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Research Article

Comparison of Controlled and Rapid Decompression in Patients with Severe Traumatic Brain Injury

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Abstract

Background: Traumatic brain injury (TBI) is a leading cause of mortality and long-term disability worldwide. Elevated intracranial pressure (ICP) is a major contributor to poor neurological outcomes in patients with severe traumatic brain injury (sTBI). Although decompressive surgery effectively reduces ICP, rapid decompression has been associated with unfavorable neurological outcomes and postoperative complications. Controlled decompression (CD), which involves gradual reduction of ICP under intraoperative monitoring, may provide a safer alternative by minimizing abrupt changes in intracranial pressure.

Objective: To compare the effectiveness of controlled decompression and rapid decompression in improving clinical outcomes and reducing postoperative complications in patients with severe traumatic brain injury.

Study Design: Randomized controlled trial.

Duration and Place of Study: This study was conducted at Pakistan Institute of Medical Sciences, Islamabad, from May 2025 to May 2026.

Methodology: This randomized controlled trial included 60 patients aged 18 to 75 years with severe traumatic brain injury (Glasgow Coma Scale score 3–8) who underwent decompressive surgery. Participants were randomly allocated to either the controlled decompression (CD; n = 30) group or the rapid decompression (RD; n = 30) group. All patients received standardized postoperative care and were followed for six months. Neurological outcomes were assessed using the Glasgow Outcome Scale-Extended (GOSE). The primary outcomes

were six-month functional neurological outcome and 30-day mortality. Secondary outcomes included postoperative complications, duration of mechanical ventilation, and length of neuro-intensive care unit (NICU) stay. Statistical analysis was performed using SPSS version 25.0, with a p-value < 0.05 considered statistically significant.

Results: Of the 95 patients assessed for eligibility, 60 met the inclusion criteria and were randomly assigned to the CD group (n = 30) or RD group (n = 30). At six months, a favourable neurological outcome (GOSE score 5–8) was observed in 16 (53.3%) patients in the CD group compared with 15 (50.0%) in the RD group, while an unfavourable outcome (GOSE score 2–4) occurred in 11 (36.7%) and 8 (26.7%) patients, respectively. A GOSE score of 1 was observed in 3 (10.0%) patients in the CD group and 7 (23.3%) patients in the RD group (p = 0.001). Thirty-day mortality was 16.7% in the CD group compared with 36.7% in the RD group (p = 0.042). Delayed hematoma formation, acute brain swelling, and cerebral infarction were reported in 10.0%, 20.0%, and 16.7% of patients in the CD group, compared with 20.0%, 26.7%, and 23.3% in the RD group, respectively. The CD group also had shorter mean NICU stays (9.1 ± 5.5 vs. 11.7 ± 5.6 days) and shorter mean duration of ventilator support (8.4 ± 4.5 vs. 10.1 ± 3.7 days).

Conclusion: Controlled decompression was associated with favorable neurological outcomes, lower 30-day mortality, fewer postoperative complications, and shorter durations of mechanical ventilation and NICU stay compared with rapid decompression in patients with severe traumatic brain injury. These findings suggest that controlled decompression may be a beneficial surgical approach for patients with severe traumatic brain injury.

Keywords: Severe traumatic brain injury; intracranial pressure; controlled decompression; rapid decompression; decompressive surgery; Glasgow Outcome Scale-Extended (GOSE).

INTRODUCTION

Traumatic brain injury (TBI) is a major global public health concern and remains one of the leading causes of mortality and long-term disability worldwide. It imposes a substantial burden on healthcare systems and significantly affects the physical, cognitive, emotional, and social well-being of patients and their families [1]. Among patients with severe traumatic brain injury (sTBI), persistently elevated intracranial pressure (ICP) is a critical determinant of secondary brain injury and is strongly associated with poor neurological outcomes and increased mortality [2].

Decompressive craniotomy (DC) is an established emergency neurosurgical procedure performed to rapidly reduce elevated ICP when conservative medical management fails [3]. Although numerous studies have demonstrated that DC effectively lowers ICP and improves cerebral perfusion, recent randomized controlled trials have raised concerns regarding its safety and long-term functional outcomes. Despite successful ICP reduction, some patients continue to experience significant neurological disability and poor functional recovery following conventional decompressive craniotomy [4].

Bifrontal temporoparietal decompressive craniotomy has been shown to effectively reduce intracranial pressure and shorten intensive care unit (ICU) stay in adults with diffuse traumatic brain injury and refractory intracranial hypertension [5]. However, Cooper et al. reported that early decompressive craniotomy was associated with significantly poorer functional outcomes at six months, as assessed by the

Extended Glasgow Outcome Scale (GOSE), compared with standard medical management [6]. One proposed explanation is that the abrupt reduction in intracranial pressure during conventional decompression may disrupt cerebral hemodynamics, resulting in cerebral hemorrhage, ischemia-reperfusion injury, impaired venous drainage, and cerebral edema due to the sudden restoration of cerebral blood flow [7,8].

Controlled decompression (CD) has emerged as a potential alternative surgical strategy to conventional decompressive craniotomy. This technique involves the gradual reduction of intracranial pressure under continuous intraoperative ICP monitoring, thereby minimizing abrupt physiological changes within the intracranial compartment [9,10]. Because cerebral autoregulation is often impaired in patients with severe traumatic brain injury, the injured brain is particularly vulnerable to sudden alterations in cerebral perfusion pressure. By allowing controlled pressure release, CD helps maintain cerebral hemodynamic stability and may reduce the risk of postoperative complications, including brain swelling, contralateral hematoma expansion, brainstem shift, ischemia-reperfusion injury, and cerebral infarction [11].

The present study aimed to compare controlled decompression with rapid decompression in patients with severe traumatic brain injury, with particular emphasis on neurological outcomes, postoperative complications, mortality, duration of mechanical ventilation, and length of neuro-intensive care unit stay.

METHODOLOGY

Study Design and Setting

This randomized controlled trial was conducted at the Pakistan Institute of Medical Sciences (PIMS), Islamabad, from May 2025 to May 2026. The study included 60 patients with severe traumatic brain injury (sTBI).

Participants

Patients aged 18 to 75 years with severe traumatic brain injury and a Glasgow Coma Scale (GCS) score of 3–8 at admission were eligible for inclusion. Written informed consent was obtained from the patients' legally authorized representatives before enrollment. The study protocol was approved by the Institutional Ethical Review Committee.

Inclusion and Surgical Criteria

Patients with severe traumatic brain injury who required emergency decompressive surgery were considered for enrollment. Emergency decompressive surgery was performed in patients whose neuroimaging demonstrated a large intracranial hematoma (≥30 mL) and/or severe brain compression, including a midline shift greater than 1 cm, compression or displacement of the lateral ventricles, or obliteration of the basal cisterns.

Intracranial pressure (ICP) was monitored, and emergency surgery was performed in patients with persistent ICP greater than 25 mmHg despite optimal medical management.

Exclusion Criteria

Patients were excluded if they had a preoperative GCS score of 3 accompanied by absent spontaneous respiration and hypotension. Additional exclusion criteria included diffuse brain swelling secondary to combined anoxia and hypotension with minimal intracranial hemorrhage, previous use of aspirin or other anticoagulant medications, known coagulation disorders, multi-organ failure, traumatic brainstem hemorrhage, or intraventricular hemorrhage.

Preoperative Assessment

All eligible patients underwent comprehensive preoperative clinical evaluation, including computed tomography

(CT) and computed tomography angiography (CTA). Baseline clinical and radiological characteristics were recorded before surgery.

Randomization and Intervention

Participants were randomly allocated to either the controlled decompression (CD) group or the rapid decompression (RD) group using the lottery method.

In the RD group, decompressive surgery was performed using the conventional technique with immediate release of intracranial pressure. In contrast, patients in the CD group underwent gradual decompression under continuous intraoperative ICP monitoring before complete decompression and hematoma evacuation.

Postoperative Management and Follow-up

Following surgery, all patients received standardized postoperative management in the Neurosurgical Intensive Care Unit (NICU). Patients were followed for six months, and neurological outcomes were assessed using the Glasgow Outcome Scale-Extended (GOSE).

The primary outcomes were 30-day all-cause mortality and neurological outcome at six months. Secondary outcomes included postoperative complications, including delayed hematoma formation, acute brain swelling, and cerebral infarction, as well as duration of mechanical ventilation and length of NICU stay.

Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 25.0. Continuous variables were expressed as mean ± standard deviation and compared using the independent-samples t-test, while categorical variables were analyzed using the Chi-square test or Fisher's exact test, as appropriate. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 95 patients were assessed for eligibility, of whom 60 patients met the inclusion criteria and were enrolled in the study. The participants were randomly allocated into two equal groups: 30 patients in the controlled decompression group (Group A) and 30 patients in the rapid decompression group (Group B). All participants were aged between 18 and 75 years.

The baseline characteristics of the study population are presented in Table 1. In Group A, 21 (70.0%) patients were male and 9 (30.0%) were female, while in Group B, 17 (56.7%) were male and 13 (43.3%) were female. All patients had a Glasgow Coma Scale (GCS) score of 3–8 at admission. The mean age was 46.1 ± 13.1 years in Group A and 51.2 ± 13.5 years in Group B. The mean intracranial pressure (ICP) was 42.5 ± 12.9 mmHg in Group A and 40.9 ± 12.1 mmHg in Group B. The mean duration from injury to decompression was 11.1 ± 6.1 hours in Group A and 12.7 ± 8.1 hours in Group B.

Regarding the mechanism of injury, road traffic accidents were the most common cause in both groups, accounting for 14 (46.7%) patients in Group A and 12 (40.0%) patients in Group B. Falls accounted for 7 (23.3%) and 8 (26.7%) patients, respectively, while other mechanisms accounted for 9 (30.0%) and 10 (33.3%) patients. The distribution of Rotterdam scores and hematoma types is presented in Table 1.

The primary outcomes are presented in Table 2. At six months, a favourable neurological outcome, defined as a GOSE score of 5–8, was observed in 16 (53.3%) patients in the controlled decompression group compared with 15 (50.0%) patients in the rapid decompression group. An unfavourable outcome, defined as a GOSE score of 2–4, was observed in 11 (36.7%) patients in Group A and 8 (26.7%) patients

in Group B. Three (10.0%) patients in Group A and 7 (23.3%) patients in Group B had a GOSE score of 1. The overall comparison of GOSE outcomes between the two groups was statistically significant (p = 0.001).

The 30-day all-cause mortality was 5 (16.7%) patients in the controlled decompression group compared with 11 (36.7%) patients in the rapid decompression group (p = 0.042).

Secondary postoperative outcomes are presented in Table 3. Delayed hematoma formation occurred in 3 (10.0%) patients in Group A and 6 (20.0%) patients in Group B (p < 0.001). Acute brain swelling occurred in 6 (20.0%) patients in Group A compared with 8 (26.7%) patients in Group B (p = 0.033). Cerebral infarction occurred in 5 (16.7%) patients in Group A and 7 (23.3%) patients in Group B (p = 0.023).

The major postoperative complications and intensive care outcomes are presented in Table 4. Major complications at six months occurred in 14 (46.7%) patients in Group A compared with 21 (70.0%) patients in Group B (p = 0.003). The mean duration of NICU stay was 9.1 ± 5.5 days in Group A and 11.7 ± 5.6 days in Group B (p = 0.002). The mean duration of ventilator support was 8.4 ± 4.5 days in Group A and 10.1 ± 3.7 days in Group B (p = 0.042).

Table 1. Baseline characteristics of the study population

Variables	**Controlled Decompression (Group A), n=30**	**Rapid Decompression (Group B), n=30**
Gender, n (%)		
Male	21 (70.0)	17 (56.7)
Female	9 (30.0)	13 (43.3)
GCS at admission, n (%)		
GCS 3–8	30 (100.0)	30 (100.0)
Intubation, n (%)		
Yes	6 (20.0)	7 (23.3)
Shock, n (%)		
Yes	3 (10.0)	2 (6.7)
Mechanism of injury, n (%)		
Road traffic accident	14 (46.7)	12 (40.0)
Falls	7 (23.3)	8 (26.7)
Other	9 (30.0)	10 (33.3)
Rotterdam score at baseline, n (%)		

Variables	**Controlled Decompression (Group A), n=30**	**Rapid Decompression (Group B), n=30**
1–2	9 (30.0)	10 (33.3)
3–4	7 (23.3)	8 (26.7)
5–6	14 (46.7)	12 (40.0)
Hematoma type, n (%)		
Subdural	12 (40.0)	14 (46.7)
Intracerebral	10 (33.3)	9 (30.0)
Epidural	8 (26.7)	7 (23.3)
Age (years), mean ± SD	46.1 ± 13.1	51.2 ± 13.5
ICP (mmHg), mean ± SD	42.5 ± 12.9	40.9 ± 12.1
Duration from injury to decompression, mean ± SD (hours)	11.1 ± 6.1	12.7 ± 8.1

Table 2. Primary Outcomes

Primary Outcomes	**Controlled Decompression (Group A), n=30**	**Rapid Decompression (Group B), n=30**	**p-value**
GOS-E Score			**0.001**
Favourable (5–8)	16 (53.3%)	15 (50.0%)	
Unfavourable (2–4)	11 (36.7%)	8 (26.7%)	
Dead (1)	3 (10.0%)	7 (23.3%)	
All-cause mortality (30 days)	5 (16.7%)	11 (36.7%)	**0.042**

Table number 3 shows the secondary outcomes in both the groups.

Table No. 3:

Secondary Outcomes	**Group A (n=30)**		**Group B (n=30)**		**p-value**
	n	**%**	**n**	**%**	
Hematoma Delayed	3	10.0	6	20.0	<0.001
Brain swelling in the acute stage	6	20.0	8	26.7	0.033
Cerebral Infarction	5	16.7	7	23.3	0.023

Table number 4 shows the complications after surgery.

Table No. 4:

Complications	**Group A (n=30)**		**Group B (n=30)**		**p-value**
	n	**%**	**n**	**%**	
Major Complications after 6 months of Surgery	14	46.7	21	70.0	0.003
Mean ± SD					
Stay in NICU (Days)	9.1 ± 5.5		11.7 ± 5.6		0.002
Ventilator Support (Days)	8.4 ± 4.5		10.1 ± 3.7		0.042

DISCUSSION

The present study compared controlled decompression (CD) with rapid decompression (RD) in patients with severe traumatic brain injury (sTBI). The findings demonstrated that patients who underwent controlled decompression experienced significantly better postoperative outcomes than those treated with rapid decompression. Controlled decompression was associated with improved neurological recovery, a higher proportion of favorable functional outcomes (GOSE score 5–8), lower 30-day mortality, and fewer postoperative complications, including delayed hematoma formation, acute brain swelling, and cerebral infarction. Furthermore, patients in the CD group required shorter durations of mechanical ventilation and neuro-intensive care unit (NICU) stay, indicating faster postoperative recovery and more efficient utilization of critical care resources.

Our findings are consistent with those reported by Bao et al., who demonstrated that controlled decompression significantly improved neurological outcomes and reduced mortality in patients with severe traumatic brain injury [12]. Their study also showed superior long-term functional recovery in patients treated with controlled decompression compared with conventional rapid decompression. Similarly, previous randomized controlled trials have reported that although decompressive craniotomy effectively reduces intracranial pressure, rapid decompression may be associated with less favorable neurological recovery despite successful ICP reduction [13–15].

In the present study, the incidence of delayed hematoma formation was significantly lower in the controlled decompression group than in the rapid decompression group (10.0% vs. 20.0%). This finding supports previous reports suggesting that gradual reduction of intracranial pressure minimizes abrupt intracranial pressure fluctuations, thereby reducing the risk of contralateral hematoma formation. Delayed postoperative hematoma is a recognized complication following decompressive craniotomy and frequently necessitates additional surgical intervention [16,17]. Huang et al. also reported contralateral epidural hematoma following decompression surgery for traumatic brain injury and highlighted the role of rapid intracranial pressure reduction in its pathogenesis [18].

Acute postoperative brain swelling was also significantly less frequent in patients who underwent controlled decompression (20.0%) than in those who underwent rapid decompression (26.7%). This observation is consistent with the hypothesis proposed by Langfitt et al., who suggested that rapid decompression causes abrupt cerebral hyperemia and vasodilation following the sudden reduction in intracranial pressure, thereby increasing the risk of intraoperative and postoperative brain swelling [19]. Furthermore, the incidence of cerebral infarction was markedly lower in the controlled decompression group (16.7%) compared with the rapid decompression group (23.3%). This finding may be explained by the gradual restoration of cerebral perfusion achieved with controlled decompression, which helps maintain cerebral hemodynamic stability and reduces ischemia-reperfusion injury by preserving the balance between arterial inflow and venous outflow [20].

Patients treated with controlled decompression also had significantly shorter NICU stays and required fewer days of mechanical ventilation than those who underwent rapid decompression. These findings suggest that controlled decompression facilitates faster postoperative recovery, improves clinical

stability, and may reduce healthcare resource utilization, ultimately contributing to better overall patient outcomes.

Despite these encouraging findings, the present study has several limitations. First, the sample size was relatively small, which may limit the statistical power of the study. Second, this was a single-center study, which may limit the generalizability of the findings to other institutions and patient populations. Finally, the follow-up period was limited to six months, and longer follow-up may provide additional information regarding long-term functional recovery. Therefore, large multicenter randomized controlled trials with extended follow-up are warranted to validate these findings and further establish the role of controlled decompression in the management of severe traumatic brain injury.

CONCLUSION

Controlled decompression demonstrated superior clinical outcomes compared with rapid decompression in patients with severe traumatic brain injury. Patients treated with controlled decompression experienced lower 30-day mortality, better neurological recovery, fewer postoperative complications, shorter durations of mechanical ventilation, and reduced neuro-intensive care unit stay. These findings suggest that controlled decompression is a safer and more effective surgical approach for the management of severe traumatic brain injury.

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Conflict of Interest

The authors declare that they have no conflicts of interest related to this study.

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