages of patients with $PbtO_2$ measurements below 30 mmHg and 35 mmHg between the good and poor prognosis groups (P > 0.05). These findings underscore the potential influence of $PbtO_2$ levels, particularly when below certain thresholds, on the neurological prognosis following TBI, warranting further investigation into the role of $PbtO_2$ monitoring in this context.

Radiological findings

The presence of diffuse axonal injury was significantly higher in the poor prognosis group compared to the good prognosis group (2.41 ± 1.20 vs. 1.72 ± 0.50, t = 8.126, P < 0.001) (**Table 3**). Furthermore, subarachnoid hemorrhage was more prevalent in the poor prognosis group compared to the good prognosis group (58.24% vs. 42.99%, t = 6.632, P = 0.010), while there were no significant differences in the incidence of intraventricular hemorrhage, base-of-skull fracture, and epidural hematoma between the two prognosis groups (all P > 0.05). These results indicate the potential impact of certain radiological findings, particularly diffuse axonal injury and subarachnoid hemorrhage, on neurological prognosis following TBI.

Laboratory findings

The platelet count was significantly lower in the poor prognosis group compared to the good prognosis group (198.4 ± 45.18×10^9/L vs. 230.7 ± 35.6×10^9/L, t = 7.174, P < 0.001), as well as a lower PaO_2 (89.69 ± 7.29 mmHg vs. 93.62 ± 6.54 mmHg, t = 4.930, P < 0.001) (**Figure 1**). However, there were no significant differences in the levels of hemoglobin, hematocrit, white blood cell count, lactate, and arterial pH between the two prognosis groups (all P > 0.05). These findings suggest that certain laboratory parameters, particularly platelet count and PaO_2, may be associated with neurological prognosis following TBI.

Treatment interventions

There were no significant differences in the utilization of decompressive craniectomy, intracranial pressure management, hypertonic saline therapy, seizure prophylaxis, or hypothermia treatment between the two prognosis groups (all P > 0.05) (**Table 4**). These results suggest that the treatment interventions evaluated in this study may not be significantly associated with the neurological prognosis following TBI.

Complications

There were no significant differences in the occurrences of seizure, permanent need for ventilator support, deep vein thrombosis, or inpatient mortality between the two prognosis groups (all P > 0.05) (**Table 5**). These findings suggest that the investigated complications may not have a significant association with the neurological prognosis following TBI.

Correlation analysis

The ISS demonstrated a positive correlation (r = 0.225, P < 0.001), while admission GCS showed a negative correlation (r = -0.275, P < 0.001) with poor neurological outcomes (**Figure 2**). Additionally, initial $PbtO_2$, PaO_2 and platelet count exhibited negative correlations (r = -0.437, P < 0.001; r = -0.236, P < 0.001; and r = -0.300, P < 0.001, respectively) with poor neurological outcomes. Subarachnoid hemorrhage, $PbtO_2$ below 25 mmHg, $PbtO_2$ below 20 mmHg and diffuse axonal injury also demonstrated positive correlations (r = 0.127, P < 0.001; r = 0.144, P < 0.001; r = 0.178, P < 0.001 and r = 0.277, P < 0.001, respectively) with poor neurological outcomes. The correlation analysis provides valuable insight into the

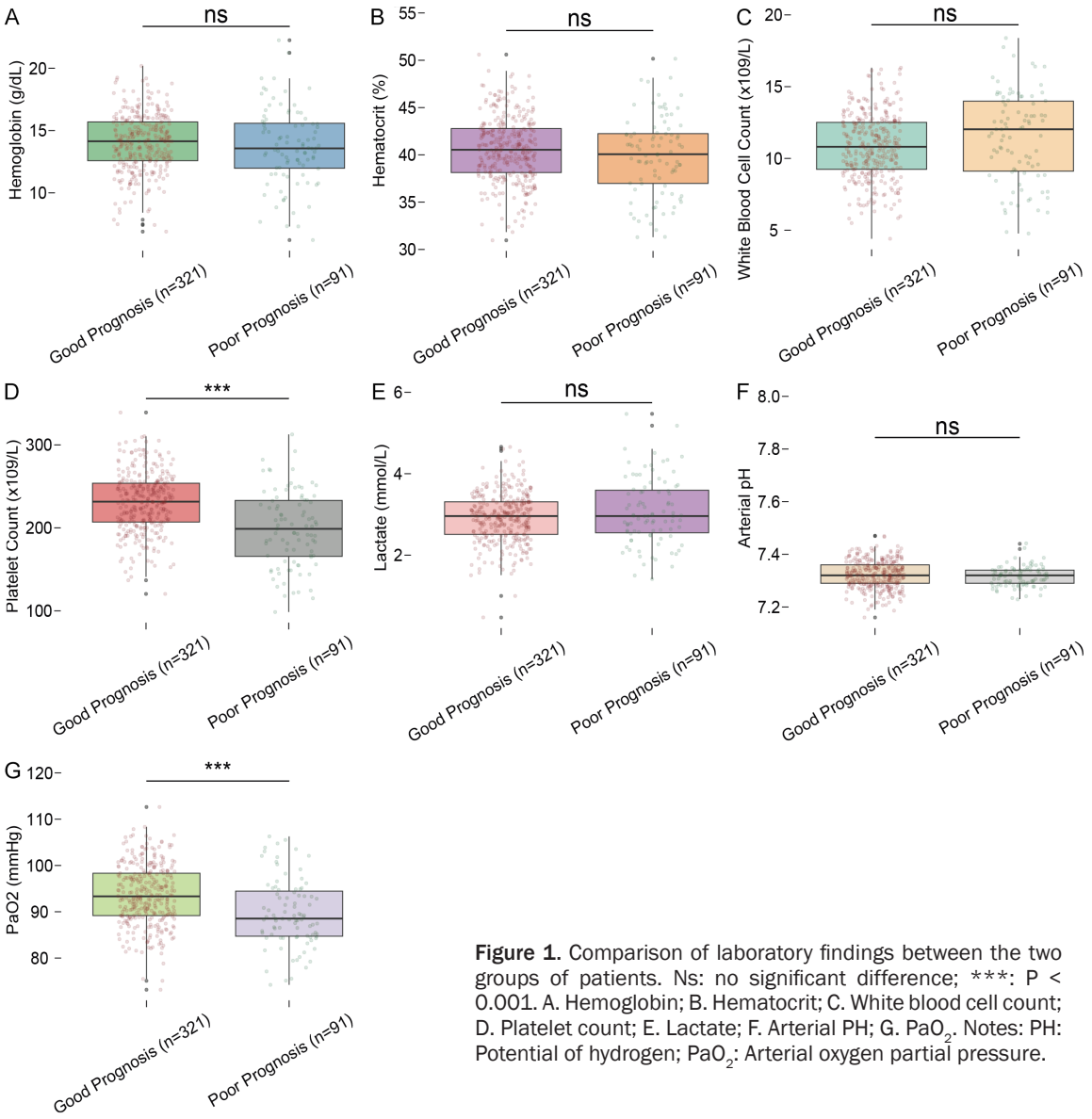

Figure 1. Comparison of laboratory findings between the two groups of patients. Ns: no significant difference; ***: P < 0.001. A. Hemoglobin; B. Hematocrit; C. White blood cell count; D. Platelet count; E. Lactate; F. Arterial PH; G. PaO_2. Notes: PH: Potential of hydrogen; PaO_2: Arterial oxygen partial pressure.

Table 4. Comparison of treatment Interventions between the two groups

Parameter	Good prognosis (n = 321)	Poor prognosis (n = 91)	χ^2	P
Decompressive craniectomy	45 (14.02%)	17 (18.68%)	1.206	0.272
Intracranial pressure management	255 (79.44%)	71 (78.02%)	0.086	0.769
Hypertonic saline therapy	61 (19.00%)	18 (19.78%)	0.028	0.868
Seizure prophylaxis	181 (56.39%)	49 (53.85%)	0.186	0.667
Hypothermia treatment	23 (7.17%)	11 (12.09%)	2.269	0.132

relationships between these influencing factors and poor neurological outcomes.

Logistic regression analysis

Logistic regression analysis shows that a higher ISS (ISS ≥ 33.53, OR: 1.101, P < 0.001) is significantly associated with an increased risk of poor neurological outcomes (**Table 6**). Conversely, a higher GCS score at admission (GCS ≥ 8.7, OR: 0.762, P < 0.001) is significantly associated with a decreased risk of poor outcomes. Similarly, a higher initial $PbtO_2$ ($PbtO_2$ ≥

Table 5. Comparison of complications between the two groups

Parameter	Good prognosis (n = 321)	Poor prognosis (n = 91)	χ^2	P
Seizure	21 (6.54%)	8 (8.79%)	0.548	0.459
Permanently needing ventilator	43 (13.40%)	15 (16.48%)	0.559	0.455
Deep vein thrombosis	20 (6.23%)	5 (5.49%)	0.067	0.795
Inpatient mortality	5 (1.56%)	3 (3.30%)	1.126	0.289

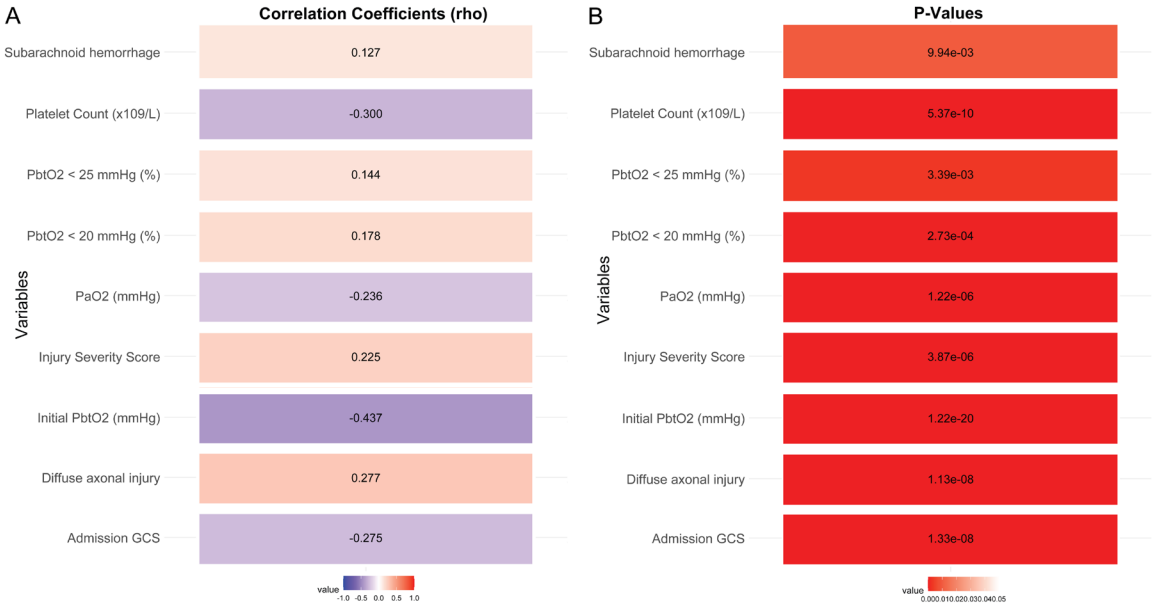

Figure 2. Correlation analysis between various influencing factors and poor neurological outcome after TBI. A. Correlation coefficient; B. *P* value. Notes: TBI: Traumatic brain injury; $PbtO_2$: Brain tissue oxygen pressure; PaO_2: Arterial oxygen partial pressure; GCS: Glasgow coma scale.

Table 6. Logistic regression analysis of various factors for poor neurological outcome post-TBI

Parameter	Coef	Odds ratio	B	beta	P
ISS ≥ 33.53	0.097	1.101	4.668	0.097	< 0.001
Admission GCS ≥ 8.7	0.271	0.762	5.838	-0.271	< 0.001
Initial $PbtO_2$ ≥ 25.04	0.259	0.772	8.27	-0.259	< 0.001
Diffuse axonal injury ≥ 2.42	1.197	3.310	6.583	1.197	< 0.001
Platelet count ≥ 194.31×10^9/L	0.021	0.979	6.338	-0.021	< 0.001
PaO_2 ≥ 89.38	0.088	0.916	4.652	-0.088	< 0.001
$PbtO_2$ < 20 mmHg (%)	1.105	3.021	3.499	1.105	< 0.001
$PbtO_2$ < 25 mmHg (%)	0.773	2.166	2.882	0.773	0.004
Subarachnoid hemorrhage	0.615	1.850	2.556	0.615	0.011

TBI: Traumatic brain injury; ISS: Injury severity score; GCS: Glasgow coma scale; $PbtO_2$: Brain tissue oxygen pressure.

25.04, OR: 0.772, P < 0.001) is associated with a decreased risk. The presence of diffuse axonal injury (diffuse axonal injury ≥ 2.42, OR: 3.310, P < 0.001) is significantly associated with an increased risk of poor outcomes. A higher platelet count (Platelet Count ≥ 194.31×10^9/L, OR: 0.979, P < 0.001) is associated with a decreased risk. A higher PaO_2 (PaO_2 ≥ 89.38, OR: 0.916, P < 0.001) is also associated with a decreased risk. Lower $PbtO_2$ ($PbtO_2$ < 20 mmHg, OR: 3.021, P < 0.001 and $PbtO_2$ < 25 mmHg, OR: 2.166, P = 0.004) is significantly associated with increased risks. Finally, the presence of subarachnoid hemor-

Table 7. The predictive value of various influencing factors for poor neurological outcome after TBI

Parameter	Sensitivities	Specificities	AUC	Youden index
ISS	0.418	0.872	0.657	0.290
Admission GCS	0.505	0.801	0.692	0.306
Initial $PbtO_2$ (mmHg)	0.813	0.648	0.804	0.461
Diffuse axonal injury	0.516	0.916	0.693	0.432
Platelet count ($\times 10^9$/L)	0.495	0.844	0.709	0.339
PaO_2 (mmHg)	0.571	0.735	0.665	0.306
$PbtO_2$ < 20 mmHg (%)	0.231	0.910	0.570	0.141
$PbtO_2$ < 25 mmHg (%)	0.319	0.822	0.571	0.141
Subarachnoid hemorrhage	0.582	0.570	0.576	0.152

TBI: Traumatic brain injury; ISS: Injury severity score; GCS: Glasgow coma scale; $PbtO_2$: Brain tissue oxygen pressure.

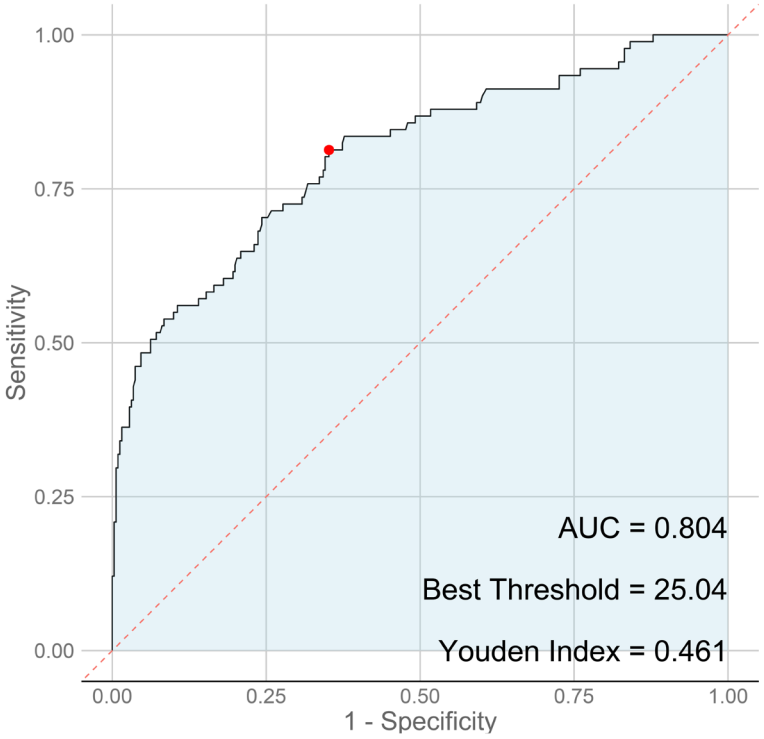

Figure 3. The predictive value of initial $PbtO_2$ for poor neurological outcome post-TBI. Notes: TBI: Traumatic brain injury; $PbtO_2$: Brain tissue oxygen pressure.

rhage (OR: 1.850, P = 0.011) is significantly associated with an increased risk. These findings highlight the importance of these clinical parameters in predicting and managing the risk of poor outcomes in TBI patients.

ROC

ROC analysis assessed the predictive value of various influencing factors for poor neurological outcome after TBI, revealing several parameters with meaningful predictive potential (**Table 7**). Specifically, initial $PbtO_2$ demonstrated the highest AUC at 0.804 (**Figure 3**), indicating strong predictive value for poor neurological outcomes. Additionally, admission GCS and platelet count also exhibited notable AUC values of 0.692 and 0.709, respectively. While ISS, PaO_2, and diffuse axonal injury showcased moderate AUC values of 0.657, 0.665, and 0.693, respectively. Although $PbtO_2$ below 20 mmHg, $PbtO_2$ below 25 mmHg, and the presence of subarachnoid hemorrhage demonstrated lower AUC values, they still provide some predictive value for poor neurological outcomes. The above results show that the initial $PbtO_2$ in predicting adverse neurological prognosis after TBI has potential utility.

Discussion

Traumatic brain injury (TBI) remains a major public health concern due to its profound impact on individuals and society. Understanding the factors that influence neurological prognosis following TBI is crucial for improving patient outcomes and guiding clinical decision-making [23-25].

The demographic and clinical characteristics of the study population revealed several noteworthy findings. Although age, gender, BMI, smoking history, alcohol consumption, hypertension, and diabetes did not significantly differ between patients with good and poor prognosis, ISS and admission GCS demonstrated clear associations with neurological outcomes. The ISS represents the severity of injuries across different body regions, whereas the GCS assesses the level of consciousness. As evidenced by our findings, a

higher ISS and lower GCS on admission were significantly associated with poor neurological prognoses, aligning with existing literature on the predictive value of these scoring systems. The association of high ISS and low GCS with poor prognosis underscores the critical role of injury severity and initial consciousness level in determining neurological outcomes after TBI [26, 27]. These results emphasize the importance of early and accurate assessment of injury severity and consciousness level for prognostication and targeted management strategies post-TBI.

The evaluation of $PbtO_2$ levels revealed intriguing findings, with initial $PbtO_2$ levels significantly higher in patients with good prognosis than in those with poor prognosis. Moreover, the percentage of $PbtO_2$ below certain thresholds (20 mmHg and 25 mmHg) differed significantly between the two prognosis groups. These findings suggest that $PbtO_2$ monitoring may offer valuable insights into managing cerebral oxygenation status post-TBI. The association of lower $PbtO_2$ levels with poorer prognosis aligns with the established role of cerebral hypoxia in TBI outcomes [28, 29]. It was well-established that maintaining adequate cerebral oxygenation is critical for minimizing secondary brain injury and enhancing recovery following TBI. The observed association between $PbtO_2$ levels and neurological prognosis underscores the potential utility of $PbtO_2$ monitoring as a prognostic tool in TBI management. Notably, the correlation and logistic regression analyses highlight the independent predictive value of $PbtO_2$ levels, particularly when they fall below certain thresholds, in determining neurological outcomes. The robust predictive potential of initial $PbtO_2$ levels, as indicated by AUC, underscores the clinical relevance of $PbtO_2$ monitoring in assessing and predicting neurological prognoses following TBI.

The radiological findings in our study revealed significant associations between certain imaging parameters and neurological prognoses. Specifically, the presence of diffuse axonal injury and subarachnoid hemorrhage demonstrated clear associations with poor neurological outcomes. Diffuse axonal injury, characterized by widespread damage to axonal fibers, was associated with cognitive and functional impairments [30-32], while subarachnoid hemorrhage can lead to increased intracranial pressure and cerebral complications [33-35]. The correlation of these radiological findings with poor neurological prognoses underscores the critical role of comprehensive neuroimaging assessments in understanding and predicting TBI outcomes. The identification of these specific radiological markers as independent risk factors for poor neurological prognosis emphasizes the need for meticulous radiological evaluation and interpretation in TBI management.

The analysis of laboratory parameters revealed notable associations between platelet count, PaO_2, and neurological prognoses. Specifically, lower platelet counts and PaO_2 levels were significantly associated with poor neurological outcomes. These findings align with the recognized impact of coagulation abnormalities and hypoxemia on TBI outcomes. Impaired platelet function and low PaO_2 levels can exacerbate secondary brain injury and negatively influence TBI recovery processes [36-38]. The association of these laboratory parameters with neurological outcomes highlights the potential utility of comprehensive laboratory assessments in prognostication and risk stratification following TBI. The predictive potential of platelet count and PaO_2 levels, as indicated by their AUC values in the ROC analysis, further emphasizes their clinical relevance in predicting neurological prognoses post-TBI.

The findings of this study underscore the multifactorial nature of prognostication in traumatic brain injury, aligning with existing literature [39] that emphasizes a comprehensive approach to evaluating TBI outcomes. Previous studies [40, 41] have consistently highlighted the predictive value of the GCS and ISS in assessing the initial severity of brain injuries. Our study corroborates these findings, demonstrating significant correlations between lower GCS and higher ISS scores with poor neurological outcomes [42]. These tools remain vital in the clinical setting, serving as reliable indicators of initial injury severity and facilitating early intervention planning.

While the present study offers valuable insights into the factors influencing neurological prognosis after TBI, several limitations should be

acknowledged. The retrospective nature and reliance on de-identified patient data may have introduced inherent biases and limitations in data collection and analysis. Prospective studies with larger sample sizes and extended follow-up periods are needed to validate and expand upon the current findings. Additionally, the single-center design may limit the generalizability of the results. Multi-center studies involving diverse patient populations and settings are warranted to corroborate the identified influencing factors and their associations with TBI prognosis.

Conclusion

In conclusion, this study underscores the prognostic significance of injury severity, brain tissue oxygenation, radiological findings, and laboratory parameters in determining neurological outcomes following TBI. These results deepen the understanding of the multifaceted determinants of TBI prognosis and suggest a potential role for $PbtO_2$ monitoring as a valuable tool in prognostic assessment. Further exploration and validation in prospective, multi-center studies are essential to refine prognostic models and enhance clinical decision-making in TBI management. Ultimately, a comprehensive evaluation of influencing factors identified in this study may contribute to the development of tailored, evidence-based strategies to optimize neurological prognosis and outcomes in TBI patients.

Disclosure of conflict of interest

None.

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