Transcranial Doppler Ultrasonography for the Management of Severe Traumatic Brain Injury After Decompressive Craniectomy

Tao Chang, Lihong Li, Yanlong Yang, Min Li, Yan Qu, Li Gao

BACKGROUND: **Cerebral hemodynamic transformation is a relatively common finding in patients with traumatic brain injury (TBI). Knowledge of cerebral hemodynamic disturbance may assist in predicting the management outcome. Transcranial Doppler ultrasonography (TCD) monitoring of patients with TBI can be used to reveal various pathologic hemodynamic changes. The objective of this study was to compare the clinical outcomes of postoperative routine intracranial pressure (ICP) monitoring versus ICP monitoring combined with TCD monitoring in patients with brain trauma after decompressive craniectomy.**

METHODS: **This was a retrospective study of 30 patients with TBI who underwent ICP combined with TCD monitoring (after 2015) compared with a historical control group of 30 patients who only underwent routine ICP monitoring (in 2013–2014). ICP, partial pressure of carbon dioxide, hemoglobin, and hematocrit values were monitored and recorded on a daily basis for 7 days after operation. Neuroimaging was also performed at admission. Neurologic outcome was assessed at 2 weeks and 6 months after operation using the Glasgow Outcome Score Extended (GOS-E). Unconditional multivariable logistic regression was conducted to analyze the factors for favorable clinical outcome.**

RESULTS: **Two weeks after operation, there were no differences in mortality rate between the 2 groups ($P = 0.643$). When considering the GOS-E score at 6 months, there were no differences in clinical prognosis between the 2 groups ($P = 0.101$), but the ICP combined with TCD monitoring group showed a higher frequency of patients with favorable outcome compared with the routine ICP monitoring group ($P = 0.043$). Unconditional multivariable logistic regression results showed that no factor was independently associated with GOS-E at 6 months.**

CONCLUSIONS: **TCD could be helpful for the serial monitoring of cerebral hemodynamic changes after decompressive craniectomy for TBI, which could be beneficial for neurologic outcome improvement.**

INTRODUCTION

Traumatic brain injury (TBI) is the structural injury and/or physiologic disruption of brain functions from any type of injury.[1] Elevated intracranial pressure (ICP) in patients with severe TBI is associated with high mortality and poor clinical outcomes.[2] Guidelines recommend cerebral perfusion pressure (CPP) values of 50–70 mm Hg and ICP lower than 20 mm Hg for the management of acute TBI.[1,3] Nevertheless, adequate individual targets are still poorly met because different patients have altered perfusion thresholds. Hypotension below

Key words
- Cerebral hemodynamics
- Decompressive craniectomy
- Intracranial pressure
- Transcranial Doppler ultrasonography
- Traumatic brain injury

Abbreviations and Acronyms
CBF: Cerebral blood flow
CBFV: Cerebral blood flow velocity
CI: Confidence interval
CPP: Cerebral perfusion pressure
DC: Decompressive craniectomy
EICA: Extracranial internal carotid artery
GOS-E: Glasgow Outcome Scale-Extended
ICP: Intracranial pressure
LR: Lindegaard ratio
MCA: Middle cerebral artery
OR: Odds ratio
PI: Pulsatility index
TBI: Traumatic brain injury
TCD: Transcranial Doppler ultrasonography
Vedv: End-diastolic velocity
Vmean: Mean velocity

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optimal CPP is associated with high mortality, whereas hypertension above optimal CPP is associated with an increased risk of severe disability.[4,5] Pathologic increase in ICP can be surgically managed with ventricular cerebrospinal fluid drainage and decompressive craniectomy (DC).[6] Nevertheless, the benefits of DC are controversial, as reviewed by Moon and Hyun.[6] It is well known that the outcomes of TBI after DC could be associated with changes in ICP and that promptly managing ICP changes could improve the patient outcomes.

ICP monitoring combined with additional neurologic monitoring measures could help understand the pathophysiology of secondary brain injury.[7] Transcranial Doppler ultrasonography (TCD) is a noninvasive, easy, and fast tool to measure cerebral blood flow velocity (CBFV) and distal vascular resistance, providing useful data about the adequacy of cerebral blood flow (CBF) in the course of TBI. TCD is widely used for the evaluation of many vascular diseases by the increase or decrease in CBFV.[8-10]

Nevertheless, it is currently unknown if TCD could allow the early detection of abnormal hemodynamics in patients with TBI and whether a targeted protocol based on ICP monitoring and TCD results could improve the clinical outcome of TBI. Therefore, the objective of this study was to compare the postoperative clinical outcomes of routine ICP monitoring versus ICP monitoring combined with TCD monitoring in patients with TBI after DC.

MATERIALS AND METHODS

Study Design and Patients

This was a retrospective study of 30 patients with TBI who underwent postoperative ICP monitoring combined with TCD monitoring (in 2015) compared with a historical control group of 30 patients who underwent routine ICP monitoring (in 2013–2014). All patients were treated in the intensive care unit of Second Affiliated Hospital of Air Force Medical Universitary. Among 423 patients with TBI who were admitted into our departments and treated in 2013–2015, 60 patients met the following inclusion criteria: 1) 14–65 years of age; 2) Glasgow Coma Scale score $\leq$8; 3) underwent DC; and 4) underwent ICP monitoring. The exclusion criteria were as follows: 1) penetrating TBI; 2) multiple organ damage; and 3) time from injury to operation >12 hours. Before 2015, the standard ICP monitoring aimed at maintaining ICP <20 mm Hg and CPP 50–70 mm Hg. Starting in 2015, TCD monitoring aimed at maintaining ICP <20 mm Hg and normal cerebral hemodynamics.[11] This study was approved by the ethics committee of Second Affiliated Hospital of Air Force Medical Universitary. The committee waived the need for informed consent.

Management Protocol, DC, and ICP

All patients included in the study received neurocritical care management.[12] DC indications and approaches were according to the guidelines.[13] ICP was monitored continuously for 7 days postoperation using a parenchymal catheter (Codman, REF-82663I [Johnson & Johnson Professional Inc., Raynham, MA, USA]). CPP was calculated as the difference between the mean arterial pressure and ICP. All DC operations were performed by an associate chief surgeon with 12 years of experience.

TCD Monitoring and Cerebral Hemodynamic Patterns

The bilateral middle cerebral artery (MCA)-M1 segment and the distal segment of the extracranial internal carotid artery (EICA) were monitored using a portable 2-MHz pulsed TCD device (Pioneer TC 2020 EME [Nicolet Biomedical, Inc., Madison, Wisconsin, USA]) via the temporal ultrasound windows. We selected the imaging depth according to the one showing the highest mean velocity (Vmean). Serial measurements of pulsatility index (PI), end-diastolic velocity (Vedv), and Vmean in the middle cerebral artery were recorded for 7 days after DC. TCD was considered abnormal when 2 of the 3 measured values were abnormal: Vmean <40 cm/s, Vedv <20 cm/s, and PI >1.4, which were defined as indicative of cerebral hypoperfusion. Vmean between 120 and 200 cm/s required additional clinical information to improve diagnostic accuracy. The Lindegaard ratio (LR), defined as the ratio of the MCA Vmean/EICA Vmean, was used to differentiate cerebral vasospasm from hyperemia. MCA Vmean between 40 and 120 cm/s along with an LR <3 was regarded as a nonspecific hemodynamic pattern. In the presence of hyperemia, both EICA and MCA mean flow velocities are raised, with an LR <3, whereas vasospasm would preferentially raise the MCA flow over the internal carotid artery, with an LR >3.[11]

Three qualified sonographers (all with >4 years of experience) conducted TCD and recorded the ICP values. The protocol for assessment and treatment of the cerebral hemodynamics by TCD are as follows (Figure 1). First, when ICP is $\leq$20 mm Hg and stable vital signs and other clinical factors which may affect the CBF are normal, TCD is used to monitor the CBF every 4 hours. However, it is necessary to reassess the CBF by TCD until 4 hours later or the disease progressed. Second, when some clinical and laboratory factors which may affect the CBF are severely abnormal, we conduct the TCD monitoring before and after correcting the abnormal clinical and laboratory parameters. When these factors are corrected, and the CBF is still abnormal, we follow the treatment algorithm to correct the abnormal CBF and maintain the therapy until it is necessary to reassess the CBF by TCD 4 hours later or the disease progressed. Third, when it is the reason of the pathophysiology of secondary brain injury for the abnormal CBF, it is necessary to reassess the CBF by TCD in these cases: taking targeted treatment of 20–30 minutes, CPP increased or decreased 10–15 mm Hg, ICP lower 20 minutes. When the CBF is normal, it is necessary to maintain the therapy until 4 hours later or the disease progressed. When the CBF remains abnormal, it is necessary to reevaluate the condition and accommodate the treatment until the CBF is normal.

Data Collection

Demographics (age and sex), injury details (date of accident, time interval between accident and hospital admission, time interval between hospital admission and operation, and mechanism of TBI), neurologic status (Glasgow Coma Scale score and pupil response on admission and before and after DC), physiologic parameters, ICP, cerebral hemodynamic parameters, cerebral hemodynamic status, and clinical outcome were recorded.

Follow-Up and Clinical Outcome Assessment

The clinical neurologic outcome was evaluated at 2 weeks and 6 months after DC using the Glasgow Outcome Scale-Extended

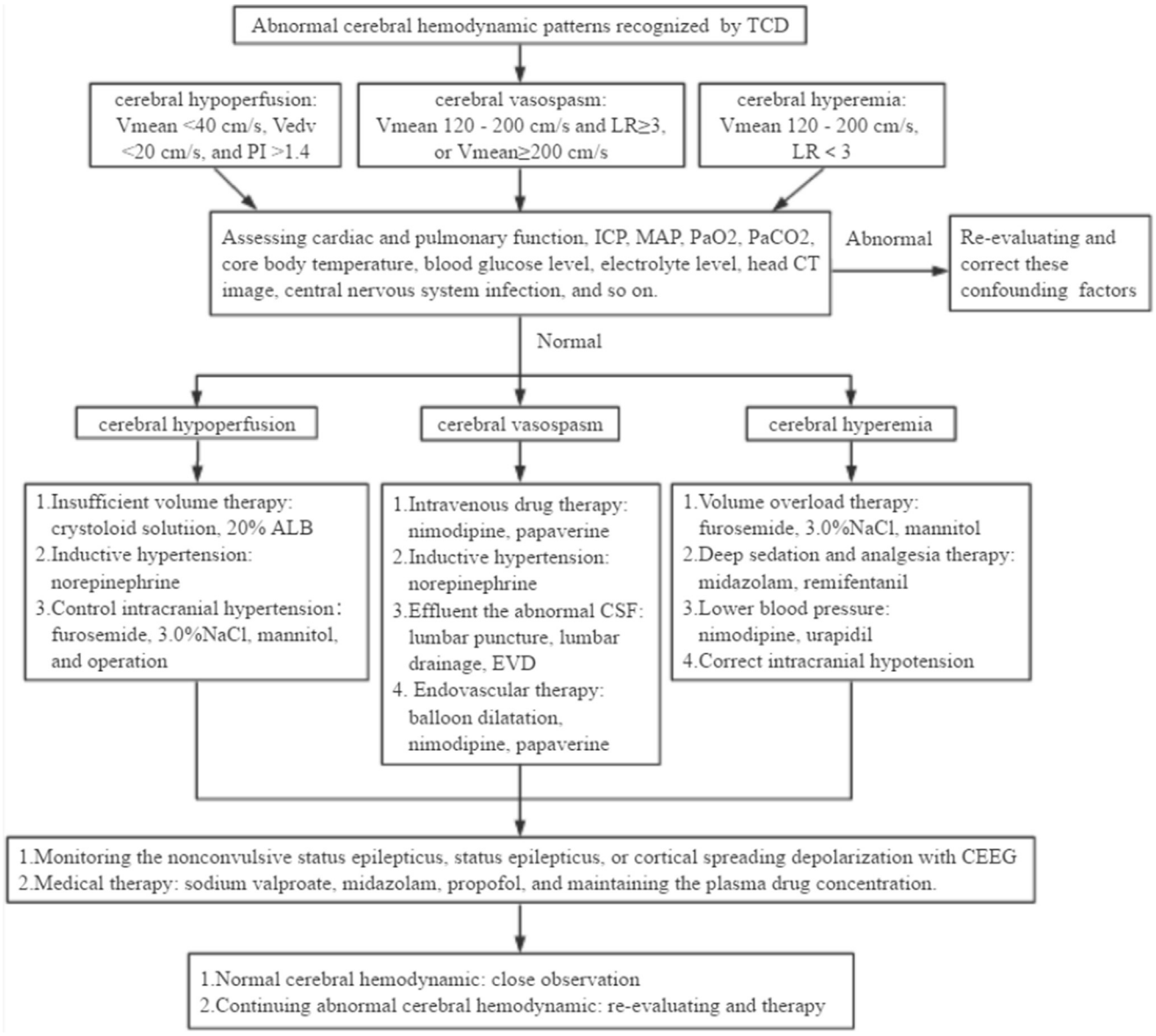

Figure 1. Assessment and treatment of the abnormal cerebral hemodynamics in patients with traumatic brain injury after decompressive craniectomy. ALB, alblumin; CEEG, continuous electroencephalogram; CSF, cerebrospinal fluid; CT, computed tomography; EVD, external ventricular drainage; ICP, intracranial pressure; LR, Lindegaard ratio; MAP, mean arterial pressure; PaCO2, partial pressure of carbon dioxide; PI, pulsatility index; TCD, transcranial Doppler ultrasonography; Vedv, end-diastolic velocity; Vmean, mean velocity; PaO2, partial pressure of oxygen.

(GOS-E). Patients with scores of 1–4 were considered to have a poor outcome, and those with scores of 5–8 were considered to have a favorable outcome.[14]

Statistical Analysis

Continuous data were presented as means ± SDs and analyzed using the Student t test. Categorical data were presented as frequencies and analyzed using the Fischer exact test. Unconditional multivariable logistic regression was conducted to analyze the risk factors of favorable clinical outcome for these patients with TBI. SPSS 19.0 (IBM, Armonk, New York, USA) was used for statistical analysis. Two-sided P values <0.05 were considered statistically significant.

RESULTS

Characteristics of the Patients

There were no differences regarding the characteristics of the patients, except regarding osmotherapy ($P = 0.004$) and sedation therapy ($P = 0.034$) between the ICP monitoring group and ICP combined with TCD monitoring group (Table 1).

ICP, Biochemistry, and Blood Gas During the First Week After Operation

During the next 7 days after operation, ICP peaked on the fourth day in the 2 groups, but there were no significant differences between them (14.4 ± 4.1 vs. 15.9 ± 4.1 mm Hg, P = 0.168). There were no differences in partial pressure of carbon dioxide, hemoglobin, and hematocrit between the 2 groups (all P > 0.05) (Figures 2–5).

Cerebral Hemodynamic Parameters (Vmean, Vedv, and PI) and Abnormal Cerebral Hemodynamic Status During the First Week After Operation

During the next 7 days after operation, PI reached the highest on day 4 (1.59 ± 0.96), whereas Vedv and Vmean were lowest on day 3 (41.69 ± 21.57 and 75.73 ± 28.53, respectively) (Figure 6). The incidence of cerebral hyperemia was 13.3% on day 1 and roughly

Table 1. Characteristics of the Patients

Clinical Characteristics	ICP Monitoring Group (n = 30)	ICP Combined with TCD Monitoring Group (n = 30)	*P* Value
Male	17 (56.7)	21 (70)	0.284
Age (years)	42.00 ± 11.96	38.50 ± 14.26	0.307
GCS score on admission	5.50 ± 1.53	5.73 ± 1.62	0.568
Pupil			0.724
Normal	11 (36.7)	14 (46.7)	
Unilateral mydriasis	15 (50.0)	13 (43.3)	
Bilateral mydriasis	4 (13.3)	3 (10.0)	
Mechanism of injury			0.882
Traffic accident	13 (43.3)	15 (50.0)	
Fall	10 (33.3)	8 (26.7)	
Attack	4 (13.3)	5 (16.7)	
Unknown	3 (10.0)	2 (6.7)	
Cranial CT scan			0.961
EDH	5 (16.7)	4 (13.3)	
SDH	7 (23.3)	6 (20.0)	
Subarachnoid hemorrhage	9 (30.0)	10 (33.3)	
MABP (mm Hg)	90.57 ± 10.7	92.67 ± 12.41	0.487
$PaCo_2$ (mm Hg)	34.13 ± 3.51	32.40 ± 5.86	0.171
HGB (g/L)	94.27 ± 20.96	99.67 ± 15.50	0.262
Hematocrit (%)	32.00 ± 6.73	30.13 ± 5.82	0.255
Time from trauma to hospital (hours)	8.73 ± 3.81	8.37 ± 5.14	0.168
Time from trauma to operation (hours)	13.10 ± 3.97	14.93 ± 4.91	0.117
Osmotherapy			0.004
Mannitol	15 (50.0)	5 (16.7)	
Hypertonic saline	7 (23.3)	19 (63.3)	
Mannitol + hypertonic saline	8 (26.7)	6 (20.0)	
Sedation therapy			0.034
Midazolam	7 (23.3)	5 (16.7)	
Dexmedetomidine	7 (23.3)	3 (10.0)	
Propofol	8 (26.7)	6 (20.0)	
Propofol + midazolam	4 (13.3)	15 (50.0)	
Propofol + dexmedetomidine	4 (13.3)	1 (3.3)	

Values are mean ± SD, number of patients (%), or as otherwise indicated.
ICP, intracranial pressure; TCD, transcranial Doppler ultrasonography; GCS, Glasgow Coma Scale; CT, computed tomography; EDH, extradural hematoma; SDH, subdural hematoma; $PaCo_2$, partial pressure of carbon dioxide; MABP, mean arterial blood pressure; HGB, hemoglobin.

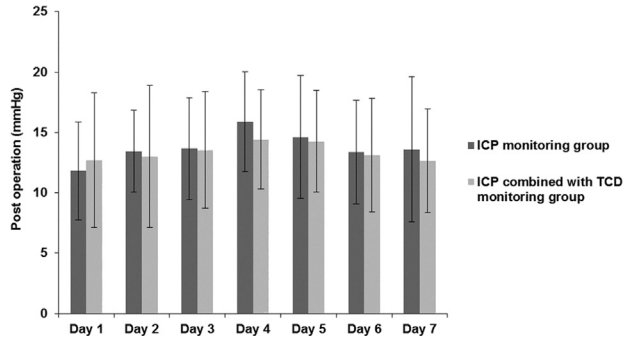

Figure 2. Comparison of postoperative intracranial pressure (ICP) (days 1–7) between the ICP monitoring group and ICP combined with transcranial Doppler ultrasonography monitoring group. All $P > 0.05$. TCD, transcranial Doppler ultrasonography.

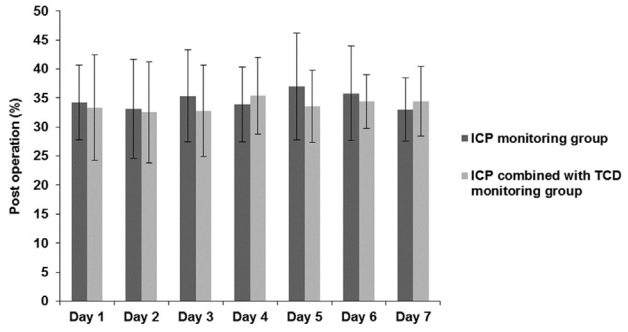

Figure 4. Comparison of postoperative hematocrit (days 1–7) between the intracranial pressure (ICP) monitoring group and ICP combined with transcranial Doppler ultrasonography monitoring group. All $P > 0.05$. TCD, transcranial Doppler ultrasonography.

showed a trend to gradually reduce during the first week after operation. The incidence of cerebral hypoperfusion reached the highest on day 3 (36.7%). The diagnosis of cerebral vasospasm, with a trend of gradual increase, was highest on day 7 (30.0%) (Table 2).

Patient Neurologic Outcomes

Two weeks after operation, there were no differences in mortality between the 2 groups (odds ratio [OR], 1.56; 95% confidence interval [CI], 0.24–10.05; P = 0.643). When considering the GOS-E score at 6 months after DC, there were no differences in prognosis between the 2 groups (OR, 1.25; 95% CI, 0.96–1.64; P = 0.101), but the ICP combined with TCD monitoring group showed a higher frequency of patients with favorable outcome than the routine ICP monitoring group (OR, 0.32; 95% CI, 0.10–0.96; P = 0.043) (Table 3).

Unconditional Multivariable Logistic Regression Analyses of Neurologic Outcomes

Six months after DC, there were statistical differences in osmotherapy, sedation therapy, and ICP on day 4 after operation between the GOS-E score >4 and GOS-E score ≤4 groups (all P < 0.05). The multivariable analysis showed that no factor was associated with neurologic outcomes at 6 months after DC (all P > 0.05) (Table 4).

DISCUSSION

Brain tissue injury and cerebral hemodynamic disturbance with elevated ICP are important contributors to poor outcome in patients with TBI.[15] According to changes in cerebral flow velocity in basal brain arteries, TCD monitoring of patients with TBI postoperation may reveal numerous pathologic hemodynamic changes such as the presence of cerebral hyperemia, vasospasm, and ischemia.[16,17] Therefore, the purpose of our research was to compare the clinical outcomes of routine ICP monitoring with ICP combined with TCD monitoring in patients with TBI after DC operation. The results showed that TCD could be helpful for the serial monitoring of cerebral hemodynamic changes after DC for TBI, which could be beneficial for neurologic outcome

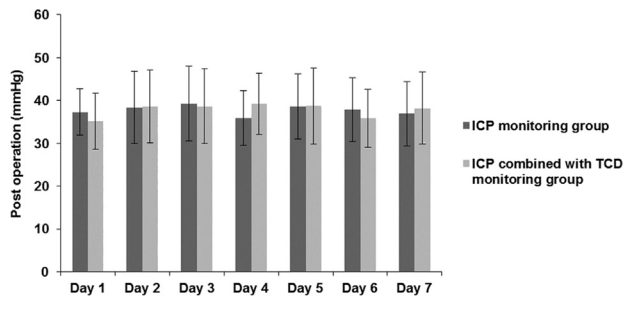

Figure 3. Comparison of postoperative partial pressure of carbon dioxide (days 1–7) between the intracranial pressure (ICP) monitoring group and ICP combined with transcranial Doppler ultrasonography monitoring group. All $P > 0.05$. TCD, transcranial Doppler ultrasonography.

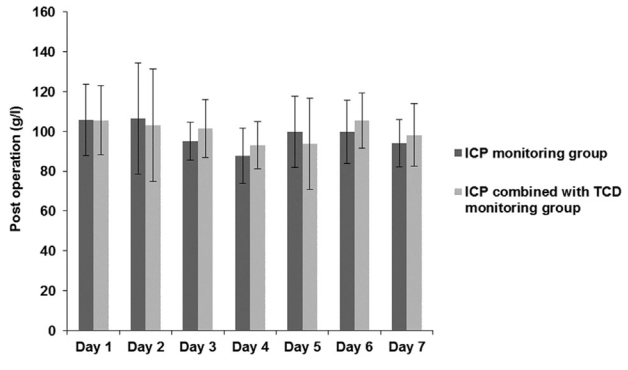

Figure 5. Comparison of postoperative hemoglobin (days 1–7) between the intracranial pressure (ICP) monitoring group and ICP combined with transcranial Doppler ultrasonography monitoring group. All $P > 0.05$. TCD, transcranial Doppler ultrasonography.

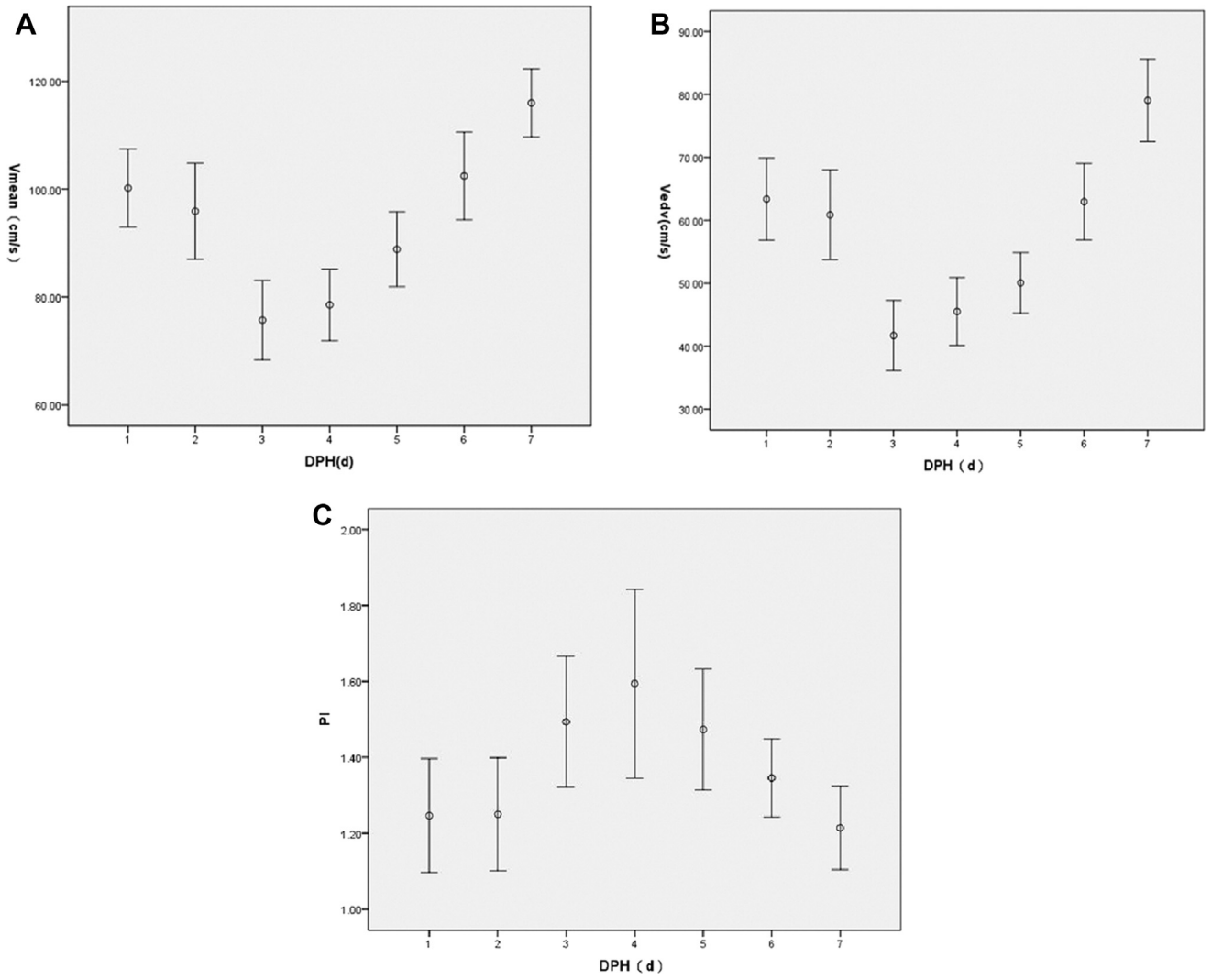

Figure 6. Changes of cerebral hemodynamic parameters (days 1–7) in the intracranial pressure combined with transcranial Doppler ultrasonography monitoring group: (**A**) mean velocity, (**B**) end-diastolic velocity, and (**C**) pulsatility index. PI, pulsatility index; Vedv, end-diastolic velocity; Vmean, mean velocity.

improvement. Unconditional multivariable logistic regression results showed that no other factor, including clinic characteristics, was independently associated with GOS-E score at 6 months.

An adequate and optimized CPP is essential to maintain optimal CBF. Guidelines recommend CPP values of 50–70 mm Hg and ICP <20 mm Hg for the management of TBI.[3] Although an ICP <20 mm Hg is acceptable after DC, it remains compromised in cerebrospinal compensation, peripheral cerebrovascular resistance, and microvascular perfusion. In addition, adequate individual targets are still poorly met because patients have different perfusion thresholds. ICP and TCD measurements can be combined to identify patients at risk for secondary neurologic deterioration post-DC to improve their prognosis.[18] This study similarly suggests that the combination of ICP monitoring and TCD could allow the observation of multiple cerebral parameters and could identify patients with impaired brain hemodynamics despite normal ICP values. Indeed, TCD is based on the assumption that CBFV in a given artery is inversely correlated to the cross-sectional area of that artery. The most reliable indices of TCD (PI, Vedv, and Vmean) are useful to evaluate cerebral hemodynamics and to provide critical information to assess the resistance status of small downstream arteries. The PI strongly correlates with ICP and CPP: an increased PI reflects an increase in ICP and decrease in CPP, and the prognosis of these patients is poor. Therefore, the PI could be used as guiding value in invasive ICP-based decisions in patients in neurologic intensive care.[19] Turek et al.[20] showed that transcranial color-coded sonography monitoring of TBI may reveal various pathologic hemodynamics

Table 2. Abnormal Cerebral Hemodynamic Status (days 1–7) in Intracranial Pressure Combined with Transcranial Doppler Ultrasonography Monitoring Group

Day	Cerebral Hyperemia	Cerebral Hypoperfusion	Cerebral Vasospasm
1	4 (13.3)	5 (16.7)	0 (0)
2	3 (10.0)	8 (26.7)	0 (0)
3	2 (6.7)	11 (36.7)	1 (3.3)
4	1 (3.3)	9 (30.0)	2 (6.7)
5	0 (0)	10 (33.3)	4 (13.3)
6	1 (3.3)	6 (20.0)	8 (26.7)
7	1 (3.3)	8 (26.7)	9 (30.0)

Values are number of patients (%).

changes and found that in nearly 50% of patients significant hemodynamic changes occurred, the most frequent being hyperemia (31.8%), followed by vasospasm (10.9%), and oligemia (9.1%).

The results of this study showed that TCD monitoring after DC could assess the cerebral hemodynamics and treat the abnormal cerebral hemodynamics, which may help reduce the extent of secondary ischemic injuries in patients with TBI and elevated ICP. There were some reasons why targeted therapy for the abnormal cerebral hemodynamics may improve the prognosis of patients with TBI. First, ICP and the PI reached the highest on day 4 (14.41 $\pm$ 4.11 and 1.59 $\pm$ 0.96, respectively), but Vedv and Vmean were lowest on day 3 (41.69 $\pm$ 21.57 and 75.73 $\pm$ 28.53, respectively). At the same time, the incidence of hypoperfusion reached the highest (36.7%) on day 3 too. All these data have indicated that an increasing PI partly reflected the increasing ICP and decreasing cerebral perfusion. Comparing with the change of CBF to the pathophysiology of secondary brain injury, the reaction of ICP may have a slight hysteresis effect. It is known that delayed interference of the abnormal cerebral hemodynamic status may contribute to irreversible damage to more neurons. Second, before we assessed CBF by TCD and treated the abnormal cerebral hemodynamic status, we should correct the serious abnormal laboratory and clinical indications first, which may not have seriously effect on ICP; however, they may be noticeably worse than the CBF. When TCD is performed in the first 24 hours after TBI, it is valid in predicting patient outcomes at 6 months and correlates significantly with ICP and CPP.[15] TCD goal-directed therapy in patients with TBI restored normal CPP about 3 hours before the availability of invasive ICP monitoring and might potentially help in reducing the extent of secondary brain injury.[16] Bor-Seng-Shu et al.[21] observed improvements in clinical examination after DC, correlating with increased flow velocity and reduction of the PI. Our study showed that there was no difference in mortality, but more patients had a favorable prognosis among patients in the ICP combined with TCD monitoring group. However, to date, there is not much experience to guide the management of patients with TBI after DC using TCD monitoring of cerebral hemodynamics.

Our study is not without limitations. First, the sample size was small and from a single center. In addition, the follow-up period was short. Only a limited panel of markers were assessed. Second, the technique is limited by a lack of high-resolution anatomic details. Third, the accuracy of TCD, as any ultrasound technique, is highly operator-dependent. In addition, patient features or vascular features (aberrant vessel course or aneurysm clip artifacts) can impair the detection of pathologic flow values. Fourth, data

Table 3. Patient Neurologic Outcomes

Neurologic Outcomes	ICP Monitoring Group (n = 30)	ICP Combined with TCD Monitoring Group (n = 30)	OR (95% CI)	*P* Value
Death at 2 weeks after operation	2 (6.7)	3 (10.0)	1.56 (0.24–10.05)	0.643
GOS-E score at 6 months after operation, median (IQR)	4.0 (3.0–5.75)	5.0 (3.0–7.0)	1.25 (0.96–1.64)	0.101
GOS-E score >4	12 (42.9)	19 (70.4)	0.32 (0.10–0.96)	0.043
GOS-E score ≤4	16 (57.1)	8 (29.6)		

Values are number of patients (%) or as otherwise indicated.
ICP, intracranial pressure; TCD, transcranial Doppler ultrasonography; OR, odds ratio; CI, confidence interval; GOS-E, Glasgow Outcome Scale-Extended; IQR, interquartile range.

Table 4. Univariable and Multivariable Analyses of Neurologic Outcomes 6 Months After Operation in the 2 Groups

Factor	GOS-E Score >4 (n = 31)	GOS-E Score ≤4 (n = 24)	Univariable Analyses		Multivariable Analyses	
			OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Osmotherapy						
Mannitol	5 (27.8)	13 (72.2)		0.017		0.680
Hypertonic saline	17 (70.8)	7 (29.2)	0.16 (0.04—0.62)	0.008	3.14 (0.10—95.46)	0.512
Mannitol + hypertonic saline	9 (69.2)	4 (30.8)	0.17 (0.04—0.82)	0.027	1.19 (0.08—16.92)	0.900
Sedation therapy						
Midazolam	2 (20.0)	8 (80.0)		0.075		0.607
Dexmedetomidine	4 (40.0)	6 (60.0)	0.38 (0.05—2.77)	0.337	0.31 (0.03—2.97)	0.307
Propofol	9 (69.2)	4 (30.8)	0.11 (0.02—0.78)	0.027	0.06 (0.00—2.26)	0.127
Propofol + midazolam	13 (72.2)	5 (27.8)	0.10 (0.02—0.62)	0.014	0.06 (0.00—1.88)	0.109
Propofol + dexmedetomidine	3 (75.0)	1 (25.0)	0.08 (0.01—1.29)	0.076	0.06 (0.00—3.70)	0.182
ICP on day 4 after operation (mm Hg) (mean ± SD*)	14.08 ± 4.46	16.48 ± 3.71	1.16 (1.00—1.34)	0.047	1.14 (0.97—1.35)	0.122
Different group			0.32 (0.10—0.96)	0.430	0.34 (0.07—1.81)	0.208
ICP monitoring group	12 (42.9)	16 (57.1)				
ICP + TCD monitoring group	19 (70.4)	8 (29.6)				

Values are number of patients (%) or as otherwise indicated. Five patients died after surgery, leaving 55 patients with available data at 6 months.
GOS-E, Glasgow Outcome Scale-Extended; OR, odds ratio; CI, confidence interval; ICP, intracranial pressure; TCD, transcranial Doppler ultrasonography.
*The highest measurement read on day 4.

about the thresholds used to define abnormal TCD are rare, and we mainly had to rely on our own clinical experience. Finally, the design of this study was a retrospective historical control study, and the results need to be validated in randomized controlled trials with a decent sample size.

The use of TCD measurements in the management of patients with TBI remains controversial. TCD allows a better understanding of the ICP and CBF and is practical for serially monitoring the progression of cerebral hemodynamics after DC. It could be a useful bedside tool for TBI management after DC.

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