

Decompressive Craniectomy in Diffuse Traumatic Brain Injury: An Industrial Hospital Study

Abstract

Context: High intracranial pressure is the most frequent cause of mortality and disability after severe traumatic brain injury (TBI) which is treated by first-line therapeutic measures. When these measures fail, second-line therapies are started. Among second-line therapies, decompressive craniectomy (DC) has been used. It improves the functional outcome in these patients. **Aim:** This study aims to analyze the clinicoradiological factors associated with the prognosis of severe TBI in patients undergoing DC. **Settings and Design:** It was a retrospective case series study from April 2014 to March 2016. **Subjects and Methods:** A total of 85 patients (admitted at Tata Main Hospital, Jamshedpur) with severe diffuse TBI with clinical and radiological evidence of intracranial hypertension who were refractory to first-tier therapies and required DC were included in our study. Cases excluded were patients with age <10 years and polytrauma patients. **Results:** Out of 85 cases, 55 were males, and thirty were females (male:female = 1.8:1) with the age ranging from 17 to 68 years. Road traffic accident was the leading cause of injury in 69.5% cases. A total of 49 (58%) patients were of Glasgow coma scale (GCS) 4–6 whereas 36 (42%) patients had GCS 7–8. Computed tomography (CT) scan brain was classified as per Marshall CT classification. Bifrontotemporal DC was done in 29% cases, and unilateral frontotemporoparietal craniectomy was done in 71%. **Conclusions:** Patients with younger age, early surgical intervention, better preoperative GCS score, and with low Marshall CT score have better prognosis.

Keywords: Decompressive craniectomy, early surgery, head trauma, Marshall computed tomography score

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Introduction

Severe traumatic brain injury (TBI) stands as one of the common causes of death in <45 years age group with a current global mortality of 39%,^[1,2] and the next most frequent cause of death and mortality after this is high intracranial pressure (ICP). Initially, general measures such as normothermia and sedation are used followed by first-line therapeutic measures such as moderate hypocapnia and mannitol. Second-line therapies are started when these measures fail to control high ICP. These includes use of barbiturates, hyperventilation, moderate hypothermia, or removal of a variable amount of skull bone (decompressive craniectomy [DC]).^[3] Hence, management of malignant posttraumatic cerebral edema remains a challenge for the neurosurgeon. In spite of all measures to control elevated ICP, mortality and morbidity remains high. DC is widely used to treat intracranial

hypertension following TBI.^[4] The causative factors of DC in the treatment of severe head injury remain unclear. On searching the literature, very few randomized studies relating to this topic exist and with almost no class 1 evidence.^[5] Whether DC is beneficial in raised ICP after severe head injury is a topic for controversy.

The aim of this retrospective study was to analyze the clinicoradiological factors associated with the prognosis of severe TBI in patients undergoing DC.

Subjects and Methods

Study design

This was a retrospective case series study undertaken from April 2014 to March 2016. The study group included 85 patients (admitted at Tata Main Hospital, Jamshedpur) with severe diffuse TBI with clinical and radiological evidence of intracranial hypertension who were refractory to first-tier therapies (conservative measures) to reduce the ICP

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and underwent DC. A preinformed consent in an uniform format was taken from all these patients for being a part of this study and their details to be published. The clinical and demographic profile, complications, and factors associated with prognosis were analyzed. Exclusion criteria were all admissions with polytrauma (chest, abdominal, or bony injuries) and patients with age <10 years.

Results

A total of 85 patients with severe TBI who underwent a DC were studied. Fifty-five (64.8%) were male, and 30 (35.2%) were female with a male:female ratio being 1.8:1. Mean age of presentation was 36 years. Most of the patients were of age group 31–40 years in 32.9% (28 cases) followed by 41–50 years in 17 cases (20%) [Table 1].

Most common mode of injury was road traffic accident in 59 (69.5%) cases, followed by fall from height in 14 (16.5%) cases and assault in 12 (14%) cases [Table 2].

Patients were divided into two groups based on Glasgow coma scale (GCS) score at presentation. Forty-nine (58%) cases had GCS score of 4–6 whereas 36 (42%) cases had GCS score 7–8 [Table 3].

Computed tomography (CT) scan brain was advised immediately after resuscitation and classified on the basis of Marshall CT classification. Most patients were of type V in 37 (43.5%) followed by type IV in 23 (27.6%) cases and type III in 20 (23.5%) cases [Table 4].

Sixty-six (77.6%) cases were treated early that is within 24 h whereas 19 (22.3%) cases were treated late after 24 h (mainly in those cases in which after initial medical management initial improvement occurred followed by deterioration) [Table 5].

Bilateral frontotemporoparietal craniectomy was done in 29% cases followed by unilateral frontotemporoparietal craniectomy in 71% cases [Table 6].

Both neurological and nonneurological complications occurred postoperatively. Among neurological complications - external cerebral herniation, postsurgical CNS, infection, hydrocephalus, and venous infarction were common. Among nonneurological complications - ventilator-associated pneumonia, dyselectrolytemia, thrombophlebitis, and sepsis were common [Table 7].

The outcome was measured by Glasgow outcome score at 6 months. It was seen that patients operated early had better surgical outcome in comparison to patients operated late [Table 8].

Patients operated with GCS score 7–8 had better surgical outcome than with GCS score 4–6. Mortality was more in patient of GCS 4–6 (18.4%) than with GCS score 7–8 (11.1%). Overall mortality was in 13 (15.3%) cases [Table 9].

Table 1: Distribution of patients as per age and gender in each decade

Age (years)	Number of patients (%)	Male (%)	Female (%)
11-20	5 (5.9)	4 (80)	1 (20)
21-30	14 (6.5)	8 (57.14)	6 (42.8)
31-40	28 (32.9)	18 (64.2)	10 (35.8)
41-50	17 (20)	11 (64.7)	6 (35.3)
51-60	16 (18.8)	11 (64.7)	5 (31.2)
61-70	5 (5.9)	3 (60)	2 (40)
Total	85	55 (64.8)	30 (35.2)

Table 2: Mode of injury with number of patients and percentage in each category

	Number of cases (%)
RTA	51 (60)
2 wheeler	30 (58.8)
4 wheeler	5 (9.8)
Pedestrian	16 (31.4)
Fall from height	15 (17.6)
Rooftop	4 (26.7)
Stairs	9 (60)
Balcony	2 (13.3)
Fall of heavy object overhead	11 (12.9)
Brick wall	7 (63.6)
Stone boulder	3 (27.7)
Television	1 (9.1)
Assault	8 (9.4)
Blunt injury	5 (62.5)
Sharp injury	1 (12.5)
Firearm	2 (25)

RTA – Road traffic accident

Table 3: Number of patients with Glasgow coma scale at the time of admission

GCS at the time of admission	Number of cases (%)
4-6	49 (57.6)
7-8	36 (42.3)

GCS – Glasgow coma scale

Younger patients had better surgical outcome than patients of age >50 years, and mortality was more in patients of >50 years age [Table 10].

Patients with higher Marshall CT grading had poorer prognosis. All mortality was in patients with Marshall CT grade IV and grade V [Table 11].

Discussion

DC has been a treatment modality since long with a controversial history. While many studies have shown the efficacy of DC in reducing ICP and improving mortality from severe TBI, others have questioned on its usefulness. Historically, the removal of different parts of the skull has been utilized in the management of severe TBI after the first reports of this surgical technique directed at controlling

Table 4: Detailed Marshall computed tomography classification with number of patients in each type

Type	Criteria	Number of cases (%)
Type I	No visible intracranial pathology	-
Type II	Midline shift of 0-5 mm Basal cisterns remain visible No high or mixed density lesions >25 cm ³	5 (5.9)
Type III	Midline shift of 0-5 mm Basal cisterns compressed or completely effaced No high or mixed density lesions >25 cm ³	20 (23.5)
Type IV	Midline shift >5 mm No high or mixed density lesions >25 cm ³	23 (27.6)
Type V	Any lesion evacuated surgically	37 (43.5)
Type VI	No high or mixed density lesions >25 cm ³ Not surgically evacuated	-

Table 5: Number of cases with respect to timing of surgery early if done within 24 h and late if done after 24 h

	Number of cases (%)
Within 24 h	66 (77.6)
More than 24 h	19 (22.3)

Table 6: Number of patients with different types of surgery performed

Types of surgery	Number of cases (%)
Unilateral frontotemporo-parietal craniectomy	61 (71)
Bilateral frontotemporal craniectomy	24 (29)

Table 7: Postsurgical complications

Neurological complication	n (%)	Nonneurological complication	n (%)
Total cases	23 (27.1)	Total cases	17 (20)
External cerebral herniation	10 (43.5)	Ventilator-associated pneumonia	5 (29.4)
Postsurgical CNS infection	2 (8.7)	Dyselectrolytemia	7 (41.2)
Hydrocephalus	9 (39.1)	Thrombophlebitis	3 (17.6)
Venous infarction	2 (8.7)	Sepsis	2 (11.8)

CNS – Central nervous system

ICH were published by Cushing.^[6] This surgical procedure does not have any effect on primary brain damage, but it can reduce the serious consequences of secondary lesions, such as the elevation of ICP and cerebral displacements or distortions. In one of the studies, Wilberger *et al.*^[7] had suggested that raised ICP was one of the most important factors in predicting outcomes following severe TBI. Most of the studies done were retrospective with

Table 8: Outcome as per Glasgow outcome score of treatment in terms of timing of surgery

GOS	Early surgery (%)	Late surgery (%)
5 (good recovery)	21 (31.8)	3 (15.8)
4 (moderate disability)	19 (28.8)	2 (10.5)
3 (severe disability)	11 (16.7)	4 (21.5)
2 (vegetative state)	8 (12.1)	4 (21.5)
1 (dead)	7 (10.6)	6 (31.6)
Total	66 (77.6)	19 (22.4)

GOS – Glasgow outcome score

Table 9: Outcome as per Glasgow outcome score with respect to Glasgow coma scale at the time of admission

GOS	GCS 4-6, n (%)	GCS 7-8, n (%)	Total
Total	49 (57.6)	36 (42.4)	85
5 (good recovery)	11 (22.4)	13 (36.1)	24 (28.2)
4 (moderate recovery)	10 (20.4)	11 (30.5)	21 (24.7)
3 (mild recovery)	10 (20.4)	5 (13.9)	15 (17.6)
2 (vegetative state)	9 (18.4)	3 (8.3)	12 (14.1)
1 (dead)	9 (18.4)	4 (11.1)	13 (15.3)

GOS – Glasgow outcome score; GCS – Glasgow coma scale

Table 10: Outcome as per Glasgow outcome scale of treatment in different age groups

GOS	11-20	21-30	31-40	41-50	51-60	61-70	Total
5 (good recovery)	3	7	7	4	3	-	24
4 (moderate recovery)	2	4	10	4	1	-	21
3 (mild recovery)	-	1	7	5	2	-	15
2 (vegetative state)	-	1	3	2	4	2	12
1 (dead)	-	1	1	2	6	3	13
Total	5	14	28	17	16	5	85

GOS – Glasgow outcome score

Table 11: Outcome as per Glasgow outcome scale in patients with respect to Marshall computed tomography grading

GOS	Marshall CT grading				Total
	II	III	IV	V	
5 (good recovery)	4	9	7	4	24
4 (moderate recovery)	1	7	10	3	21
3 (mild recovery)	-	2	2	11	15
2 (vegetative state)	-	2	2	8	12
1 (dead)	-	-	2	11	13
Total	5	20	23	37	85

CT – Computed tomography; GOS – Glasgow outcome score

small patient populations of variable composition in terms of age and management criteria. Taking this point into consideration, the role of DC in patient care with TBI has been an upcoming field for researchers also. A Cochrane Collaboration review in 2006^[8] showed only one randomized study in children.^[9] The overall conclusion from the review

of various studies involving nonrandomized retrospective and prospective trials suggested the benefit for DC in TBI but were not conclusive enough with evidence to support the routine use of DC. Other randomized studies have since been on paper, and the result of the DECRA study was released in 2011.^[10] Limited DC was introduced in 1966 by Miyazaki and subsequently popularized by Kjellberg and Prieto in 1971.^[11] In severe TBI, two groups of patients must be recognized. First (Group A) are the patients with mass lesions and diffuse raised ICP (such as extensive/severe acute traumatic subarachnoid hemorrhage or subdural hematoma) who require surgery to address the cause of their raised ICP. These patients show a very rapid progression toward intractable raised ICP, which becomes unresponsive to medical management if surgical intervention is delayed. Second (Group B) are the patients without any surgically amenable mass lesions but with medically intractable raised ICP. A third group as given in literature is from centers where ICP is not monitored and where the severity of ICP is found on clinical examination.^[12] Patients in group A are more likely to improve with DC than patients in group B, even more if the surgery is done early. Advances in imaging diagnostics and the neurointensive management of severe TBI have been able to keep up interest in the correct use of DC for elevated ICP. However, controversies with this technique exist in relation to precise indications, timing, and long-term functional results along with the need to study the complications and costs associated with the DC procedure.^[7,8,13,14] Though DC is a simple technical procedure, the cost involved adds to the economic burden of any society and complications commonly occur, sometimes with significant clinical impacts on patients outcome. Some unavoidable complications following the removal of part of cranial vault occurs because of changes in the dynamics of cerebrospinal fluid (CSF) circulation and cerebral blood flow.^[15] External herniation, measured as brain tissue in the center of the bone defect >1.5 cm above the plane of the outer table of the cranium, was found to occur in 26% of 108 craniectomies.^[16] Herniation in the early postdecompression period occurs quite often and has been thought to be related to progressive swelling of the underlying brain. Unavailability of a protective skull leading to reduced resistance and increased hydrostatic pressure may be an important reason for this. Transcapillary leakage of fluid causing edema in these circumstances has been demonstrated in animal studies but not in patients with craniectomy.^[16] Infections associated with DC may occur in the intermediate or late postdecompression period. Factors attributing to increased infection include larger scalp incisions, compromise of vascular pedicle to the flap or air sinuses from the large bone flap, and duroplasty using artificial substitutes.^[17] Hydrocephalus has been reported in between 10% and 40% of patients in the delayed period (>1 month).^[18-20] It could be that symptomatic hydrocephalus results from the failure of the altered CSF dynamics.^[21] When making decisions for surgery, the nature

of associated complications has to be kept in mind, and they have an important role too. Although the surgery of DC is relatively simple, it also has significant potential for adverse outcomes, especially considering the emergency nature of the procedures and the chance that younger neurosurgeons are more likely to undertake the surgery. With increased severity of disease, elderly patients and those on aspirin or other anticoagulants complications of decompressive craniectomy have been found to be increased.^[17] Until now, there has been no class I clinical evidence related to DC for the management of refractory intracranial hypertension in severe TBI in adults.^[8,10,22] It should also be emphasized that no evidence exists for the execution of primary DC.^[23-25] The European study RESCUE-ICP, currently in development, has confirmed that DC has a place in the management of patients with refractory ICP.

In our study, we were unable to show a statistically significant association between the presence of complications (neurological or systemic) or the type of DC (primary or secondary) in relation to mortality, which can be explained in part by the small sample size. Our study also has several limitations. It is a descriptive, retrospective study with a relatively small number of patients, conducted in a single center, which reduces statistical power. Our study population is heterogeneous in terms of their clinical characteristics, indications, and opportunity for DC, and technique used, which limits the precision of our results. No long-term follow-up of patients was done.

DC should be considered in patients with TBI with refractory and elevated ICP. DC is not simple, standard operation without adverse effects. Although associated with complications, the risk of complications following DC should be weighed against the life-threatening circumstances under which this surgery is performed. In patients who suffered severe head injury with refractory intracranial hypertension, early DC employed in the first few hours after injury before the onset of irreversible ischemic changes may be an effective method to treat the secondary deterioration that commonly leads to death or severe neurological deficit.

Conclusions

Early surgery, better GCS score on admission, relatively younger age, and lower Marshall CT grade on admission show a better surgical outcome.

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Conflicts of interest

There are no conflicts of interest.

References

- Rosenfeld JV, Maas AI, Bragge P, Morganti-Kossmann MC, Manley GT, Gruen RL. Early management of severe traumatic brain injury. *Lancet* 2012;380:1088-98.
- Faul M, Wald MM, Rutland-Brown W, Sullivent EE, Sattin RW. Using a cost-benefit analysis to estimate outcomes of a clinical treatment guideline: Testing the Brain Trauma Foundation guidelines for the treatment of severe traumatic brain injury. *J Trauma* 2007;63:1271-8.
- Sahuquillo J, Arikan F. Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury. *J Pediatr Surg* 2006;41:83-7.
- Stiver SI. Complication of decompressive craniectomy for traumatic brain injury. *Childs Nerv Syst* 2006;22:1268-74.
- Chibbaro S, Tacconi L. Role of decompressive craniectomy in the management of severe head injury with refractory cerebral edema and intractable intracranial pressure. *Acta Neurochir (Wien)* 2008;150:1241-7.
- Cushing H. The establishment of cerebral hernia as a decompressive measure for inaccessible brain tumors: With the description of intramuscular methods of making the bone defect in temporal and occipital regions. *Surg Gynecol Obstet* 1905;1:297-314.
- Wilberger JE Jr., Harris M, Diamond DL. Acute subdural hematoma: Morbidity, mortality, and operative timing. *J Neurosurg* 1991;74:212-8.
- Sahuquillo J, Arikan F. Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury. *Cochrane Database Syst Rev* 2006;1:CD003983.
- Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, *et al.* A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. *Childs Nerv Syst* 2001;17:154-62.
- Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, *et al.* Decompressive craniectomy in diffuse traumatic brain injury. *N Engl J Med* 2011;364:1493-502.
- Kjellberg RN, Prieto A Jr. Bifrontal decompressive craniotomy for massive cerebral edema. *J Neurosurg* 1971;34:488-93.
- Mezue WC, Ndubuisi C, Ohaegbulam SC, Chikani M, Erechukwu U. Cranial bony decompressions in the management of head injuries: Decompressive craniotomy or craniectomy? *Niger J Clin Pract* 2013;16:343-7.
- Farahvar A, Gerber LM, Chiu YL, Härtl R, Froelich M, Carney N, *et al.* Response to intracranial hypertension treatment as a predictor of death in patients with severe traumatic brain injury. *J Neurosurg* 2011;114:1471-8.
- Cushing H. I. Subtemporal decompressive operations for the intracranial complications associated with bursting fractures of the skull. *Ann Surg* 1908;47:641-4.
- Finkelstein EA, Corso PS, Miller TR. Incidence and Economic Burden of Injuries in the United States. New York: Oxford University Press; 2006.
- Yang XF, Wen L, Shen F, Li G, Lou R, Liu WG, *et al.* Surgical complications secondary to decompressive craniectomy in patients with a head injury: A series of 108 consecutive cases. *Acta Neurochir (Wien)* 2008;150:1241-7.
- Stiver SI. Complications of decompressive craniectomy for traumatic brain injury. *Neurosurg Focus* 2009;26:E7.
- Howard JL, Cipolle MD, Anderson M, Sabella V, Shollenberger D, Li PM, *et al.* Outcome after decompressive craniectomy for the treatment of severe traumatic brain injury. *J Trauma* 2008;65:380-5.
- Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. *J Neurosurg* 2006;104:469-79.
- Morgalla MH, Will BE, Roser F, Tatagiba M. Do long-term results justify decompressive craniectomy after severe traumatic brain injury? *J Neurosurg* 2008;109:685-90.
- Guerra WK, Gaab MR, Dietz H, Mueller JU, Piek J, Fritsch MJ. Surgical decompression for traumatic brain swelling: Indications and results. *J Neurosurg* 1999;90:187-96.
- Sahuquillo J, Martínez-Ricarte F, Poca MA. Decompressive craniectomy in traumatic brain injury after the DECRA trial. Where do we stand? *Curr Opin Crit Care* 2013;19:101-6.
- Chen SH, Chen Y, Fang WK, Huang DW, Huang KC, Tseng SH. Comparison of craniotomy and decompressive craniectomy in severely head-injured patients with acute subdural hematoma. *J Trauma* 2011;71:1632-6.
- Kolias AG, Belli A, Li LM, Timofeev I, Corteen EA, Santarius T, *et al.* Primary decompressive craniectomy for acute subdural haematomas: Results of an international survey. *Acta Neurochir (Wien)* 2012;154:1563-5.
- Li LM, Kolias AG, Guilfoyle MR, Timofeev I, Corteen EA, Pickard JD, *et al.* Outcome following evacuation of acute subdural haematomas: A comparison of craniotomy with decompressive craniectomy. *Acta Neurochir (Wien)* 2012;154:1555-61.