American Journal of
Emergency & Critical Care Medicine

Research Article

Is Intracranial Pressure Monitoring Necessary in Patients with Mild to Moderate Traumatic Brain Injury with Multiple Trauma? A Case Series

Koichi Hayakawa[1]*, Kazuhisa Yoshiya[2], Osamu Tasaki[1], Hiromu Iwamura[2], Daiki Wada[2], Fukuki Saitou[2], Jiro Iba[3], Hitoshi Ikegawa[2], Tadahiko Shiozaki[3], Yasushi Nakamori[2], Satoshi Fujimi[4], Takeshi Shimazu[3] and Yasuyuki Kuwagata[2]

[1]*Nagasaki University Hospital Emergency Medical Center, Nagasaki, Japan*
[2]*Department of Emergency and Critical Care Medicine, Kansai Medical University General Medical Center, Osaka, Japan*
[3]*Department of Traumatology and Acute Critical Medicine, Osaka University Graduate School of Medicine, Osaka, Japan*
[4]*Department of Emergency Medicine, Osaka General Medical Center, Osaka, Japan*

***Address for Correspondence:** Koichi Hayakawa, Nagasaki University Hospital Emergency Medical Center, 1-7-1 Sakamoto, Nagasaki 52-8501, Japan, Tel: +81-95-819-7765; Fax: +81-95-819-7978; E-mail: 11210329k@gmail.com

Submitted: 22 April 2020; **Approved:** 24 April 2020; **Published:** 08 May 2020

Citation this article: Hayakawa K, Yoshiya K, Tasaki O, Iwamura H, Wada D, et al. Is Intracranial Pressure Monitoring Necessary in Patients with Mild to Moderate Traumatic Brain Injury with Multiple Trauma? A Case Series. American J Emerg Crit Care Med. 2020;3(1): 025-030.

ABSTRACT

Background: Patients with Traumatic Brain Injury (TBI) with multiple trauma may experience unexpected growth of an intracranial hematoma or hemodynamic stroke. To detect such deteriorations, Intracranial Pressure (ICP) monitoring is performed in patients with traumatic intracranial hematoma, even if brain injury is mild to moderate when they require anesthesia or sedation. However, the necessity of ICP monitoring for patients with mild to moderate TBI with multiple trauma has not been clarified.

Purpose: The purpose of this case series was to evaluate the efficacy of ICP monitoring in patients with mild to moderate TBI with multiple trauma.

Methods: During the period from January 2006 through June 2013, 507 patients with TBI were transferred to our trauma centers. The subjects of this case series were patients with TBI with a Glasgow Coma Scale score of ≥ 9 points who required ICP monitoring because of the administration of anesthesia or sedation for the treatment of extracranial injuries. We retrospectively reviewed their clinical course and the necessity for therapy to control ICP.

Results: A total of 14 patients met the inclusion criteria. Eight patients required therapy to decrease ICP because their ICP increased to > 25 mmHg or because their cerebral perfusion pressure decreased. ICP monitoring was found to be useful for these patients. All patients who required therapy to decrease ICP showed high-density spots on computed tomography angiography or had high levels of plasma D-dimer.

Conclusion: ICP monitoring may be useful in patients with mild to moderate TBI with multiple trauma who also have risk factors for neurological deterioration.

Keywords: Intracranial pressure monitoring; Mild to moderate traumatic brain injury; Multiple trauma

ABBREVIATIONS

AIS: Abbreviated Injury Severity; CT: computed Tomography; GCS: Glasgow Coma Scale; ICP: Intracranial Pressure; ISS: Injury Severity Score

INTRODUCTION

Intracranial Pressure (ICP) monitoring is a Level IIb recommendation in the Brain Trauma Foundation's guidelines published in the journal *Neurosurgery* in 2016 [1]. The guidelines recommend that ICP be monitored in all salvageable patients with severe TBI and abnormalities on Computed Tomography (CT). However, the indications for ICP monitoring in patients with mild to moderate TBI have not been sufficiently discussed. Usually, it is possible to detect deteriorations in the level of consciousness of patients if they are not sedated. Patients with multiple trauma may experience unexpected growth of an intracranial hematoma or hemodynamic stroke. To detect such unexpected deteriorations, ICP monitoring is usually performed in patients with traumatic intracranial hematoma, even if their brain injury is mild to moderate, when they require anesthesia or sedation to treat extracranial injuries. The necessity of ICP monitoring for patients with mild to moderate traumatic brain injury with multiple trauma has not been clarified. The purpose of this case series was thus to evaluate the efficacy of ICP monitoring in such patients.

MATERIALS AND METHODS

Patients

All subjects were part of an ongoing prospective outcome database of patients with TBI. During the period from January 2006 through June 2013, 507 patients with TBI were transferred to our two trauma centers. Satisfaction of all of the following criteria was required for inclusion in this case series: [1] intracranial hematoma was present except in patients with subarachnoid hemorrhage only, [2] the patient's Glasgow Coma Scale (GCS) score was > 8 points just before sedation, [3] extracranial injuries with an Abbreviated Injury Severity (AIS) score of ≥ 3 were present, and [4] ICP monitoring was initiated because anesthesia or sedation was necessary for the treatment of extracranial injuries. The patient flow diagram is shown in figure 1.

All patients underwent the same initial standardized treatment protocol, which included appropriate resuscitation and stabilization in accordance with the Advanced Trauma Life Support Guidelines [2]. If an intracranial hematoma was detected, we routinely performed CT angiography to rule out intracranial vascular injury unless severe renal dysfunction was present.

A ventricular catheter was used, if available, for ICP monitoring; otherwise, a parenchymal or subdural monitoring system, the Codman MicroSensor ICP monitoring device (Johnson and Johnson, Raynham, MA), was used with a cranial burr hole. If ICP increased over time, we performed CT. If an intracranial hematoma required evacuation, we performed craniotomy or trepanation to reduce the effect of the mass in accordance with the Guidelines for Surgical Treatment of Traumatic Brain Injury [3]. We performed the following therapeutic interventions to decrease ICP when it increased and stayed > 25 mmHg for about 30 minutes. First, we lowered the patient's body temperature to 37°C if it was above 38°C (normothermia therapy). Cerebrospinal fluid drainage was used for further reduction of ICP. If this drainage was ineffective in controlling the ICP or could not be performed, barbiturate therapy and then mild hypothermia therapy were provided according to a published protocol [4]. Hypothermia therapy was induced by core cooling with the injection of cold saline into the stomach and surface cooling with water-circulating blankets placed above and below the patient. If it was difficult to induce or

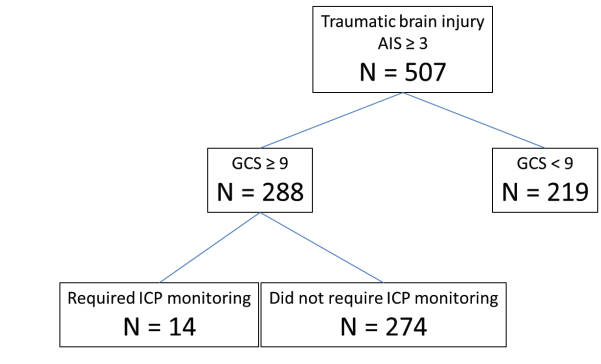

Figure 1: Patient flow diagram. AIS: Abbreviated Injury Severity; GOS: Glasgow Outcome Scale; ICP: Intracranial Pressure.

maintain hypothermia due to shivering, muscle relaxants were administered. The core temperature measured in the bladder was maintained between 33.5°C and 34.5°C. Hypothermia therapy was continued until it was considered unnecessary or deemed ineffective. Although decompressive craniectomy was not performed routinely for refractory intracranial hematoma that was not controllable with mild hypothermia, it was performed if the patient was likely to survive and if the patient's family requested it. We aimed to maintain the cerebral perfusion pressure (the difference between mean arterial pressure and intracranial pressure) at or above 50 mmHg by ICP control and the administration of intravenous fluids and vasopressors to increase blood pressure. Mannitol was not routinely used but was used in patients with findings of impending herniation. Hypertonic saline was not used.

In this case series, we investigated patient characteristics, clinical courses, necessity of intervention to decrease ICP, and complications of ICP monitoring.

Data collection

Age, GCS score, systolic blood pressure, base excess, serum lactate, D-dimer, and the patient's Injury Severity Score (ISS) were evaluated at admission. Outcomes were assessed at discharge according to GOS scores. Leakage of the contrast agent on CT angiography, such as shown in figure 2, was defined as high-density spots. Medical records were also retrospectively investigated to determine whether the patients were taking antiplatelet agents or anticoagulants.

The present case series represents a retrospective analysis of prospectively collected data. This case series was carried out according to the principles of the Declaration of Helsinki and was approved by the institutional review board of Osaka University Hospital (approval number: 17216). The board waived the need for informed consent because only existing clinical data were analyzed.

Statistical analysis

An unpaired *t*-test was used to compare continuous variables. All *p* values were two-sided, and $p < 0.05$ was considered statistically significant. All analyses were performed using SPSS for Windows Ver. 21.0 (SPSS, Inc., Chicago, IL, USA).

RESULTS

Fourteen patients met the inclusion criteria, and the characteristics of these patients are shown in tables 1 and 2. The average ISS value was 39.3, indicating that these patients were severely injured. Eight patients required interventions to decrease their ICP because it had increased to > 25 mmHg, and ICP monitoring was found to be useful for these patients. The contents of the interventions are shown in table 1. Only four patients required normothermia therapy, and four patients required invasive treatment such as decompressive craniectomy. All patients who required interventions to decrease their ICP had high levels of plasma D-dimer, > 70 μg/mL, or showed high-density spots on CT angiography. In addition, our analyzes showed a tendency for patients with epidural hematoma to require more interventions. Table 3 shows a comparison of the patients who did and did not require interventions. There was no significant difference between the two groups in ISS, GCS scores, systolic blood pressure, or base excess values. The plasma D-dimer levels of the patients who required interventions were higher than those of the patients not requiring intervention.

DISCUSSION

ICP monitoring is recommended in the Brain Trauma Foundation's Guidelines for the Management of Severe Traumatic Brain Injury, but indications for ICP monitoring for patients with mild to moderate TBI are not specified. Extracranial injuries have the potential to induce systemic changes, such as coagulopathy, hemorrhagic shock, and elevated levels of reactive oxygen species, all of which have the potential to influence the secondary injury process of TBI [5]. Several clinical studies have reported that the presence of concomitant extracranial injury increases mortality in patients with TBI [6,7]. Concomitant multiple trauma was associated with increased mortality in patients with mild to moderate but not those with severe TBI. If a patient is not sedated or intubated, the clinician can immediately notice any deterioration in their level of consciousness, but in patients with multiple trauma who are sedated and intubated, notice of such deterioration is delayed, often resulting in an irrevocable situation. To detect such unexpected deteriorations, ICP monitoring is usually performed in patients with traumatic intracranial hematoma, even if their brain injury is mild to moderate, when they require anesthesia or sedation to treat injuries elsewhere in the body. The present case series shows that high plasma D-dimer levels or high-density spot(s) on CT angiography may be risk factors for neurological deterioration and good indications for ICP monitoring, especially in patients with mild to moderate TBI who experience multiple trauma.

Several previous studies reported that plasma D-dimer levels are a prognostic indicator of TBI [8-11]. The high-density spot sign on CT angiography is also a predictor of the expansion of intracranial hematoma in patients with endogenous cerebral hemorrhage [12-14]. Contrast extravasation on CT angiography predicts hematoma expansion and mortality in acute traumatic subdural hematoma

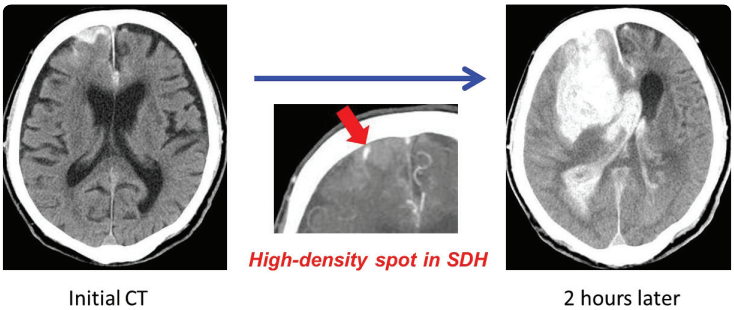

Figure 2: Computed tomography angiography images of a patient presenting with a high-density spot in the subdural hematoma (SDH) area. The red arrow indicates the leakage of the contrast medium.

Table 1: Patient characteristics I.

Case	Age Sex Type of ICH	ISS	Indication for sedation	Interventions required to decrease ICP	Therapeutic intervention(s)	Pupil at the start of intervention	Prognosis (GOS at discharge)
1	56F SDH, SAH	48	Splenic injury	+	NT	Isocoria	MD
2	19M CON	48	Pelvic fracture	+	DC	Anisocoria	MD
3	25M EDH, CON	29	Intestinal injury	+	VD, HT, DC	Isocoria	GR
4	58M EDH, SDH, SAH, CON	34	Bil. Femor fracture	+	NT	Isocoria	MD
5	63M EDH, SAH, CON	50	Lung injury	+	HE, DC	Anisocoria	MD
6	77M SAH, CON	48	Pelvic fracture	+	HT, DC	Isocoria	D
7	35F IVH	57	Aortic injury	+	NT	Isocoria	MD
8	45M SDH, SAH	41	Pelvic fracture	+	NT	Isocoria	SD
9	71F SAH	57	Pelvic fracture	-	-	-	D
10	44F SAH, CON	45	Aortic injury	-	-	-	GR
11	51M SAH	27	Lung injury	-	-	-	MD
12	77M SDH, SAH, CON	22	Hemothorax	-	-	-	SD
13	33F EDH, SDH, CON	27	Lung injury	-	-	-	MD
14	88F CON	25	Lower limb open fracture	-	-	-	SD
Case	Age Sex Type of ICH	ISS	Indication for sedation	Interventions required to decrease ICP	Therapeutic intervention(s)	Pupil at the start of intervention	Prognosis (GOS at discharge)
1	56F SDH, SAH	48	Splenic injury	+	NT	Isocoria	MD
2	19M CON	48	Pelvic fracture	+	DC	Anisocoria	MD
3	25M EDH, CON	29	Intestinal injury	+	VD, HT, DC	Isocoria	GR
4	58M EDH, SDH, SAH, CON	34	Bil. Femor fracture	+	NT	Isocoria	MD
5	63M EDH, SAH, CON	50	Lung injury	+	HE, DC	Anisocoria	MD
6	77M SAH, CON	48	Pelvic fracture	+	HT, DC	Isocoria	D
7	35F IVH	57	Aortic injury	+	NT	Isocoria	MD
8	45M SDH, SAH	41	Pelvic fracture	+	NT	Isocoria	SD
9	71F SAH	57	Pelvic fracture	-	-	-	D
10	44F SAH, CON	45	Aortic injury	-	-	-	GR

11	51M SAH	27	Lung injury	-	-	-	MD
12	77M SDH, SAH, CON	22	Hemothorax	-	-	-	SD
13	33F EDH, SDH, CON	27	Lung injury	-	-	-	MD
14	88F CON	25	Lower limb open fracture	-	-	-	SD

SDH: Subdural Hematoma; EDH: Epidural Hematoma; SAH: Traumatic Subarachnoid Hemorrhage; CON: Contusional Hemorrhage; IVH: Intraventricular Hemorrhage; ISS: Injury Severity Scale; GOS: Glasgow Outcome Scale; NT: Normothermia; HT: Hypothermia; VD: Ventricular Drainage; DC: Decompressive Craniectomy; HE: Hematoma Evacuation; GR: Good Recovery; MD: Moderate Disability; SD: Severe Disability; D: Death

Table 2: Patient characteristics II.

Case	Interventions required to decrease ICP	GCS	sBP	BE	Lac	HDS	D-dimer	Antiplatelet	Anticoagulant
1	+	12	84	-1.6	27	-	70.9	-	-
2	+	14	92	-4.3	37	-	117	-	-
3	+	13	129	-9	48	-	97	-	-
4	+	12	110	-2.6	27	+	21.4	-	-
5	+	12	84	-16	115	-	117	-	-
6	+	9	100	-2.3	11	***	70.4	-	-
7	+	11	120	-18	171	-	107	-	-
8	+	13	166	-6	65	***	77.3	-	-
9	-	15	79	-9	48	-	66.4	+	-
10	-	12	130	***	***	-	6.3	-	-
11	-	12	158	-14	144	-	34.6	-	-
12	-	9	144	3.3	10	-	***	-	-
13	-	9	150	-10	46	-	34.9	-	-
14	-	11	118	***	***	-	62.4	-	-

GCS: Glasgow Coma Scale; sBP: Systolic Blood Pressure on Admission (mmHg); BE: Base Excess (mmol/L); Lac: Lactate (mg/dL); HDS: High-Density Spot; D-dimer: Serume D-dimer Level (μg/mL)
*** not measured or performed

Table 3: Parameters of patients requiring/not requiring interventions to decrease ICP.

	Interventions required to decrease ICP	No interventions required to decrease ICP	*p* value
ISS	44.4±9.1	33.8±13.9	0.145
GCS	12.0±1.5	11.3±2.2	0.547
sBP (mmHg)	110.6±27.6	129.8±28.7	0.235
BE (mmol/L)	-7.4±6.3	-7.4±7.4	1.000
Lac (mg/dL)	62.6±54.2	62.0±57.3	0.986
D-dimer (μg/mL)	86.8±34.0	42.5±27.9	0.049

Unpaired t-test
ISS: Injury Severity Scale; GCS: Glasgow Coma Scale; sBP: Systolic Blood Pressure on Admission; BE: Base Excess; Lac: Lactate; D-Dimer, Serum D-Dimer Level

[15]. The results of the present study agree with the findings of these previous reports.

LIMITATIONS

We acknowledge several limitations of this case series. First, this was a retrospective clinical study performed at just two facilities with a small number of patients. Second, we used no control groups, so we cannot strictly evaluate the efficacy of ICP monitoring in patients with mild to moderate TBI and multiple trauma. Third, it is unknown whether interventions to decrease ICP when it is > 25 mmHg contribute to improvements in prognosis. A randomized controlled study including a greater number of patients at multiple institutions will be needed to address this question.

CONCLUSION

In a selective population of patients with mild to moderate TBI, multiple extracranial injuries susceptible to surgical intervention, radiological findings of potential neurological deterioration (high-

density spot(s) on CT angiography), and high plasma D-dimer levels, ICP monitoring may be useful as an effective monitoring method to guide therapy.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This case series was carried out according to the principles of the Declaration of Helsinki and approved by the institutional review board of Osaka University Medical Hospital (approval number: 17216). The board waived the need for informed consent because only existing clinical data were analyzed.

AVAILABILITY OF DATA AND MATERIALS

The dataset analyzed during the current case series is available from the corresponding author upon reasonable request.

AUTHORS' CONTRIBUTIONS

All authors contributed to the design of this case series. KH, KY, HIwa, DW, FS, HIke and JI collected data. OT, YK, TS, YN, SF, TS and YK supervised the case series. KH and OT analyzed and interpreted the data. KH and OT contributed to writing the manuscript. All authors have read and approved the manuscript.

ACKNOWLEDGEMENTS

We would like to thank Editage (www.editage.com) for English language editing.

REFERENCES

1. Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GWJ, Bell MJ, et al. Guidelines for the management of severe traumatic brain injury. Fourth Edition. Neurosurgery. 2016; 1-10.
2. American College of Surgeons Committee on Trauma. Advanced trauma life support program for doctors: Student course manual. Chicago, IL: American College of Surgeons; 1999.
3. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of traumatic brain injury. Neurosurgery. 2006; 58 Suppl 2: 1-61.
4. Shiozaki T, Hayakata T, Tasaki O, Hosotubo H, Fuijita K, Mouri T, et al. Cerebrospinal fluid concentration of anti-inflammatory mediators in early-phase severe brain damage. Shock. 2005; 23: 406-10. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/15834305
5. McDonald SJ, Sun M, Agoston DV, Shultz SR. The effect of concomitant peripheral injury on traumatic brain injury pathobiology and outcome. J Neuroinflammation. 2016; 13: 90. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/27117191
6. Leitgeb J, Mauritz W, Brazinova A, Majdan M, Wilbacher I. Impact of concomitant injuries on outcomes after traumatic brain injury. Arch Orthop Trauma Surg. 2013; 133: 659-668. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/23463257
7. Siegel JH, Gens DR, Mamantov T, Geisler FH, Goodarzi S, MacKenzie EJ. Effect of associated injuries and blood volume replacement on death, rehabilitation needs, and disability in blunt traumatic brain injury. Crit Care Med. 1991; 19: 1252-1265. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/1914482
8. Lingsma H, Andriessen TM, Haitsema I, Horn J, van der Naalt J, Franschman G, et al. Prognosis in moderate and severe traumatic brain injury: External validation of the IMPACT models and the role of extracranial injuries. J Trauma Acute Care Surg. 2013; 74: 639-646. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/23354263
9. Tong WS, Zheng P, Zeng JS, Guo YJ, Yang WJ, Li GY, et al. Prognosis analysis and risk factors related to progressive intracranial haemorrhage in patients with acute traumatic brain injury. Brain Inj. 2012; 26: 1136-1142. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/22630115
10. Suehiro E, Koizumi H, Fujiyama Y, Yoneda H, Suzuki M. Predictors of deterioration indicating a requirement for surgery in mild to moderate traumatic brain injury. Clin Neurol Neurosurg. 2014; 127: 97-100. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/25459251
11. Foaud HM, Labib JR, Metwally HG, El-Twab KM. Plasma D-dimer as a prognostic marker in ICU admitted Egyptian children with traumatic brain injury. J Clin Diagn Res. 2014; 8: 1-6. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/25386495
12. Brouwers HB, Chang Y, Falcone GJ, Cai X, Ayres AM, Battey TW, et al. Predicting hematoma expansion after primary intracerebral hemorrhage. JAMA Neurol. 2014; 71: 158-164. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/24366060
13. Orito K, Hirohata M, Nakamura Y, Takeshige N, Aoki T, Hattori G, et al. Leakage sign for primary intracerebral hemorrhage: A novel predictor of hematoma growth. Stroke. 2016; 47: 958-963. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/26931155
14. Dowlatshahi D, Brouwers HB, Demchuk AM, Hill MD, Aviv RI, Ufholz LA, et al. Predicting intracerebral hemorrhage growth with the spot sign: The effect of onset-to-scan time. Stroke. 2016; 47: 695-700. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/26846857/
15. Romero JM, Kelly HR, Delgado Almandoz JE, Hernandez-Siman J, Passanese JC, Lev MH, et al. Contrast extravasation on CT angiography predicts hematoma expansion and mortality in acute traumatic subdural hemorrhage. AJNR Am J Neuroradiol. 2013; 34: 1528-1534. **PubMed:** https://www.ncbi.nlm.nih.gov/pubmed/23449655