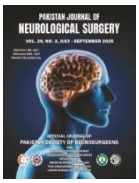

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Original Research

Controlled Decompression Effects in Patients with Severe Traumatic Brain Injury: A Randomized Controlled Trial

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ABSTRACT

Objective: To investigate whether controlled decompression therapy reduces the incidence of comorbidities and enhances recovery in sTBI patients.

Materials and Methods: In a Khyber teaching hospital, sTBI patients aged 18 to 75 years were randomly divided into one of two groups: controlled decompression (CD) surgery (n = 26) and fast decompression surgery (n = 26) in a randomized control experiment. The primary outcome markers were 30-day all-cause mortality and the Extended-Glasgow Outcome-Scale (GOS-E) score at six months. Secondary outcomes included delayed bleeding, posttraumatic brain infarction, and intraoperative brain infarction.

Results: The greatest improvement in six-month GOS-E score was a remarkable reduction in 30-day all-cause mortality in the controlled decompression (CD) group in comparison to the group fast decompression group of the participants (15.3% compared with 24.6%, a P value of -0.042). Additionally, subjects in the group of supervised decompression surgery had lower intraoperative brain edema (19.2% versus 42.3%, p-value 0.033), late bleeding (11.5% versus 30.7%, p-value 0.048), and cerebral infarction after trauma than the rapid decompression group (15.0% versus 22.4%, with a p-value less than 0.001).

Conclusion: Controlled decompression (CD) surgical interventions have been found to significantly uplift the outcomes for individuals with severe traumatic brain injury (sTBI) and reduce the likelihood of related health conditions so but a more comprehensive understanding of the importance of standardized directed decompression surgical intervention in the management of sTBI requires multicenter randomized controlled trials.

Keywords: Brain Edema, Controlled Decompression, Decompressive Craniectomy, Intracranial Pressure, Neurotrauma, Traumatic Brain Injury.

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INTRODUCTION

A major public health issue is traumatic brain injury (TBI), which has a high incidence and death rate as well as a substantial impact on the lives of victims and their families.[1,2] Poor outcomes in individuals with severe TBI (sTBI) may be caused by uncontrollably high intracranial pressure (ICP).[3] For patients with sTBI, decompressive craniotomy (DC) is a quick way to lower ICP.[4] Although some studies have found that DC (rapidly releasing the ICP) is the standard surgical procedure that improves prognosis,[5] recent RCTs have found that there might be a link between DC to mortality, postoperative complications, and high rates of disability.[6]

While bifrontal temporoparietal Decompressive craniotomy (DC) has proven effective in reducing intracranial pressure (ICP) and shortening ICU stays for fatal diffuse traumatic brain injury (TBI) and unyielding ICP in adults, Cooper et al, found that its early application is linked to significantly poorer outcomes at the 6-month mark, as measured by the GOSE score.[7] Individuals with severe traumatic brain injury (sTBI) may experience immediate complications during surgery if the intracranial pressure (ICP) is significantly reduced too quickly. Additionally, a rapid decrease in ICP can lead to cerebral hemorrhage and subsequent damage due to ischemia-reperfusion. This may occur due to the quick restoration of arterial blood flow while simultaneously restricting venous outflow.[8]

In contrast to standard craniotomy, which releases the ICP quickly, CD in DC allows the ICP to be released gradually. All of the phases are decided by the ICP during the surgical operation. When the ICP rises to a level where a craniotomy is necessary following sTBI, the cerebral arteries will lose their ability to regulate blood flow of the cerebrovascular pathway, and the brain veins will constrict.[9,10] To avoid a rapid collapse of the skull and dura, which might result in an improper venous outflow and a rush of arterial blood into the brain tissue, it is imperative that intracranial pressure (ICP) be properly managed during surgery. To maintain the delicate balance between cerebral blood intake and outflow, CD is essential. Sudden withdrawal of ICP can also lead to the displacement of the brain stem, causing rapid expansion of the hematoma on the opposite side as the pressure decreases. CD not only helps to limit potential ischemia-reperfusion damage but also addresses immediate intraoperative cranial encephalocele and prevents post-surgical brain infarction. The primary goal is to maximize the preservation of brain circulatory and nervous system function by ensuring a carefully managed adjustment of intracranial pressure.[8]

For over a decade, surgeons have been working on improving the management of severe traumatic brain injuries (sTBIs). They have explored treatment options such as supervised ventricular draining and careful pooled blood evacuation, believing that these methods could improve outcomes for sTBI patients. However, there is a significant gap in the form of a lack of well-conducted randomized trials that directly compare the benefits of these different surgical interventions.

MATERIAL AND METHODS

Selection of Sample

Eligible patients were between 18 and 75 years old, with a Glasgow Coma Scale (GCS) score of three to eight upon hospitalization, and provided informed consent. If there was a large-volume hematoma (30 mL) or observable brain tissue compression (e.g., diversion from the midline >1 cm, pressure, and deviation of the lateral ventricles and basal cistern), Decompressive craniotomy (DC) was carried out. A preoperative GCS score of 3, presentation without reduced blood pressure and respiration, a combination of anoxia and hypotension resulting in brain swelling with minimum intracranial blood loss following trauma, coagulation diseases, a history of using aspirin, and multi-organ dysfunction were among the

exclusion criteria. Furthermore, specific damage sites like hematomas in the ventricle or brain stem were also reasons for exclusion.

This randomized controlled trial was prospectively registered in the Iranian Registry of Clinical Trials (IRCT), which is a WHO-recognized primary clinical trial registry, under the registration number IRCT20230907059376N1. The registration was completed before the enrollment of participants.

Random Allocation of the Patients

The randomization of the participants was done through a lottery method to either controlled or fast decompression following sTBI before surgery.

Sample Size

The mortality rate of patients with TBI after decompressive craniotomy was 27%.[11] With this assumption, it was estimated that a total of 52 patients were needed with the Fleiss and CC method, keeping the power at 50 and alpha 5.

Evaluation of Symptoms

To evaluate alterations in brain vascular flow and blood vasculature and provide guidance for treatment, all patients were screened with a basic CT and CT angiography (CTA) before surgery. The disappearance of the cisterna ambiens, potential midline shift, compression of ventricles, or other abnormalities were examined when the Glasgow Coma Scale (GCS) score decreased by more than two, prompting a comprehensive review of the CT findings. If intracranial pressure (ICP), measured using Codman equipment from the USA, persisted and exceeded 25 mmHg after mannitol dehydration treatment, urgent craniotomy was recommended. The craniotomy and placement of the ICP monitor were carried out concurrently when a patient had a cerebral hernia and a sizable hematoma, with the ICP monitor going in just before the bone flap and dura were opened.

Together, the surgeons decided if DC was

required. The patients were allocated at random to have controlled or fast decompression surgery after both neurosurgeons approved the procedure, and the patients' families gave their approval.

Surgical Procedure

Standard surgical techniques were used to achieve a rapid craniotomy.[12] The dura was entirely opened by a normal large craniotomy (twelve to fifteen cm), allowing the ICP to be quickly, totally, and uncontrollably discharged. ICP monitoring was given to each patient in this group. During the procedure, the rate of ICP decline was uncontrolled, and the surgical plan did not consider the ICP. The timely removal of the hematoma and brain contusion tissue was the main objective of the procedure.

The goal of CD was to guarantee that the ICP would gradually release over the whole treatment using a variety of techniques. I.C.P dropped at a rate of 10-15 mmHg per ten minutes.[(8)] Before the craniotomy, an I.C.P explorer was implanted to get the initial I.C.P. The best option was a brain tissue monitor, followed by a ventricular intracranial pressure monitor.

The ejection of cerebrospinal fluid (CSF) is started to bring the intracranial pressure (ICP) below 40 mmHg if the ICP is discovered to be higher than a predetermined threshold. After that, a craniotomy surgery is carried out on a 12 by 15 cm bone window to pressurise the brain and avoid a sharp drop in intracranial pressure when the bone is removed. The dura is broken via a little incision, usually no more than 5 mm in diameter. The ICP is progressively lowered while the hematoma and brain contusion tissue are correctly aspirated. When there are no visible indications of enlarged brain tissue and the intracranial pressure drops to less than 10 mmHg, the meningeal membrane is widened, and the brain contusion or hematoma is extracted.

Monitoring of patients after Surgery

After the surgical procedure, the individuals were promptly moved to the neurosurgical intensive care unit (NICU) for further observation and treatment. The post-operative management plan was developed collaboratively by a neurosurgeon and a NICU physician. Patients in both groups received the same post-operative care. Patients with elevated ICP underwent therapeutic hypothermia for 7–10 days. ICP was regularly checked with an ICP sensor, which was normally taken out around a week following surgery. Every two hours, the ICP and vital signs were monitored and noted. If the patients were stable at 1, 24, and 72 hours following the procedure, the cranial CT data were routinely evaluated.

Follow-up & Outcome Measures

The GOSE score was used to measure neurological outcomes after the surgery.[13] The evaluations were conducted by blinded investigators six months after the injury through one-on-one interviews. The thirty-day all-suspected death rate and the post-neurosurgical prognosis were the main outcomes that were assessed. Three groups were identified using the GOSE score: favorable (scoring 5-8), unfavorable (score 2-4), and deceased (score 1). The GOSE score was used to quantify function and a better lifestyle.

While analyzing the surgical complications, the incidence of cerebral infarction, immediate operative brain oedema, and delayed hematoma was compared between the two groups. Delayed hematoma was defined as the discovery of an intracranial hematoma on a subsequent scan that was initially missed. Acute operational brain swelling was identified by a sudden increase in intracranial pressure (ICP) during surgery, causing brain tissue protrusion from the bone defect site, which was confirmed by postoperative CT scans showing brain swelling and elevated ICP.

Ischemic brain lesions that were detected post-surgery on CT scans were classified as cerebral

infarctions during hospitalization. Cerebral infarctions appeared as abnormally hypodense patches with distinct borders in areas with arterial blood flow, while uneven density indicated brain contusion in damaged areas. CTA or perfusion CT confirmed the outcome of cerebral contusion when necessary. Transcranial Doppler (TCD) was used to assess daily cerebral blood flow.

Analysis

Patients were analyzed using the principle of intention to treat. Using the t-test, we compared baseline characteristics. The Pearson test was used to analyze data. The value of significance was $P<0.05$, using SPSS 25 for statistical analysis.

RESULTS

A total of 80 patients were assessed, among whom 52 were randomly selected for the surgical treatment of severe TBI, from February 2022 to January 2023, among which 26 patients were assigned to the Controlled decompression group and 26 fast decompression group. The basic properties are mentioned in Table 1.

The 30-day all-cause mortality in this trial differed significantly between the quick decompression and control groups. According to the findings, controlled decompression surgery lowered the 30-day mortality risk by 9%. Additionally, six months following surgery, there was a prominent scientific difference in the GOSE category between the two groups. The decompression control group surpassed the GOSE group in terms of outcomes (Table 2).

The occurrence of brain swelling during surgery and sudden brain herniation was much less frequent in the monitored decompression group compared to the fast-track decompression group. Delayed hematoma, a post-operative complication caused by brain contusions or countercoups, can significantly worsen the condition of acute encephalocele and pose a serious risk.

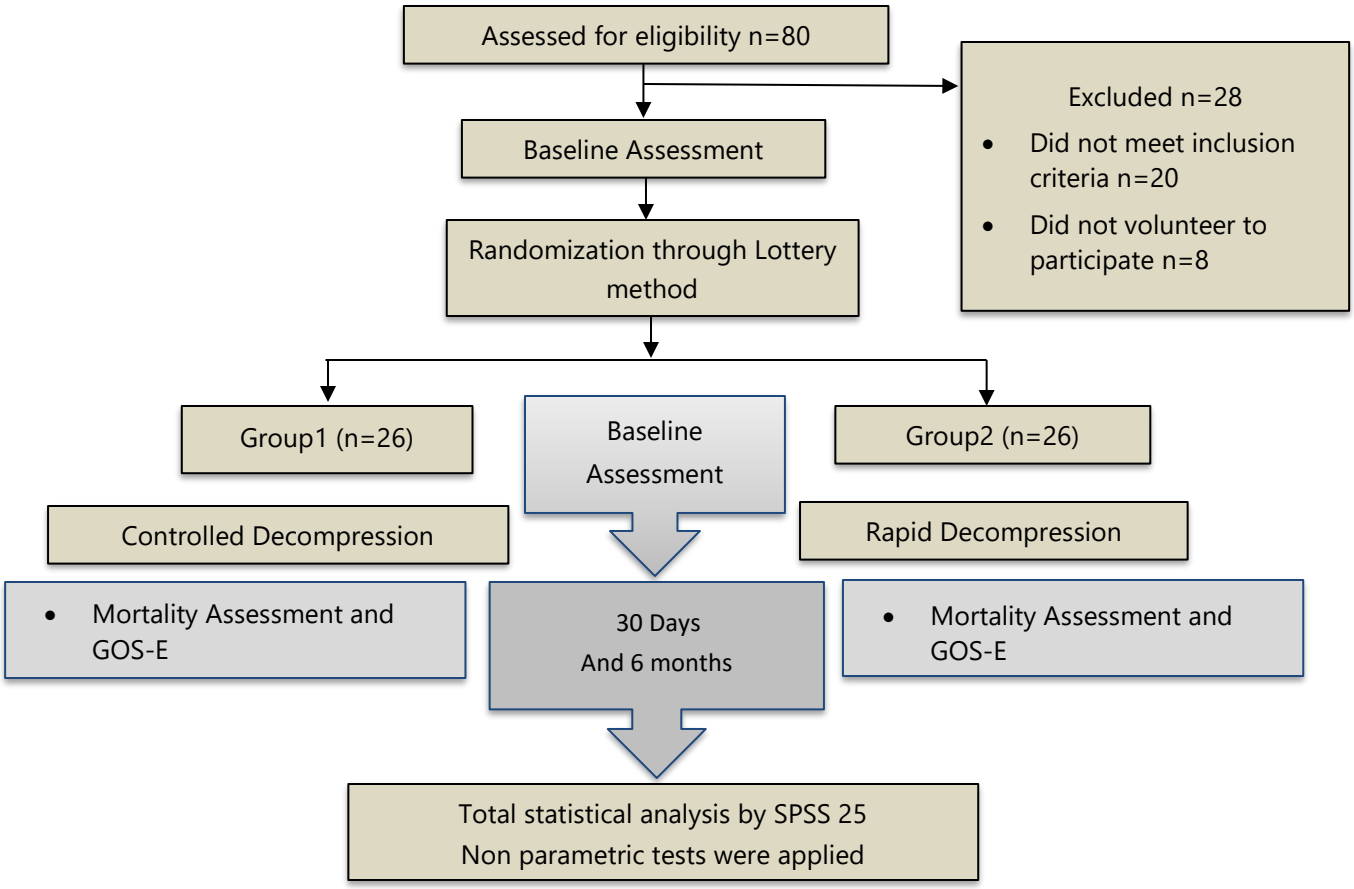

Figure 1: Consort Diagram

Table 1: Basic Properties

Variables	Controlled Decompression (n=26)	Conventional Decompression (n=26)
Age (years)	45.5 ± 12.98	51.23± 13.7
Duration from injury to Decompression	11.1 ± 6.0	12.7 ± 8.2
ICP	42.5 ± 12.9	41.2 ± 11.9
GCS score at time of admission		
3-5	8 (30.7%)	7 (26.9%)
6-8	10 (38.4%)	11 (42.3%)
Intubated	5 (19.2%)	6 (23.0%)
Shock	3 (11.5%)	2 (7.6%)
Gender		
Male	18 (69.2%)	15 (57.6%)
Female	8 (30.7%)	11 (42.3%)
Rotterdam Score at baseline		
1-2	8 (30.7%)	9 (34.6%)
3-4	6 (23.0%)	7 (26.9%)
5-6	12 (46.1%)	10 (38.4%)
Mechanism of Injury		
RTA	12 (46.1%)	10 (38.4%)
Falls	6 (23.0%)	7 (26.9%)
Other	8 (30.7%)	9 (34.6%)

Hematoma Type		
Intracerebral	9 (34.6%)	8 (30.7%)
Epidural	7 (26.9%)	6 (23.0%)
Subdural	10 (38.4%)	12 (46.1%)

Table 2: Between-Group Analysis of Primary Outcomes

	Controlled Decompression n=26	Conventional Decompression N=26	P value
GOS-E			
5-8 score (Favorable)	11 (42.3%)	8(30.7%)	0.001
2- 4 score (Unfavorable)	8(30.7%)	3(11.5%)	
1 score (Dead)	3(11.5%)	6 (23.0%)	
All-cause mortality (30 Days)	4 (15.3%)	9 (24.6%)	0.042

Pearson Test of Chi-Square

Table 3: Secondary Outcomes for Between Groups.

	Controlled Decompression n=26	Conventional Decompression n=26	P value
Hematoma Delayed	3 (11.5%)	8 (30.7%)	< 0.001
Cerebral Infarction	4 (15.3%)	14 (53.8%)	0.023
Brain swelling in the acute stage	5 (19.2%)	11 (42.3%)	0.033

Pearson Test of Chi-Square

Table 4: Complications After Surgery

	Controlled Decompression	Conventional Decompression	P value
Stay in NICU (Days)	9.2 ± 5.4	11.5 ± 5.7	0.002
Ventilator Support (Days)	8.2 ± 4.4	10.2 ± 3.6	0.042
Major Complications after 6 months of Surgery	12 (46.1%)	18 (69.2%)	0.003

Post-traumatic cerebral infarction (PTCI), which occurs after TBI, is among the most severe secondary injuries. The frequency of delayed hematoma and PTCI varied significantly between the groups. NICU delay and oxygenation were also reduced in the controlled group compared to the fast decompression group (Tables 3 & 4).

DISCUSSION

Our research indicates that performing strategic decompression surgery can lead to improved outcomes for patients. This surgery significantly reduces the occurrence of immediate brain edema and delayed hematoma. We have noticed that patients who underwent controlled decompression had better results and reduced mortality rates. Six months post-injury, the supervised decompression group exhibited superior recovery compared to the quick decompression group. Additionally, the controlled decompression group had a below-thirty-day mortality rate than the quick decompression group. Bao et al, observed that among cases with severe traumatic brain injury (sTBI) and malignant diffuse brain swelling, 21.6% achieved satisfactory

recovery, while 18.9% succumbed to the condition.[6] In a recent study, out of the 201 cases in the surgical intervention group who had refractory elevated intracranial pressure (ICP > 25 mmHg), only 10% succumbed to their condition. In contrast, the medical management group had 188 patients facing similar challenges, out of which 48.9% experienced fatal outcomes. It is important to note that neither group had a full recovery.[14] Therefore, in our quick decompression group, both the death rate and the percentage of patients who demonstrated satisfactory recovery fell within the limits as per evidence. In our study, controlled decompression produced superior outcomes. The primary causes can be connected to issues following surgery.

During our investigation, we found that the fast decompression group had a 30.7% rate of delayed hematoma occurrence, while the controlled decompression group had a significantly lower incidence of 11.5%. This indicates that implementing surgical technology and controlled decompression is effective in substantially reducing the risk of delayed hematoma. As we have previously reported, our research showed that over 20% of patients with fatal traumatic brain injury (sTBI) experienced delayed bleeding after surgery, and a significant number of these patients may require a secondary intervention for hematoma removal.[8] The majority of researchers see post-operative hemorrhage, which often happens at the site of the surgery but can sometimes happen distantly, as a well-known, uncommon, but significant consequence of intracranial operations.[15,16] Contralateral epidural/ subdural hematoma is another possibility.

According to Huang et al, distant epidural hemorrhage (EDH) occurred in 7.9% of patients who had decompressive hemicraniectomy for TBI. Prior studies of these sequelae reported a lower prevalence of delayed contralateral hematoma than we did because they were mostly case reports, lacked comprehensive feedback analyses, and included all TBI cases, not only those with sTBI.[15] It is not clear what causes contralateral epidural hematoma following DC surgery. Additionally, there is concern that contralateral calvarial fractures may lead to such hematomas.[17,18] After decompression surgery, the blockage that causes bleeding on one side of the brain is immediately relieved, leading to the formation of a blood clot on the opposite side. Our observations show that decompression performed too quickly or leading to an immediate decrease in intra-cranial pressure (ICP) increases the risk of delayed formation of a blood clot on the opposite side. However, as ICP gradually decreases, the risk of delayed clot formation also decreases significantly. In addition, both the monitored and rapid decompression groups had acute cerebral edema. The rate of cerebral edema in the first group was 23% lower than in the rapid decompression group. Late thickening of blood clot on the opposite side of the brain is the most common cause of brain edema following craniotomy, although the exact cause is still unknown. Langfitt et al, suggested that this phenomenon could be related to mechanical or biological changes that occur during surgery.[19] Furthermore, Langfitt et al, suggested that the process may be connected to either the quick decrease of ICP or the increased cerebral blood volume and dilatation of the cerebrovascular after craniotomy. Therefore, controlled decompression was more successful in lowering brainstem displacement, delayed hematoma formation, and acute intraoperative encephalitis incidence.[20]

CONCLUSION

Based on our research, we discovered that individuals with sTBI who had controlled decompression had noteworthy results after six months. Those who had controlled decompression had lower rates of cerebral infarction, delayed intracranial hemorrhage, and intraoperative acute brain swelling than those who had fast decompression. Additionally, patients who had

controlled decompression had a lower 30-day all-cause death rate. According to our early research, controlled decompression may be a useful strategy for lowering both postoperative cerebral hemorrhage and immediate cerebral edema.

RECOMMENDATIONS

We contend that the use of controlled decompression may lessen ischemia-reperfusion injury and progressively restore cerebral blood flow and volume. Further multicenter trials with bigger participant groups are necessary to validate, along with the expansion of our results.

The relatively small number of cases in each group, which lowers the study's precision of the analysis, is the source of our study's shortcomings. It's also critical to note that this study was restricted to one location. In light of the encouraging findings of our study, we suggest that more multicenter trials that are comprehensive, well-structured, and comparable are necessary.

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Additional Information

Disclosures: Authors report no conflict of interest.

Ethical Review Board Approval: The study conformed to the ethical requirements.

Human Subjects: Consent was obtained by all patients/participants in this study.

Conflicts of Interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following:

Financial Relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

Other Relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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AUTHORS CONTRIBUTIONS

Sr.#	Author's Full Name	Intellectual Contribution to Paper in Terms of:
1.	Mahboob Khan	1. Study design and methodology.
2.	Abdul Hameed Khan, Laila Ghaffar	2. Paper writing.
3.	Mehboob Khan, Nayyab Orakzai	3. Data Collection and calculations.
4.	Farooq Shehzada, Abdul Basit Khan	4. Analysis of data and interpretation of results.
5.	Mahboob Khan	5. Literature Review and referencing.
6.	Farooq Shehzada	6. Editing and quality insurer.