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The effects of post-intubation hypertension in severe traumatic brain injury

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ABSTRACT

Introduction. The effect of post-intubation hypertension in severe traumatic brain injury (TBI) patients remains uncertain. We aimed to determine the relationship between post-intubation hypertension (mean arterial pressure (MAP) > 110mmHg) and outcomes in severe TBI.

Methods. In this retrospective cohort study, adults who presented with isolated TBI and a MAP ≥ 70mmHg were assessed. Data were retrieved from our institutional trauma registry and the admission list of our neurosurgical intensive care unit (ICU).

Results. We enrolled 126 patients, 81 of whom had a MAP ≤ 110 mmHg after intubation and were assigned to group 1; 45 patients who had a MAP > 110 mmHg were assigned to group 2. Only age (P = 0.008), heart rate (HR; P = 0.036), and MAP before intubation (P < 0.001) were significantly different between groups. We found no significant intergroup differences in mortality (35.8 vs. 35.6%, P = 1.000) or in the motor function of survivors at discharge (P = 0.333). The length of ventilator-dependent (median: 2.0 vs. 5.0 days; P = 0.003) and ICU stays (median: 4.5 vs. 10.0 days; P = 0.005) were significantly longer in group 2. Post-intubation hypertension remained significantly associated with longer ICU stay (≥ 7 days) and poor neurologic outcome (motor < 4 at discharge) after adjusting for other variables (post-intubation MAP >110 mmHg, P < 0.034, OR 3.119, 95% CI 1.087–8.953).

Conclusion. Post-intubation hypertension was associated with longer ventilator-dependent and ICU stays in patients with severe TBI.

Key words: endotracheal intubation, hemodynamics, blood pressure, mean arterial pressure, intracranial hemorrhage.

Introduction

Background

A previous study has reported that induction with pre-treatment before intubation in severe traumatic brain injury (TBI) was underutilization of optimal premedication. Kuzak et al. reported that only 10.2% patients received optimal pre-treatment with an effective dose. (1) Although hypotension, due to pre-treatment medication use during intubation, is of concern, most would argue that intensive management of blood pressure is contraindicated in the first few hours after TBI, to minimize the risk of hypotension and cerebral hypoperfusion. (2-5) The effect of permissive hypertension via withholding the pre-treatment and blood pressure control in severe TBI remains uncertain.

In an emergency, it is difficult and unethical to delay endotracheal intubation until an intracranial pressure monitor has been inserted. Instead of direct measurement of intracranial pressure (ICP), mean arterial pressure (MAP) is more readily available during endotracheal intubation in emergency departments (EDs). Arterial hypertension may worsen brain edema and expansion of the hematoma by excessive intravascular pressures, especially if autoregulation has been altered. (6-8) Previous studies have shown an association between hypertension at hospital admission and increased mortality. (9,10) Lifting the blade of the laryngoscope during endotracheal intubation in patients with severe TBI induces laryngeal stimulation and a sympathetic

response, temporarily increasing heart rate (HR), MAP, and ICP. (11,12) Guidelines recommend maintaining the ICP below 20 mm Hg in patients with severe TBI. (13) Stein et al. reported that the number of brief 5-min episodes of intracranial hypertension and cerebral hypoperfusion is predictive of poor outcome after severe TBI. (4) Nevertheless, there is limited evidence of the effects of post-intubation hypertension on the outcomes in patients with TBIs. We aimed to determine the relationship between post-intubation hypertension and outcomes in severe TBI patients.

Materials and Methods

Study design and setting

We conducted a retrospective cohort study at a tertiary trauma center that has approximately 25,000 visits annually for traumatic injury and a round-the-clock, onsite neurosurgeon. Emergency physicians and trauma surgeons are in charge of these patients during ED boarding. This study was approved by the Hospital Ethics Committee on Human Research and qualified as exempt from the requirement to obtain informed consent.

Patient population

The study cohort consisted of adult patients (>18 years of age) who presented to our ED between January 1, 2007 and December 31, 2009 with isolated TBI and definite intracranial hemorrhage, revealed by brain computed tomography (CT) scans. We excluded patients who had a crushed airway, additional major injuries, prehospital intubation, and a MAP < 70 mmHg in whom pre-treatment with rapid sequence induction (RSI) intubation was contraindicated. Data were retrieved from our institutional trauma registry and the admission list of our neurosurgical intensive care unit (ICU).

Methods and measurements

All patients (except those with crushed airways or cardiac arrest) had their blood pressure and heart rate checked before and immediately after endotracheal intubation in our ED. The nurse in charge of the patient performed the blood pressure and heart rate measurements and recorded the whole process, noting vital signs before and after endotracheal intubation, and the agents used during RSI intubation. Guidelines suggest blood pressure reduction with a target blood pressure of 160/90 mmHg or a MAP of 110 mmHg for hypertensive patients with acute intracerebral hemorrhage. (14,15) A MAP of 120 mmHg might be considered acceptable for those who present with a history of hypertension in such circumstances. (16) A survey of adverse events, using strict criteria with MAP 110 mmHg is feasible, especially in a severe traumatic brain injury scenario when the past history might not be available. Therefore, we assigned patients who had a MAP ≤ 110 mmHg and > 110 mmHg after intubation, to groups 1 and 2, respectively.

Patients underwent emergency surgery, if this was indicated, after consultation with a neurosurgeon. All patients were then admitted to the neurosurgical ICU. A trained study assistant who was blinded to the study purpose performed the chart review and data derivation using a standardized template with clear definitions and codes. During the study, the first authors provided quality improvement feedback after the data analysis by holding periodic meetings with the assistant. Several variables were collected including patient demographics (age, sex, initial Glasgow coma scale score (GCS), diagnosis, and performance of emergent surgery), vital signs before and after intubation, mannitol or labetalol use before intubation, pre-treatment, sedatives, and paralytic agents used for RSI.

Outcome measures

The primary outcome was patient mortality. The secondary outcomes included the Glasgow coma scale motor function score of survivors at discharge (derived from the last note before discharge) and the length of the ventilator-dependent and ICU stay of the survivors.

Statistical tests

The data were analyzed using SPSS 13.0 for Windows (SPSS, Chicago, IL). Demographic characteristics and patient outcomes were compared between groups. Fisher's exact test was applied for categorical data as indicated, and Mann–Whitney U test was used for continuous variables. A P-value < 0.05 was considered statistically significant. Logistic regression was used to identify the independent association of post-intubation hypertension and an adverse outcome after adjusting for other variables. An adverse outcome of survivors was defined as having a GCS motor score of four or less at discharge, or a prolonged ICU stay of seven or more days.

Results

In total, 225 patients were identified from the institutional trauma registry and neurosurgical ICU admission list during the study period. Of the 99 patients who were excluded from the study, 4 had a crushed airway, 74 had additional injuries, 1 had received prehospital intubation, 3 had a MAP < 70 mmHg before intubation, and 17 had incomplete records. Thus, 126 patients were enrolled for analysis. Most of these patients were men (81.0%), and their median age was 52.0 years (interquartile range: 34.0–70.0). Eighty-one patients who had a MAP ≤ 110 mmHg after intubation were assigned to group 1 while 45 patients who had a MAP $>$ 110 mmHg were assigned to group 2 (figure 1). The only significant differences in baseline characteristics detected between groups were mean age in years ($P = 0.008$), HR ($P = 0.036$), and MAP ($P < 0.001$) before intubation (table 1).

The study outcomes are shown in table 2. We found no significant intergroup differences in patient mortality (35.8 vs. 35.6%, $P = 1.000$) or Glasgow coma scale motor score among survivors at discharge ($P = 0.333$). However, significantly longer ventilator-dependent (median: 2.0 vs. 5.0 days; $P = 0.003$) and ICU stays (median: 4.5 vs. 10.0 days; $P = 0.005$) were detected among patients in group 2. Table 3 shows that post intubation hypertension remained significantly associated with an adverse outcome after adjusting for other vari-

Table 1. Baseline characteristics of patients with and without hypertension (MAP > 110 mmHg) after intubation.

	Group 1[a] N = 81	Group 2[b] N = 45	P-value
Patient characteristics			
Male, N (%)	67 (82.7)	35 (77.8)	0.490
Median age in years (IQR)	43.0 (31.3–60.0)	61.0 (48.0–74.0)	0.008
Vital signs before intubation, median (IQR)			
Heart rate, per minute	108.5 (85.3–126.0)	95.0 (83.0–112.5)	0.036
MAP, mmHg	96.7 (90.2–112.7)	119.0 (105.7–141.5)	<0.001
GCS of motor score at ED boarding	4.0 (1.0–5.0)	4.0 (2.5–5.0)	0.333
Diagnosis, N (%)			
EDH	18 (22.2)	5 (11.1)	0.152
SAH	30 (37.0)	15 (33.3)	0.703
SDH	41 (50.6)	27 (60.0)	0.354
ICH	37 (45.7)	27 (60.0)	0.140
Skull fracture	15 (18.5)	5 (11.1)	0.320
Medications before intubation, N (%)			
Mannitol	6 (7.4)	3 (6.7)	1.000
Labetalol	0 (0)	1 (2.2)	0.357
RSI, N (%)			
Lidocaine	31 (38.3)	20 (44.4)	0.571
Fentanyl	2 (2.5)	2 (4.4)	0.616
Midazolam	48 (59.3)	27 (60.0)	1.000
Rocuronium	72 (88.9)	41 (91.1)	0.770
Emergent operation, N (%)	45 (55.6)	21 (46.7)	0.358

ED, emergency department; EDH, epidural hemorrhage; GCS, Glasgow coma scale; ICH, intracranial hemorrhage; IQR, interquartile range; MAP, mean arterial pressure; N, number; RSI, rapid sequence induction; SAH, subarachnoid hemorrhage; SDH, subdural hemorrhage.
[a]Patients had a MAP ≤ 110 mmHg after intubation.
[b]Patients had a MAP >110 mmHg after intubation.
Variables were tested using Fisher's exact test (male, diagnosis, medications before intubation, RSI, emergent operation) or a Mann–Whitney U test (age, vital signs).

ables (MAP >110 mmHg after intubation, P < 0.034, OR 3.119, 95% CI 1.087–8.953).

Discussion

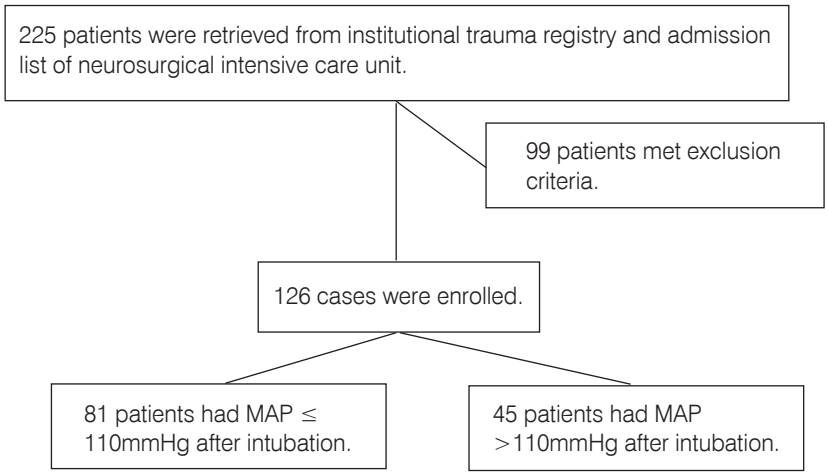
Post-intubation hypertension and outcomes among patients with severe TBI

Post-intubation hypertension developed in 35.7% (45/126) of patients included in our study. We found delayed recovery with prolonged weaning (median: 2.0 vs. 5.0 days; P = 0.003) and longer ICU stay (median: 4.5 vs. 10.0 days; P = 0.005) among patients who developed post-intubation hypertension compared with those who did not. There was a significant association between post-intubation hypertension and adverse outcomes, including a longer ICU stay of more than 7 days and a GCS motor score of less than 4 at discharge. Patients who had post-intubation hypertension were almost three times more likely to develop adverse outcomes (OR 3.119; 95% CI: 1.087-8.953). Zafar et al. reported an OR for mortality of 1.6 (95% CI: 1.32–1.96) among patients with hypertension (systolic blood pressure > 140 mmHg in the ED) when compared with patients who presented with a blood pressure between 120 and 140 mmHg. (10) Walia et al. reported increased mortality with a MAP >100 mmHg during the first 24 h after severe TBI. (9) Our study focused on a sub-group of severe TBI patients

who presented without hypotension. Based on our study results, RSI intubation and post-intubation blood pressure management should be considered in severe TBI patients who present without hypotension. Our study indicates that the development of post-intubation hypertension, in addition to initial coma scale, independently predicts adverse outcomes. In order not to prompt harmful reactions, appropriate management with pre-treatment and pharmacologic interventions, should be considered. A further prospective study to validate our proposal should be conducted.

Possible explanations of our study results

There might be several explanations of our study results. First, an abnormally high MAP not only raises CPP but might also worsen brain edema and expansion of the hematoma, especially if autoregulation has been altered in severe traumatic brain injury. (6-8) Under normal circumstances, autoregulation of the body diverts blood to vital organs such as the brain while responding to stimuli. (17) Instead of increasing perfusion of normal brain tissue, high MAP may have a deleterious systemic effect, such as respiratory distress syndrome. (18,19) Therefore, post-intubation hypertension might provoke secondary brain injury. Previous reports suggest that hypertension should be treated when MAP is above120 mm Hg, but optimal management remains undefined. (16) Second, patients with elevated blood pressure after intubation might experience delayed recovery. Ley et al. found that systolic blood pressure > 160 mmHg at admission was a significant predictor for pneumonia in TBI patients (OR, 1.79; 95% CI, 1.30–2.46; P = 0.0004). (20) They concluded that elevated systolic blood pressure at



MAP, mean arterial pressure.

Figure 1. Patients enrolled in the study. Number of patients included and excluded from the study.
Patients older than 18 years of age who presented to our emergency department with isolated traumatic brain injury and definite intracranial hemorrhage between January 1, 2007 and December 31, 2009 made up the study cohort. Patients who presented with other major injuries, had received pre–hospital intubation, or who had incomplete medical records were excluded (4 had crushed airways, 74 had additional injuries, 1 received pre–hospital intubation, 3 had MAP < 70 mmHg before intubation, and 17 had incomplete records).

Table 2. Outcomes for patients with and without hypertension (MAP > 110 mmHg) after intubation.

	Group 1[a] N = 81	Group 2[b] N = 45	P-value
Vital signs after intubation, median (IQR)			
Heart rate, per minute	102.0 (80.5–122.8)	104.0 (83.0–116.0)	0.972
MAP, mmHg	93.0 (79.8–99.7)	124.0 (115.3–133.5)	–
Median ventilator–dependent stay of survivors in days (IQR)	2.0 (1.0–5.0)	5.0 (2.0–12.0)	0.003
Median ICU stay of survivors in days (IQR)	4.5 (2.0–9.0)	10.0 (5.0–22.0)	0.005
GCS motor score of survivors at discharge, median (IQR)	6.0 (6.0–6.0)	6.0 (4.0–6.0)	0.333
Patient deaths, N (%)	29 (35.8)	16 (35.6)	1.000

GCS, Glasgow coma scale; ICU, intensive care unit; IQR, interquartile range; MAP, mean arterial pressure; N, number.
[a] Patients had a MAP ≤ 110 mmHg after intubation.
[b] Patients had a MAP >110 mmHg after intubation.
Variables were tested using Fisher's exact test (patient deaths) and a Mann–Whitney U test (vital signs after intubation, median length of ventilator–dependent and ICU stays of survivors in days, GCS motor score of survivors at discharge).

admission was associated with worse outcomes in terms of delay. Whether aspiration associated with respiratory distress syndrome, with an irritable or hypertensive state, or poor chest toilet function, due to prolonged comatose state, resulted in longer ventilator dependent and ICU stay is not known. Our results echo the findings of previous studies. Third, arterial hypertension occurred due to sympathetic hyperactivity. Sympathetic hyperactivity is also characterized by various symptoms such as change of consciousness, dystonia, hyperthermia, tachycardia, tachypnea, diaphoresis, and agitation. The model that predicts adverse outcomes of severe TBI might be multifactorial and may primarily depend on the injury itself. (21-25) Intense damage with a higher severity of brain injury may cause higher stimuli and sympathetic activity. In short, post-intubation hypertension may indicate a higher severity of injury with ultimate adverse outcome.

Table 3. Post–intubation hypertension (MAP>110 mmHg) was associated with adverse outcomes* in severe TBI survivors after adjusting for variables by logistic regression analysis.

	Odds ratio (95% CI)	P-value
MAP > 110 mmHg after intubation	3.119 (1.087 – 8.953)	0.034
Age	0.992 (0.967 – 1.018)	0.542
GCS of motor score at ED boarding	0.697 (0.488 – 0.995)	0.047
Diagnosis of ICH	1.599 (0.623 – 4.109)	0.329

ED, emergency department; GCS, Glasgow coma scale; ICH, intracranial hemorrhage; MAP, mean arterial pressure; TBI, traumatic brain injury.
*Adverse outcomes: ICU stay of survivors ≥ 7 days or GCS motor score ≤ 4 at discharge

Anti-hypertensive or pre-treatment medications use in our study population

In our study, only 9 (7.1%) patients received mannitol before intubation, and 40.5% (51/126) and 59.5% (75/126) had pre-treatment and sedation during RSI intubation, respectively. Kuzak et al. reported that 84.2% (48/57) of severe TBI patients received pre-treatment during induction before intubation. (1) Only 6 patients (10.2%) received optimal treatment with an effective dose, and so pre-treatment was evidently underutilization of optimal premedication. Our study results remind clinicians of the importance of blood pressure management in these patients, by demonstrating the harmful effects of post-intubation hypertension. The idea of permissive arterial hypertension and concerns regarding hypotension due to pre-treatment agents in this patient group should be abandoned. A prospective investigation is warranted to determine the relevant algorithms for the management of blood pressure after severe TBI as this affects outcomes.

Limitations

The present study should be interpreted in the context of the following limitations. First, our study was retrospective, and the data were collected from a computer database and by chart review. Although we made every effort to remain objective, possible errors may have been introduced. Second, the retrospective design of our study may raise concerns in terms of selection bias. Nevertheless, the characteristics of the patients enrolled were similar to those reported in previous studies. Third, there might have been confounding factors in our study. To minimize their influence, we strictly enrolled only patients who fulfilled our clearly defined inclusion criteria. All enrolled patients had isolated TBI with definite brain CT findings. In addition, none of the patients had missing blood pressure records prior to or after intubation. Fourth, this study was conducted in a university-affiliated teaching hospital, which may limit the general applicability of our findings. A comparative study using another study design would be of interest. Fifth, the duration of the post-intubation blood pressure measurements was derived from the patient charts, which might not be precise. Thus, we may have underestimated the prevalence of post-intubation hypertension. Instead of measuring hemodynamic changes immediately after intubation, the association of sustained hypertension should be emphasized. Therefore, we attempted to observe the effects of post-intubation hypertension by comparing other outcomes, such as neurologic outcomes and length of ICU stay.

Conclusion

Post-intubation hypertension is associated with delayed recovery and longer ventilator-dependent and ICU stays among patients with severe TBI.

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Yi-Ming Weng, and Chih-Chuan Lin conceived the study. Chih-Chuan Lin supervised the data collection. Chien-Wei Cheng, Ching-I Kuo , undertook recruitment of participating patients and managed the data, including quality control. Yi-Ming Weng, and Chi-Chun Lin, provided statistical advice and analyzed the data; Chih-Chuan Lin chaired the data oversight committee. Yi-Ming Weng and Chien-Wei Cheng drafted the manuscript, and all authors contributed substantially to its revision. Chih-Chuan Lin takes responsibility for the paper as a whole.

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