ORIGINAL ARTICLE

Role of Decompressive Craniectomy in the Management of Traumatic Brain Injury Associated with Elevated ICP and Brain Edema

SHAHID NAWAZ, FAKHAR HAYAT, SARFARAZ KHAN
SARAH REHMAN, NOOR SARDAR
Neurosurgery Department, Medical Teaching Institution (MTI), District Headquarter (DHQ), Gomal Medical College (GMC), Dera Ismail Khan
DOI: https://doi.org/10.36552/pjns.v23i3.355

ABSTRACT

Objective: *The aim of this study was to analyze the outcome of decompressive craniectomy in patients of traumatic head injury done in MTI, DHQ teaching hospital.*

Materials and Methods: *189 patients with head injury were operated in a period of 15 months (April 2018-June 2019). Among 189 patients only 50 (32 men and 18 women) were treated with decompressive craniectomy (DC). We analyze only 50 cases that were treated with DC. Demographic details, GCS, time of DC and complications were recorded. Glasgow Outcome Scale was used as a measure of clinical outcome.*

Results: *Out of 50 patients, 18 (36%) showed a complete recovery, mild disability was found in 10 (20%) patients. The percentage of severe disability was observed in 7 (14%) patients asexual condition existed in 5 (12%) patients and the mortality rate was 12% (6 patients). 4 (8%) patients did not report us back. We excluded them from our final result analysis. A good result was presented in 28 patients (56%). Age was found to have a statistically significant association with clinical outcomes (p = 0.002). Moreover, the patients experiencing DC within 18 hours had an improved result (p = 0.001). The better GCS score before surgery was associated with good results (p = 0.001).*

Conclusion: *Decompressive craniectomy is associated with better clinical outcomes in patients with traumatic brain injury associated with refractory cerebral edema and elevated intracranial pressure.*

Key Words: *Decompressive craniectomy (DC), intracranial pressure (ICP), Traumatic brain injury (TBI), duraplasty.*

INTRODUCTION

Traumatic brain injury (TBI) leads to substantial mortality around the globe.[1] TBI is associated with increased intracranial pressure (ICP) as a result of cerebral edema, diminished cerebral perfusion, and brainstem herniation.[2] TBI is distinct as a severe injury to the head produced by blunted or piercing trauma or from acceleration/deceleration forces without worsening, inherited complications.[3,4] In the United States, nearly fifty thousand deaths occur due to TBI annually.[5,6] US Center for Disease Control and Prevention (CDC) defines TBI as a bump or shock to the brain, or penetrative cerebral injury affecting brain functioning.[7] The TBIs are very often found in blast victims and are associated with the production of high-pressure waves along with exposure to the projectiles.[8]

The intensity of TBI ranges from mild to severe. Common symptoms are headache, nausea, dizziness, coma, loss of consciousness (LOC). TBI is often assessed via Glasgow Coma Scale Score (GCS).[9,10] Severity of TBI can be easily evaluated using GCS, especially in emergency cases. Each part of this scale explains the main function of the patient.[11] GCS score between 13 – 15 is categorized as mild, score 9 – 12

Date of Submission: 28-7-2019
Date of Printing: 30-9-2019

moderate and 8 or less as severe TBI.[12,13]

The systematic approach is adopted for the management of brain edema and increased ICP.[14,15] DC had been applied to control ICP linked with unusual situations, comprising of ischemic disease, intracranial neoplasm, and diffuse edema from TBI. The advantage of DC in the handling of malicious infarction had been shown by previous study.[16]

Initial conservative treatment modalities are vitally stabilizing patient, improved ventilation, and head-up position of the patient. Treatment options include administration of hypertonic saline, Mannitol, and inotropes.[17,18]

Despite the improved treatment algorithms and advanced monitoring systems, the mortality rate is high among patients with head injuries. DC is an effective treatment modality for patients with TBI with cerebral edema, increased ICP, decompensated intracranial hypertension.[19,20]

The aim of this study was to present our experience of decompressive craniectomy in patients of traumatic head injury associated with elevated ICP and brain edema in MTI, DHQ teaching hospital.

MATERIAL AND METHODS

Study Design

This prospective, study was conducted from April 2018 – June 2019.

Data Collection

The study was initiated after approval from the research ethical review committee. The data was collected from the MTI, DHQ teaching hospital.

189 patients with head injury were operated in our hospitals in a period of 15 months (April 2018-June 2019). Among 189 patients, only 50 (32 men and 18 women) were treated with decompressive craniectomy (DC). So here we analyze only 50 cases that were treated with DC. All the selected subjects were provided duraplasty (39 unilateral and 11 bilateral) along wide DC (> 35 cm^2). 29 patients were operated within 18 hours of trauma, and remaining underwent DC within 52 hours.Patients were selected on the basis of elevated ICP. Non-invasive measurement of intracranial pressure was made. Ocular ultrasonography was employed to measure ICP. Optic nerve sheath diameter (ONSD) recorded with the help of probe placed over closed eyelids. ONSD of > 5.2 corresponds to ICP of 25 mmHg.

Management

Intracranial pressure was maintained below 22 mmHg, and cerebral perfusion pressure (CPP) was maintained at 75 mmHg or above. An external ventricular drain was placed in 6 (12%) cases. In the residual patients, intraparenchymal ICP bolt was used.

Head elevation up to 30 was used to improve venous drainage. In case of ICP increases, the first treatment modality was provisional modest hyperventilation with a CO2 and Mannitol bolus.

Inclusion Criteria

In case of failure of conservative and medical treatment, i.e., high ICP (≥ 25 mmHg) for greater than 30 – 35 minutes, barbiturate coma and decompressive craniectomy were considered. Treatment option was decided depending upon the patient, though, considering elevated in ICP value, age of the subject, pupil magnitude and response, along with injury duration. According to the treatment algorithms adopted, DC was considered as the last treatment option, after the failure of other options. Demographic details, GCS, time of DC and complications were recorded. Glasgow Outcome Scale was used as a measure of clinical outcome. GOS was measured one week after surgery. GOS of 4 and 5 were considered as clinically good results.

Exclusion Criteria

Those who refused surgery or did not gave the concept were excluded similarly patients with comorbiditis were also excluded.

Data Analysis

Data was entered and analyzed using SPSS version 22. Frequency and percentage of tables were generated. The Chi-square test was applied. $P \leq 0.05$ was considered as statistically significant.

RESULTS

50 patients were included in this study. 18 (36%) were female and 32 (64%) were male. Mean age of patients was 52 years with a standard deviation of 10.6years. Their demographic and treatment profile is given in Table 1. All subjects were provided duraplasty with 39 (78%) unilateral and 11 (22%) bilateral, along wide DC (> 35 cm2). 29 (58%) patients were operated within 18 hours of trauma, and remaining underwent DC in 52 hours (Table 1).

Table 1: *Detail of subjects and treatment.*

Variables		Frequency (N)	Percentage (%)
Gender	Male	32	64
	Female	18	36
Duraplasty	Unilateral	39	78
	Bilateral	11	22
Operated Time	18 hours	29	58
	52 hours	21	42

In Table 2, the outcome of the clinical study is shown. Out of 50 patients 18 (36%) show complete recovery, mild disability was found in 10 (20%) patients. The percentage of severe disability was observed in 7 (14%) patients, come in 5 (12%) patients and the mortality rate was 12% (6 patients). 4 (8%) patients do not report us back; we excluded them from our final result analysis.

Table 2: *Clinical outcome.*

Variables	Frequency (N)	Percentage (%)
Complete recapture	18	36
Mild disability	10	20
Severe disability	7	14
Vegetative state	5	10
Death rate	6	12
No-follow up	4	8

Generally, a good result was reported in 28 patients (56%). Majority of the patients who presented satisfactory clinical results were of younger age with 33 years. Poor outcomes were reported among patients of mean age 52 years. Age was found to have a statistically significant association with clinical outcomes ($p = 0.002$). Moreover, patients experiencing DC within 18 hours had an improved result. ($p = 0.001$). As anticipated, a better GCS score before surgery was associated with good results ($p = 0.001$) (Table 3).

COMPLICATIONS

The surgical complication rate was 12.3%. Five patients showed cerebral contusion following DC (two contralaterally, three ipsilateral).Two patients with advanced hydrocephalus treated with shunt placement. Three patients who had contaminated wounds with Staphylococcus aureus, were treated with IV antibiotics.

Table 3: *Clinical outcome in association with age, time of DC and GCS.*

Variables	Frequency (N)	Mean Age 33 Yrs	Mean Age 51 Yrs	Surgery in 18 Hrs	Surgery in 52 Hrs	Low Level of GCS*	High Level of GCS*
Complete recovery	18	11	7	12	6	7	11
Mild disability	10	4	6	7	3	6	4
Severe disability	7	2	5	6	1	3	4
Asexual condition	5	1	4	2	3	3	2
Death rate	6	3	3	2	4	3	3
Total	46	21	25	29	17	22	24

**Low level of GCS (3 – 5), High level of GCS (6 – 8)*

DISCUSSION

TBI is associated with significant morbidity and mortality, accounting high economic burden.[20] Brain edema resulting from trauma, elevated ICP and lower CPP leading to brain ischemia.[21-22]

These factors are related to poor clinical

outcomes[23-25] In past few years, numerous researcher[26,27] have shown a decrease in mortality rate and better clinical outcomes in patients of cerebral edema by treatment with DC.[28] Our study advocates similar findings. Patients of TBI with early surgical intervention showed a better outcome as compared to those who were treated at 52 hours. Faleiro et al.[29] analyzed 89 patients into < 6 h, 6–24 h, and > 24 h for DC and established that patients who were treated timely had 59% mortality as associated with the 53% of patients who had the surgery later. Al-Jishi et al.[30] initiated that the primary DC had 45.5% good outcomes and 40.9% mortality whereas, secondary DC had 73.1% good outcomes and 15.4% mortality. The mortality rate after decompressive craniectomy ranges from 13.5% to 90%.[31]

Polin et al.[32] stated a 23% mortality and 37% improved clinical outcome in patients of TBI after DC. According to another study with 12 months follow-up, 19% mortality rate was reported with 58% minor disability.The current study showed a 12% mortality rate and 20% minor disability. Wettervik et al.[33] reported that the relative risk (RR) of mortality at discharge or six months was 0.62 with *P*value = 0.03 and further added that the mortality rate is decreased with the timely DC as related to the usual medical management and late DC.

Honeybul et al,[34] supported DC for severe TBI (2004–2010). He done his research on 186 patients and indicated that not a single patient developed a level of moderate disability, numerous did seem to have modified to their incapacity and recalibrated their potentials for the worth of life to a level of disability that they have earlier supposed intolerable.

We did a one-sided craniectomy, in patients with edema, limited to only one cerebral hemisphere. Among patients of generalized cerebral edema, frontal decompression bilaterally was done. This approach is in agreement with other previous studies.[31] According to the literature, [31] commonly reported complications include hygroma, hydrocephalus, meningitis, wound contamination, and cerebral contusion. We identified 3 wound infections, 5 cases of brain contusion and 3 cases with hydrocephalus. These statistics mark the decompressive craniectomy as a harmless practice, thus frequent use of DC is advocated. In previous studies, the time period between the decompressive craniectomy and the cranioplasty may vary from four weeks up to 12 months.[35]

In our setting, the cranioplasty used to perform during 2 – 4 week duration, as this approach can decrease the hazard of hydrocephalus and epilepsy.It also favors the timely restoration of patient functionality and reducing the complications. We observed significant clinical progress after an early cranioplasty. Early surgical intervention and early cranioplasty are thus advocated.[35]

The DECRA test printed by Cooper et al.[36] in 2011, was the famed RCT to regulate the therapeutic outcome of DC in TBI. For the duration of 2002 to 2010, 155 patients who had TBI and either GCS score was lower than 8 or CT weredemonstrated a moderate diffuse brain injury were registered. Patients with refractory ICP (ICP > 20 mmHg) for 15 minutes within a 1-hour period) were divided to 2 group and 72 patients implemented DC plus maximal medical care and 82 patients had maximal medical supervision counting barbiturate and hypothermia. The assumption of this work is DC lessening ICP and the measurement of stay in the intensive care unit, but is related with more uncomplimentary results.[36]

Analyses of ICP-related outcomes from DECRA have reached from opinions that ICP decrease may not essentially result in better outcomes, to disapproval of DECRA study proposal signifying that an advanced ICP threshold be used for accomplishment DC in TBI.[37,38] The DECRA trial intended to measure the usefulness of initial DC (within 72 hours after trauma) in moderate ICH (ICP > 20 mmHg) for fifteen minutes within a one hour period.[36] On the other hand, the purpose of RESCUEicp trial was to evaluate the efficacy of DC in a last-stage usage with refractory ICH (ICP > 25 mmHg) for lasting more than 1 – 12 hours. The patients with intracranial hematoma were not involved in DECRA trial, but in RESCUEicp sample, the patients with intracranial hematoma were accounted for nearly 20% of cases. The difference of two trials in procedure of surgical way is unilateral hemicraniectomy was not allowable in DECRA trial dissimilar in RESCUE ICP trial. This analysis provisions the discussions of preceding hypothesis that DC only surges the number of patients enduring in a vegetative state.[39]

LIMITATIONS OF STUDY

This study was single centric, there is a need of muti-centric study with larger sample size.

CONCLUSION

Decompressive craniectomy is associated with better

clinical outcomes in patients of traumatic brain injury. It is an effective technique to decrease intractable elevated intracranial pressure.

Additional Information

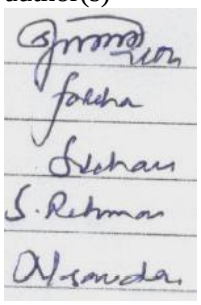
Disclosures: Authors report no conflict of interest.
Human Subjects: Consent was obtained by all patients/ participants in this study.
Conflicts of Interest:
In compliance with the ICMJE uniform disclosure form, all authors declare the following:
Financial Relationships: All authors have declared that 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.

Address for Correspondence:
Dr Shahid Nawaz, Assistant Professor & Head Department of Neurosurgery, Medical Teaching Institution (MTI), District Headquarter (DHQ), Gomal Medical College (GMC), Dera Ismail Khan
Email: nsgshahidkhattak2012@gmail.com

REFERENCES

1. Brain Trauma Foundation Guidelines for the management of severe traumatic brain injury. J Neurotrauma, 2017; 24 Suppl. 1: 1–106.
2. Gouello G, Hamel O, Aschnoune K, Bord E, Robert R, Buffenoir K. Study of the long-term results of decompressive craniectoomy after severe traumatic brain injury base on a series of 60 consecutive cases. Sci World J. 2014:1–10.
3. Goodman MD, Makley AT, Lentsch AB, Barnes SL, Dorlac GR, Dorlac WC, et al. Traumatic brain injury and aeromedical evacuation: when is the brain fit to fly? J Surg Res. 2018; 164: 286–293.
4. 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. 2016; 17: 154–162.
5. Nirula R, Millar D, Greene T, McFadden M, Shah L, et al. Decompressive craniectomy or medical management for refractory intracranial hypertension: an AAST-MIT propensity score analysis. J Trauma ACS. 2015; 76 (4): 944–955.
6. Champion HR, Holcomb JB, Young LA. Injuries from explosions. J Trauma. 2019; 66: 1468–1476.
7. Shah SM, Kelly KM. Emergency Neurology: Principles and Practice, Cambridge University Press, Cambridge, UK, 2013.
8. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. Lancet. 2017; 2: 81–84.
9. Evans RW. Neurology and Trauma, Oxford University Press, 2016.
10. Kraus JF, Nourjah P. The epidemiology of mild, uncomplicated brain injury. J Trauma, 2018; 28: 1637–1643.
11. Saatman KE, Duhaime A, Bullock R et al. Classification of traumatic brain injury for targeted therapies and Workshop Scientific team and Advisory Panel Members. J Neurotrauma, 2018; 25: 719–738.
12. Elf K, Nilsson P, Enblad P. Outcome after traumatic brain injury improved by an organized secondary insult program and standardized neuro-intensive care. Crit Care Med. 2015; 30: 2129–2134.
13. Menon DK. Cerebral protection in severe brain injury: physiological determinants of outcome and their optimisation. Br Med Bull. 2016; 55: 226–258.
14. Polderman KH, Tjong Tjin Joe R, Peerdeman SM, Vantertop WP, Girbes AR. Effects of therapeutic hypothermia on intracranial pressure and outcome in patients with severe head injury. Intensive Care Med. 2017; 28: 1563–1573.
15. Hutchinson PJ, Menon DK, Kirkpatrick PJ. Decompressive craniectomy in traumatic brain injury – time for randomised trials? Acta Neurochir (Wien). 2015; 147: 1–3.
16. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, et al. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol. 2017; 6: 215–222.
17. Roberts I. Barbiturates for acute traumatic brain injury. Cochrane Database Syst Rev. 2:CD000033. 2018.
18. Doerfler, A., Forsting, M., Reith, W., et al. Decompressive craniectomy in a rat model of malignant cerebral hemispheric stroke: experimental support for an aggressive therapeutic approach. J. Neurosurg. 2017; 85: 853–859.
19. Fisher, C.M., and Ojeman, R.G. Bilateral decompressive craniectomy for worsening coma in acute subarachnoid hemorrhage: observation in support of the procedure. Surg. Neurol. 2015; 41: 65–74.
20. Jennett B. Epidemiology of severe head injury: socioeconomic consequence of avoidable mortality and morbidity. In: Scriabine A, Teasdale GM, Tettenborn D, Young W, editors. Nimodipine. Pharmacological and clinical results in cerebral ischemia. Berlin 7 Springer; 2019: 225 – 33.
21. Dewitt DS, Jenkins JW, Prough DS. Enhanced vulnerability to secondary ischemic insults after experimental traumatic brain injury. New Horizons, 2015; 3: 376 – 83.

22. Kunze E, Meixensberger J, Janka M, et al. Decompressive craniectomy in patients with uncontrollable intracranial hypertension. Acta Neurochir (Suppl. 71): 16 - 8.
23. Juul N, Morris GF, Marshall SB. Intracranial hypertension and cerebral perfusion pressure: influence on neurological deterioration and outcome in severe head injury—The Executive Committee of the International Selfotel Trial L. F Marshall. J Neurosurg. 2017; 92: 1 – 6.
24. Langfitt TW. Incidence and importance of intracranial hypertension in head injury patients. In: Beks JWF, Bosch DA, Brock M, editors. Intracranial pressure III. Berlin7 Springer; 2015: 67 – 72.
25. Marshall LF, Marshall SB, Klauber MR, et al. A new classification of head injury based on computerized tomography. J Neurosurg. 2018; 75 (Suppl. 1): 14 - 20.
26. Miller JD, Becker DP, Ward JD. Significance of intracranial hypertension in severe head injury. J Neurosurg. 2017; 47: 503 - 16.
27. Marshall LF. Head injury: past, present and future. Neurosurgery, 2019; 47: 546 - 61.
28. Whitfield PC, Patel H, Czosnyka M, et al. Bifrontal decompressive craniectomy in the management of post-traumatic intracranial hypertension. Br J Neurosurg. 2015; 14: 292.
29. Faleiro RM, Faleiro LC, Caetano E, Gomide I, Pita C, Coelho G, et al. Decompressive craniotomy: Prognostic factors and complications in 89 patients. Arq Neuropsiquiatr. 2018; 66: 369-73.
30. Al-Jishi A, Saluja RS, Al-Jehani H, Lamoureux J, Maleki M, Marcoux J, et al. Primary or secondary decompressive craniectomy: Different indication and outcome. Can J Neurol Sci. 2017; 38: 612-20.
31. Munch E, Horn P, Schurer L, et al. Management of severe traumatic brain injury by decompressive craniectomy. Neurosurgery, 2015; 47: 315 - 21.
32. Polin RS, Shaffrey ME, Bogaev CA, et al. Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral oedema. Neurosurgery, 2018; 41: 84 - 92.
33. Wettervik TS, Lenell S, Nyholm L, Howells T, Lewén A, Enblad P, et al. Decompressive craniectomy in traumatic brain injury: Usage and clinical outcome in a single centre. Acta Neurochir (Wien). 2018; 160: 229.
34. Honeybul S, Ho KM, Lind CR, Gillett GR. Validation of the CRASH model in the prediction of 18-month mortality and unfavorable outcome in severe traumatic brain injury requiring decompressive craniectomy. J Neurosurg. 2014; 120: 1131-7.
35. Bostrom S, Bobinski L, Zsigmond P, et al. Improved brain protection at decompressive craniectomy—a new method using Palacos R-40 (methylmethacrylate). Acta Neurochir. 2015; 147: 279 – 81.
36. 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: 1493–1502.
37. Tagliaferri F, Zani G, Iaccarino C, Ferro S, Ridolfi L, Basaglia N, et al. Decompressive craniectomies, facts and fiction: a retrospective analysis of 526 cases. Acta Neurochir (Wien), 2012; 154: 919–926.
38. Honeybul S, Ho KM, Lind CR. What can be learned from the DECRA study. World Neurosurg. 2013; 79: 159–161.
39. Kolias AG, Kirkpatrick PJ, Hutchinson PJ. Decompressive craniectomy: past, present and future. Nat Rev Neurol. 2013; 9: 405–415.

AUTHORSHIP AND CONTRIBUTION DECLARATION			
Sr.#	**Author's Full Name**	**Intellectual/Contribution to Paper in Terms of:**	
1.	**Shahid Nawaz** (Main/Principal Author).	1. Basic Study Design and wrote introduction.	Signature by the author(s) 
2.	Fakhar Hayat (2nd Author)	2. Wrote Discussion	
3.	SarfarazKhan (3rd Author)	3. Analysis of data, References and interpretation of results etc.	
4.	**Sarah Rehman** (4th Author)	4. Literature review and manuscript writing and data Calculations	
5.	**Noor Sardar** (5th Author)	5. Data analysis and methodology	

Date of Submission: 28-07-2019
Date of Revision: 15-08-2019
Date of Online Publishing: 25-09-2019
Date of Print: 30-09-2019