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Predictive value of intracranial pressure-related parameters and coagulation on the prognosis in patients with traumatic brain injury.

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Keywords: brain trauma; intracranial pressure; coagulation; prognosis.

Abstract. This study investigated the factors affecting the prognosis of patients with traumatic brain injury (TBI) and assessed the predictive value of intracranial pressure-related parameters and coagulation on prognosis. Seventy TBI patients admitted between January 2020 and January 2021 were categorized into good prognosis (n=42) and poor prognosis (n=28) groups according to the Glasgow Outcome Scale (GOS) score upon discharge. Factors affecting prognosis were analyzed, and differences in intracranial pressure and coagulation between the two groups were compared. The receiver operating characteristic curve (ROC) was used to calculate the predictive value of intracranial pressure-related parameters and coagulation function on prognosis. Within 24 h postoperatively, the good prognosis group had lower levels of intracranial pressure (ICP) and partial pressure of oxygen in brain tissue (PbtO2) and higher cerebral perfusion pressure (CPP) and cerebral hemodynamic parameters than the poor prognosis group ($p<0.05$). The good prognosis group had significantly lower prothrombin time (PT), activated partial thromboplastin time (aPTT),and prothrombin time (TT) levels and higher platelets (PLT) and fibrinogen (Fib) levels than the poor prognosis group ($p<0.05$). Regression analysis revealed that CPP, systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), mean blood flow velocity (Vm), PLT, Fib were independent protective factors for the prognosis, and ICP, PbtO2, PT, APTT, and TT were risk factors for prognosis. The ROC revealed that ICP, CPP, PbtO2, Vs, Vd, Vm, APTT, PLT, Fib exhibited high diagnostic value for poor prognosis (AUC=0.732, 0.940, 0.796, 0.706, 0.914, 0.729, 0.876, 0.709, 0.866), with ICP showing the

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highest diagnostic sensitivity and Vd showing the highest diagnostic specificity. Intracranial pressure-related parameters and coagulation indicators are prognosis-related indexes in patients with TBI, and the prognosis and survival can be improved by controlling intracranial pressure and improving coagulation.

Valor predictivo de los parámetros relacionados con la presión intracraneal y la coagulación en el pronóstico de pacientes con traumatismo craneoencefálico.

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Palabras clave: traumatismo cerebral; presión intracraneal; coagulación; pronóstico.

Resumen. Este estudio investigó factores influyentes en el pronóstico de pacientes con traumatismo craneoencefálico (TCE) y evaluó la utilidad predictiva de parámetros relacionados con la presión intracraneal y la coagulación. Setenta pacientes ingresados entre enero de 2020 y enero de 2021 se dividieron en dos grupos según su pronóstico: bueno (n=42) y malo (n=28), según la Escala de Resultados de Glasgow (GOS) al alta. Se analizaron factores de pronóstico y se compararon las diferencias en presión intracraneal y coagulación. La curva ROC evaluó el valor predictivo de estos parámetros. En las primeras 24 horas postoperativas, el grupo de buen pronóstico mostró menor presión intracraneal (PIC) y mayor presión de perfusión cerebral (CPP) y parámetros hemodinámicos cerebrales ($p<0,05$). También presentaron niveles más bajos de tiempo de protrombina (TP), tiempo parcial de tromboplastina (TTPA) y trombina (TT), y niveles más altos de plaquetas (PLT) y fibrinógeno (Fib) ($p<0,05$). El análisis de regresión identificó a CPP, la velocidad de flujo sistólico (Vs), la velocidad de flujo diastólico (Vd) , la velocidad de flujo medio (Vm), PLT y Fib como factores protectores independientes, mientras que PIC, oxígeno cerebral (PbtO2), TP, TTPA y TT fueron factores de riesgo. La ROC demostró que PIC, CPP, la presión parcial de oxigeno cerebral ($PbtO_2$), Vs, Vd, Vm, TTPA, PLT y Fib tenían un alto valor diagnóstico para mal pronóstico (AUC=0.732, 0.940, 0.796, 0.706, 0.914, 0.729, 0.876, 0.709, 0.866), siendo PIC el más sensible y Vd el más específico. Los parámetros intracraneales y de coagulación se relacionan con el pronóstico en pacientes con TCE, y mejorar el control de la PIC y la coagulación puede mejorar la supervivencia.

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INTRODUCTION

Traumatic brain injury (TBI) is an injury to the brain caused by a blow or jolt to the head from blunt or penetrating trauma where the dura mater remains intact while the contents of the cranial cavity have not yet come into contact with the outside. A blow or bump mainly causes it, or a jolt to the head, the head suddenly and violently hitting an object, or when an object pierces the skull and enters brain tissue [1,2]. The

skull of patients with TBI is deformed and fractured, causing movement of brain tissue in the cranial cavity, resulting in increased pressure on the cranial vault, compression of cerebral vessels, damage to the central nervous system, and inadequate vascular perfusion, which seriously threatens the patients' lives[3]. For now, monitoring of intracranial pressure and pre-and post-operative coagulation can aid in selecting surgical procedures, and strict control and adjustment of the intracranial pressure can minimize post-operative mortality in patients. However, their effect on post-operative recovery and prognostic outcome still needs to be explored [4]. Seventy patients with TBI were enrolled in this study, and the effects of intracranial pressure-related parameters and coagulation indicators on prognosis were explored, aiming to provide new ideas for improving the quality of prognosis of TBI patients.

MATERIALS AND METHODS

Baseline data

Seventy patients with TBI admitted to The First People's Hospital of Yibin between January 2020 and January 2021 were enrolled, and patients with Glasgow Outcome Scale (GOS) [5] scores of grades III-IV were classified as the good prognosis group (n=42), and patients with GOS scores of grades I-II or death were defined as the poor prognosis group (n=28). The study has been approved by the medical ethics committee of The First People's Hospital of Yibin. The research objects and their families were informed, and they signed a fully informed consent form.

Inclusion criteria: TBI was confirmed by cranial CT scan, and history of cranial trauma was determined; patients with TBI confirmed by clinical examination; patients who sought medical help within a short period after the onset of the disease, and there might be progression; patients who received craniocerebral trauma surgery in our hospital. Exclusion criteria: TBI combined with more severe organ dysfunction; combined or with other coagulation-related diseases; developed critical and life-threatening conditions after admission, with a predicted survival time of less than three months; incomplete medical records and lost to follow-up.

METHODS

After patients were enrolled, baseline data was collected from both groups, including gender, age, cause of injury, Glasgow coma scale (GCS) upon admission, time from injury to admission, length of stay in the neurosurgical intensive care unit (NICU), and parameters related to intracranial pressure (ICP).

In both groups, 5 mL of venous blood were collected from the anterior region of the elbow in a fasting state, followed by centrifugation (3000 r/min, 10 min) at 4 °C to obtain platelet-poor plasma, and was stored at -80 °C. The coagulation indicators were measured by an optical coagulation analyzer (BIObase, XL1000C, China), including prothrombin time (PT), activated partial thromboplastin time (APTT), thrombin time (TT), platelets (PLT), and fibrinogen (Fib) [6,7].

TBI surgery was determined according to the Guidelines for the Treatment of Major Craniocerebral Trauma (4th edition), and ICP, partial oxygen pressure ($PbtO_2$), cerebral perfusion pressure (CPP), and cerebral blood flow velocity were recorded within 24 h after surgery [8,9]. ICP was monitored using an intracranial pressure monitor (HP Inc., USA). ICP levels and ICP waveform amplitude were recorded every hour, and non-invasive mean ICP was calculated according to the potential superposition principle. With a GMS-5002 oxygen sensor (GMS, Germany), $PbtO_2$ was measured during surgery and 24 h after surgery with a normal brain area in the lateral brain as the puncture point by piercing the electrode and probing the needle about 34 mm deep into the meninges. The recording interval was 15 seconds. The arterial blood pressure was monitored using a non-invasive blood pressure monitor (HP,

USA). The instrument depicted pressure waves by arterial blood pressure fluctuations. The mean arterial blood pressure was calculated with multiple sets of pressure waves. Cerebral hemodynamics was monitored using an EK-1000B transcranial Doppler ultrasound instrument (Jiangsu Yikang Electronic Technology Co., Ltd., 201182230620) with a probe frequency of 2 MHz to detect cerebral blood flow velocity in the cerebral arteries, including systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), and mean blood flow velocity (Vm) [10].

Follow-up visits

Patients were followed up consistently for three months after discharge, and GOS scores were recorded. Grade V (low disability): light damage with minor neurological and psychological deficits; Grade IV (moderate disability): no need for assistance in everyday life; employment was possible but may require special equipment; Grade III (Severe disability): severe injury with permanent need for help with daily living, Grade II (Persistent vegetative state): severe damage with prolonged state of unresponsiveness and a lack of higher mental functions; Grade I (Death): severe injury or death without recovery of consciousness.

Statistical analysis

The data of this study were analyzed using IBM® SPSS 22.0 software. The count data were expressed as n and % and examined by the χ^2 test. The measurement data were expressed by ($\bar{x}\pm$sd) and examined by the t-test. The correlation of intracranial pressure-related parameters with coagulation function and poor prognosis was analyzed using the Pearson correlation analysis. Binary logistic regression was used to analyze the factors influencing the prognosis of TBI, and the ROC was used to calculate the predictive value of intracranial pressure-related parameters and coagulation for prognosis. An area under the curve (AUC) of 0.7 to 0.9 represents a specific predictive value, and an AUC > 0.9 represents a higher predictive value. $p<0.05$ represents a statistically significant difference.

RESULTS

Comparison of baseline data

There were no significant differences between the two groups in terms of gender, age, cause of injury, GCS score upon admission, time from injury to admission, duration of ICP-related parameter monitoring, and duration of NICU stay ($p>0.05$) (Table 1).

Comparison of intracranial pressure-related parameters between the two groups

Within 24 h after surgery, patients in the good prognosis group had significantly lower ICP and $PbtO_2$ levels and significantly higher CPP and cerebral hemodynamic indexes than those in the poor prognosis group ($p<0.05$) (Table 2).

Changes in coagulation in both groups

Within 24 h after surgery, patients in the good-prognosis group had significantly lower PT, APTT, and TT levels and significantly higher PLT and Fib levels than those in the poor-prognosis group ($p<0.05$) (Table 2).

The predictive value of intracranial pressure-related parameters and coagulation on prognosis

ICP, $PbtO_2$, PT, APTT, TT were significantly positively correlated with poor prognosis, and CPP, Vs, Vd, Vm, PLT, and Fib were significantly negatively correlated with poor prognosis ($p<0.05$, Table 3). Logistic regression analysis revealed that CPP, Vs, Vd, Vm, PLT, and Fib were prognostic independent protective factors, and ICP, $PbtO_2$, PT, APTT, and TT were prognostic risk factors (Table 4). ICP, CPP, $PbtO_2$, Vs, Vd, Vm, APTT, PLT, and Fib exhibited high diagnostic value for poor prognosis (AUC>0.7), with ICP showing the highest diagnostic sensitivity and Vd showing the highest diagnostic specificity (Table 5 and Fig. 1).

Table 1
Comparison of general information between the two groups.

Variable		Good prognosis group (n=42)	Poor prognosis group (n=28)	t/χ^2 values	*p*
Gender (m/f, n)		23/19	15/13	0.010	0.922
Age (years)		41.72±14.48	43.88±15.36	0.597	0.553
Cause of injury	Blow	8 (19.05)	6 (21.43)	0.060	0.807
	Fall	11 (26.19)	8 (28.57)	0.048	0.826
	Traffic accidents	16 (38.10)	11 (39.29)	0.010	0.920
	Other	7 (16.67)	3 (10.71)	0.486	0.486
Admission GCS score		8.45±3.04	7.84±2.42	0.890	0.377
Time from injury to admission (hours)		2.25±1.53	2.40±1.32	0.424	0.673
Duration of ICP-related parameters monitoring (hours)		148.68±10.72	151.76±12.19	1.115	0.269
Length of stay in NICU (hours)		90.59±40.43	79.48±48.37	1.041	0.302

Glasgow Coma Score (GCS), Intracranial Pressure (ICP), Neurosurgical Intensive Care Unit (NICU).

Table 2
Comparison of intracranial pressure-related parameter and coagulation parameters between the two groups.

Variable		Good prognosis group (n=42) ($\bar{x}$±sd)	Poor prognosis group (n=28) ($\bar{x}$±sd)	t/χ^2 values	*p*
ICP (mmHg)		14.00±5.70	26.25±13.49	5.239	≤0.001
CPP (mmHg)		81.18±6.94	48.98±19.95	9.650	≤0.001
$PbtO_2$ (mmHg)		9.17±5.62	19.31±9.58	5.580	≤0.001
Hemodynamics (cm/s)	Vs	84.75±13.24	75.24±11.30	3.117	0.003
	Vd	57.86±7.50	46.53±4.75	7.092	≤0.001
	Vm	53.44±6.61	46.38±8.85	3.818	≤0.001
PT (s)		13.27±2.89	15.47±3.10	3.031	0.003
APTT (s)		30.52±3.63	37.93±5.54	6.769	≤0.001
TT (s)		17.30±3.18	19.29±3.98	2.318	0.023
PLT (10^9/L)		148.62±45.69	119.41±30.28	2.972	0.004
Fib (g/L)		2.51±0.35	2.12±0.15	6.399	≤0.001

Intracranial pressure (ICP), cerebral perfusion pressure (CPP), partial pressure of oxygen in brain tissue ($PbtO_2$), systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), mean blood flow velocity (Vm), prothrombin time (PT), activated partial thromboplastin time (aPTT), prothrombin time (TT), seconds (s), platelets (PLT), fibrinogen (Fib).

DISCUSSION

Closed craniocerebral trauma is a common and severe TBI, and its incidence increases year by year. Compared with open craniocerebral trauma, the symptoms and signs of closed craniocerebral trauma are less obvious, but its potential harm cannot be ignored. Patients with closed craniocerebral trauma not only face the threat of life safety but also may lead to long-term neurological dysfunction and decreased quality of life.

Table 3

Correlation analysis of intracranial pressure-related parameters, coagulation and poor prognosis.

Variable	ICP	CPP	$PbtO_2$	Vs	Vd	Vm	PT	APTT	TT	PLT	Fib
r	0.394	-0.746	0.502	-0.349	-0.703	-0.388	0.316	0.638	0.257	-0.355	-0.559
p	0.001	≤0.001	≤0.001	0.003	≤0.001	0.001	0.008	≤0.001	0.032	0.003	≤0.001

Intracranial pressure (ICP), cerebral perfusion pressure (CPP), partial pressure of oxygen in brain tissue ($PbtO_2$), systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), mean blood flow velocity (Vm), prothrombin time (PT), activated partial thromboplastin time (aPTT), prothrombin time (TT), platelets (PLT), fibrinogen (Fib), r (Pearson correlation coefficient).

Table 4

Factors influencing the prognosis of patients with TBI.

Variable	B	S.E.	Wald	p	Exp (B)	95% EXP (B) Confidence interval	
						lower limit	upper limit
ICP	0.121	0.032	14.458	≤0.001	1.129	1.060	1.201
CPP	-0.204	0.051	16.078	≤0.001	0.815	0.738	0.901
PbtO2	0.169	0.043	15.266	≤0.001	1.184	1.088	1.289
Vs	-0.065	0.024	7.511	0.006	0.937	0.894	0.982
Vd	-0.286	0.068	17.716	≤0.001	-798	0.657	0.858
Vm	-0.124	0.039	10.168	0.001	0.883	0.818	0.953
PT	0.250	0.091	7.499	0.006	1.284	1.074	1.535
APTT	0.358	0.084	18.367	≤0.001	1.431	1.215	1.686
TT	0.163	0.074	4.805	0.028	1.177	1.017	1.361
PLT	-0.018	0.007	7.422	0.006	0.982	0.969	0.995
Fib	-4.780	1.213	15.533	0.000	0.0080	0.001	0.090

B>1 is a risk factor and B<1 is a protective factor.

Intracranial pressure (ICP), cerebral perfusion pressure (CPP), partial pressure of oxygen in brain tissue (PbtO2), systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), mean blood flow velocity (Vm), prothrombin time (PT), activated partial thromboplastin time (aPTT), prothrombin time (TT), platelets (PLT), fibrinogen (Fib).

Predicting the prognosis of patients with closed craniocerebral trauma has been the focus of the medical community. Understanding the prognostic influencing factors and effective predictive indicators can help doctors better assess patients' conditions and predict their future development trends and provide an important basis for clinical decision-making to take timely intervention measures to improve patient prognosis.

TBI is frequent in the neurosurgical emergency setting, with a mortality rate of 30%-50% for severe TBI, resulting in disability or other types of complications. For TBI, a hard blow to the head could cause an intracranial hematoma, cerebral edema, brain herniation due to brain tissue dislocation, and elevated ICP (ICP >20 mm Hg for more than 15 min within one hour of the blow), which threaten the life and health of the patient [11,12]. Due to an increase in deadly traffic accidents, falls on construction sites, and collisions, the incidence of TBI is showing an increasing trend [13]. For the current treatment options of TBI, decompressive craniectomy and cerebrospinal fluid drainage, nutritional support, and ICP monitoring can significantly reduce the length of

Table 5
Analysis of the predictive value of intracranial pressure-related parameters and coagulation for poor prognosis.

Variable	AUC	Jorden index	Sensitivity	Specificity	Standard Error	Asymptotic 95% confidence interval		
						p	lower limit	upper limit
ICP	0.732	0.547	0.976	0.571	0.069	0.001	0.598	0.867
CPP	0.940	-0.834	0.905	0.071	0.036	≤0.001	0.869	1.000
$PbtO_2$	0.796	0.523	0.952	0.571	0.055	≤0.001	0.687	0.905
Vs	0.706	-0.416	0.452	0.036	0.062	0.004	0.585	0.827
Vd	0.914	0.095	0.893	0.893	0.037	≤0.001	0.841	0.987
Vm	0.729	-0.488	0.952	0.464	0.064	0.001	0.604	0.854
PT	0.686	0.345	0.881	0.464	0.065	0.009	0.559	0.814
APTT	0.876	0.679	0.929	0.750	0.049	≤0.001	0.779	0.973
TT	0.651	0.310	0.881	0.429	0.069	0.033	0.516	0.787
PLT	0.709	-0.464	0.500	0.036	0.063	0.003	0.586	0.833
Fib	0.866	0.691	0.929	0.762	0.047	≤0.001	0.775	0.958

Area under the curve (AUC) Intracranial pressure (ICP), cerebral perfusion pressure (CPP), partial pressure of oxygen in brain tissue ($PbtO_2$), systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), mean blood flow velocity (Vm), prothrombin time (PT), activated partial thromboplastin time (aPTT), prothrombin time (TT), platelets (PLT), fibrinogen (Fib).

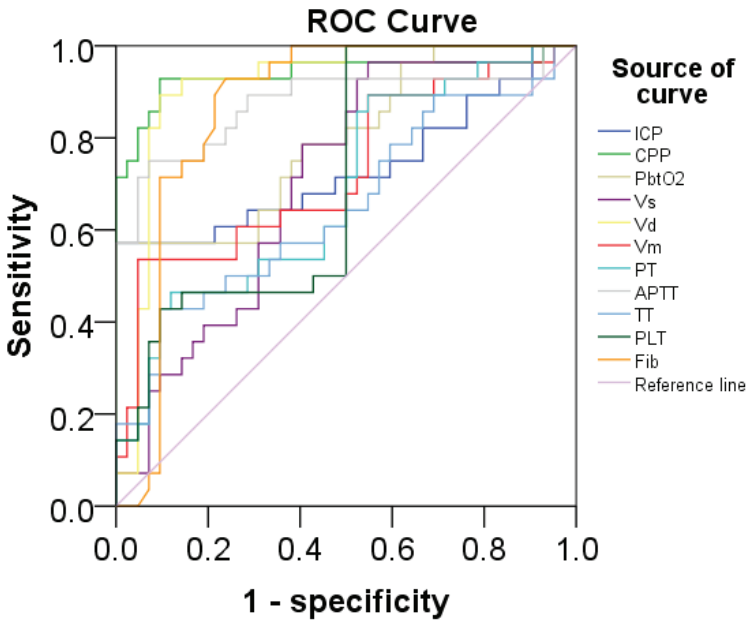

Fig. 1. ROC analysis of the predictive value of intracranial pressure and coagulation function on adverse prognosis. Intracranial pressure (ICP), cerebral perfusion pressure (CPP), partial pressure of oxygen in brain tissue ($PbtO_2$), systolic blood flow velocity (Vs), end-diastolic blood flow velocity (Vd), mean blood flow velocity (Vm), prothrombin time (PT), activated partial thromboplastin time (aPTT), prothrombin time (TT), platelets (PLT), fibrinogen (Fib).

hospitalization and mortality of patients, indicating that there is a close link between the quality of prognosis and ICP levels [14].

In a study by Pan *et al.* [15] on factors affecting the prognosis of TBI patients, patients with favorable outcomes had significantly lower average ICP and pressure reactivity index (PRx) and exhibited significantly higher CPP compared with patients with unfavorable outcomes. ICP is the pressure exerted by fluids such as cerebrospinal fluid (CSF) inside the skull and on the brain tissue. When an external blow to the cranium triggers cerebral edema, there is an increase in CSF as well as the volume of brain tissue. Other space-occupying lesions, such as hematomas, may also occur, causing obstruction of the CSF circulation and increasing the CSF pressure and ICP in the cranial cavity [16,17]. In this study, we enrolled patients with TBI during the same period and found that the ICP and $PbtO_2$ levels within 24 h after surgery in patients with good prognosis were significantly lower while CPP was significantly higher than those in the poor prognosis group, which is consistent with the findings of other scholars mentioned above [16,17].

Meanwhile, this study analyzed hemodynamic indices and found that all cerebral hemodynamics in the poor prognosis group were lower than those in the good prognosis group. The results of the correlation analysis showed a significant negative relationship between CPP, Vs, Vd, Vm, PLT, and Fib and poor prognosis ($r= -0.746, -0.349, -0.703, -0.388, -0.355, -0.559$, $p <0.05$). This implies an association between decreased cerebral perfusion pressure, venous suction velocity, velocity of arterial drainage, brain parenchymal volume, PLT count, and Fib and a poor outcome. Lower cerebral perfusion pressure, venous suction velocity, arterial drainage velocity, brain parenchyma volume, PLT count, and Fib may cause brain tissue hypoxia, insufficient perfusion, and impaired coagulation, affecting the patient's recovery.

In the ROC diagnostic curve, ICP, CPP, $PbtO_2$, Vs, Vd, Vm, APTT, PLT, and Fib all had high diagnostic values for poor prognosis. Vs, Vd, and Vm were all independent protective factors for the prognosis of TBI patients. The reason may be that the cerebral vascular has regulatory functions. When cranial injury occurs, ICP is elevated, disturbing brain tissue blood oxygen levels producing a large amount of hemoglobin, 5-hydroxytryptamine, and angiotensin. The cerebral vascular regulatory center alleviates blood oxygen deficiency by regulating ICP, thus repairing damaged brain nerves and protecting the intracranial microcirculatory balance.

Brain tissue is the organ with the highest level of prothrombin kinase. Under the influence of cranial damage and ICP, coagulation dysfunction of brain tissue also affects intracranial blood-oxygen balance, which affects the outcome and prognosis of TBI. In terms of the development pathway of coagulation dysfunction in TBI, when the cranial brain is damaged, and the blood-brain barrier is impaired, the extrinsic coagulation mechanism is activated by stress, and a large amount of thrombin is released from brain tissue into the blood circulation, resulting in a local hypercoagulable state in the cranial brain [18]. In the good prognosis Group, PT, APTT, and TT levels were significantly lower, while PLT levels were higher than those in the poor prognosis group, and all factors were independent influencing factors for poor prognosis. This suggests that with impaired coagulation mechanisms, platelet levels may decrease rapidly. Platelets play a crucial role in blood coagulation and can accumulate on damaged blood vessel walls to form a thrombus for hemostasis. If platelet numbers are reduced or their function is abnormal, it may lead to decreased coagulability, increasing the risk of bleeding. Brain tissue is very important for adequate blood oxygen supply, and the decline of platelets may lead to unbalanced cerebrovascular regulation, further affecting the blood oxygen level of brain tis-

sue and impairing brain function [19,20]. This is probably because TBI causes local tissue damage and bleeding, releasing a large number of tissue factors and cytokines that activate the coagulation system. However, when the injury is too severe or lasts too long, the coagulation function may be inhibited, and the platelet aggregation and blood coagulation capacity may be reduced. This leads to a decrease in the platelet levels. Second, TBI may lead to the rupture and bleeding of the capillaries in the brain tissue. Platelets accumulate at vascular damage and seal the ruptured capillaries by forming platelet primary hemostatic plugs to prevent further bleeding. Therefore, when thrombocytopenia occurs, it may indicate damage to platelet consumption and further bleeding. In addition, platelets release secretions such as serotonin, dopamine, and angiotensin, which are involved in vascular regulation and hemodynamic balance in the lining of blood vessels. When platelet levels decrease, this cerebrovascular regulation mechanism may be affected, leading to abnormal blood oxygen levels in brain tissue, further impairing cerebrovascular regulation. Therefore, the decrease in platelet levels may be one of the manifestations of impaired coagulation mechanisms and abnormal cerebrovascular regulation in patients with TBI. These changes may impact the prognosis of patients with TBI, and further studies can explore these associations and determine their predictive value for the prognosis of patients with TBI. However, this study only analyzed the correlation between intracranial pressure-related parameters, coagulation indexes, and prognosis without correlating intracranial pressure with coagulation indexes, and further exploration is still needed to determine their relationship.

In conclusion, intracranial pressure-related parameters and coagulation indicators are all related to the prognosis of patients with TBI, and the prognostic quality and survival of patients can be improved by regulating intracranial pressure and improving coagulation function.

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Data Availability

Due to the nature of this research, participants did not agree for their data to be shared publicly, so supporting data is unavailable.

Conflicts of interest

The authors declare that they have no competing interests.

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Participation in development and writing of the paper

Concept and design: LS and FC. Acquisition of data, literature review, and refinement of manuscript: All authors. Analysis and interpretation of data: HL and SY. Manuscript writing: LS. Review of final manuscript: FC.

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