# OUTCOME OF DECOMPRESSIVE CRANIECTOMY (DC) FOR SEVERE TRAUMATIC BRAIN INJURY (STBI) IN ADULTS

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### ABSTRACT

**Objective:** To evaluate the outcomes of decompressive craniectomy (DC) in adults with severe traumatic brain injury (STBI).

Study Design: Observational cross-sectional.

Place and Duration of Study: Neurosurgical unit CMH Rawalpindi from July, 2011 to June 2014.

*Material and Methods:* Total of 39 patients who underwent DC for STBI were included in the study. Patients of both sexes and of age range 20 – 48 (32.03 ± 8.01) years were included in the study. The DC was performed within 24 and after 24 hours. Parameters recorded were mortality, neurological outcome / complications like brain herniation, wound dehiscence, cerebrospinal fluid (CSF) leak, contusion expansion, sinking flap syndrome, subdural hygromas and hydrocephalus. Data was analyzed by using SPSS version 17 and descriptive statistics, frequency, rate and percentage was computed for presentation of qualitative outcomes.

**Results:** Favourable neurological outcome was seen in 21 patients (53.85%) where as 6 patients (15.38%) had moderate to severe disability and 3 patients (7.69%) were vegetative respectively. Patients operated within 24 hours and with Glasgow coma scale (GCS) range 6-8 had better outcome. . Overall 9 patients (23.08%) did not survive the injury and procedure.

*Conclusion:* As high mortality is associated with STBI, DC is an effective option to lower down the refractory intracranial hypertension with an acceptable surgical outcome.

Keywords: Decompressive craniectomy, Neurological outcome.

#### INTRODUCTION

STBI Worldwide is common а neurosurgical problem which can cause massive brain swelling leading to uncontrolled raised intracranial pressure (ICP), resulting in severe brain damage or even death. Therefore, reduction of raised ICP is an important factor in treatment of STBI<sup>1</sup>. Essentially STBI is a clinical condition having Glagcow coma scale (GCS) equal to or less than 82. Decompressive craniectomy (DC) is a neurosurgical procedure in which a portion of skull is removed along with durotomy so that swollen brain tissue can have normal cerebral perfusion and reduction in ICP3. The accepted pathophysiology of massive brain swelling after STBI is due to excitotoxic peaks, facilitating an influx of calcium into cell and mitochondrial metabolic disturbance leading to cell death. These events cause decrease in cerebral blood flow with

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inadequate oxygen delivery, activating the cascade of vicious cycle causing more and more brain swelling and rise in ICP<sup>4</sup>. There has been a renewed interest in DC over recent years, but its role remains controversial with more complications at extremes of age<sup>5</sup>. There are two standard forms of DC, the bilateral (bi-frontal) and unilateral craniectomy. These differ in terms of the part of the skull which is to be removed. Currently there is no consensus on the optimal extent of the craniectomy, although unilateral craniectomy is the more common, though having fewer complications<sup>6</sup>. The large calvarial defects make these patients vulnerable to a number of complications like sinking flap syndrome<sup>18</sup>. The aim of this study is to evaluate the outcomes and procedure related complications of DC after STBI in adult population.

#### MATERIAL AND METHODS

This observational cross-sectional study was conducted in neurosurgical department of CMH Rawalpindi, from July 2011 to June 2014. Patients of both sexes and of age range 20 - 48 (32.03 ± 8.01) years, who underwent DC for STBI with clinical and radiological evidence of

uncontrolled intracranial hypertension were included in the study. Patients with age more than 50 years and less than 18 years, penetrating head trauma, comminuted and compound fracture of the skull, GCS 3, fixed dilated pupils and hemodynamic instability were excluded. Total of 39 patients were selected for the study using stratified sampling. Informed written consent was obtained for each case from family. After resuscitation and emergency pre-op workup, patients were taken to operation theatre, laid supine, rolled towel placed beneath ipsilateral shoulder and head turned towards contra-lateral side, horizontal with the floor. Under aseptic conditions, midline marked, question mark incision started at zygoma, with posterior extent 2-3 cm posterior to standard trauma flap taking care of transverse sinus and torcula. Then the superior incision was taken parasaggitally 2-3 cm away from midline to widow's peak keeping in view the safety of superior saggital sinus (SSS). Myocutaneous flap reflected, five burr holes were made in the following locations: (1) temporal squamous bone just superior to posterior root of zygomatic arch, (2) keyhole area behind frontal end of zygomatic arch anteriorly, (3) 3 cm above transverse sinus and 3 cm lateral to inion, (4) parietal parasaggital and (5) frontal parasagittal area. Bone flap was fashioned, elevated with care and removed for storage in abdominal wall (having no facility of bone graft refrigeration). Added temporal decompression was done and bone edges were waxed. Radial durotomy done to relieve pressure on the traumatized swollen brain, subdural clots removed and dura loosely laid over swollen brain. Surgicel was used as on lay over the entire area of exposed brain, followed by muscle and aponeurotic layer overlay without suturing. Haemostasis was secured and then closure of scalp in two layers with sub galeal drain in place. Bone flap was placed abdominal under strict asepsis in the subcutaneous pouch. Postoperative patient were nursed in ICU with standard monitoring.All those patients who survived (DC) underwent cranioplasty that was timed ranging from 44-89 (66.2 ± 11.50) days using

native bone flap preserved in the abdominal wall.

Data was analyzed by using SPSS version 17. The variables recorded on proforma sheets were age, sex, etiology, duration of onset, timing of DC, Pre-op GCS, pupillary reaction, neurological outcome, wound dehiscence, brain herniation, sinking flap syndrome, CSF leak, expansion of the contusion, hydrocephalus, Glasgow outcome scale (GOS) and death. Relevant descriptive statistics, frequency, rate and percentage was computed for presentation of qualitative outcomes complications. Quantitative variables like age, time etc. was presented as mean ± standard deviation.

## RESULTS

Total of 39 patients with STBI underwent decompressive hemi-craniectomy at our centre. Mean age of the cohort was 32.03 years (SD ± 8.01), age range 20-48 years however only three patients (7.69%) were female. The DC was performed within 24 hours in 28 (71.79%) and >24 hrs in 11 (28.20%) who reported after 24 hours. Computed tomography (CT) scan confirmed the presence of complex acute subdural hematoma (ASDH) in 33 (84.62%) and global diffuse edema with evident contusions in 6 patients (15.38%). Clinically pre-op GCS was 6-8 in twenty six (66.67%) and 4-5 in thirteen (33.33%) patients respectively. The early outcome was assessed in 44-89 days with mean  $66.2 \pm 11.50$  days of the injury in patients who survived the injury. Survivors subsequently underwent autologous cranioplasty. Outcome was good in 53.85% (GOS-5), fair with moderate to severe disability (GOS 3-4) in 15.38% and vegetative (GOS-2) in 7.69%. Sinking flap syndrome was found in 17.94% cases whereas external brain herniation, CSF leak, hydrocephalus, expansion of contusion, subdural hygromas were found in 5.1%, 2.56%, 5.1%, 10.3%, 10.3% cases respectively. Overall mortality rate was 23.08% (table-2).

## DISCUSSION

Worldwide STBI is the leading cause of mortality and morbidity in trauma patients<sup>7</sup>. Global diffuse brain edema and multiple cerebral contusions are the two most common causes of death after STBI<sup>8</sup>. The DC is an established tool in the neurosurgical

The main objective of DC is to lower down the raised ICP and its sequale that in turn leads

Variable		N=39	Percentage
Gender	male	36	92.31%
	female	3	7.69%
Etiology	ASDH	33	84.61%
	Contusions with global edema	6	15.38%
Timing of DC	<24hrs	28	71.79%
	>24hrs	11	28.21%
Pre-op GCS	6-8	26	66.67%
	4-5	13	33.33%
Table-2: Outcome and complications after DC (n=39).			
Outcome/Complications		N=39	Percentage
Improved neurological outcome (GOS -5)		21	53.85%
Moderate to Severe disability(GOS 3-4)		6	15.38%
Vegetative(GOS -2)		3	7.69%
Wound dehiscence		1	2.56%
External Brain herniation		2	5.1%
Sunken flap syndrome		7	17.94%
CSF leak		1	2.56%
Hydrocephalous		2	5.1%
Expansion of contusion		4	10.3%
Subdural hygroma		4	10.3%
Mortality		9	23.08%

Table-1: Demographics and other variables of DC patients (n=39).

armamentarium for managing refractory intracranial hypertension for more than hundred years. Harvey Cushing described DC in 1905 as a palliative method against brain herniation in inoperable brain tumours9. There is increasing interest for DC in the management of raised ICP from STBI. The DC was initially considered as a second line treatment procedure where conservative treatment failed but recently DC is being encouraged as an early procedure when it is clinically indicated and in settings with resource constraints of ICP monitoring<sup>8</sup>. This shift may be attributed to better and frequent use of computed tomography (CT), as well as earlier and more aggressive surgical and intensive care therapy, however, morbidity and mortality remain high<sup>10</sup>. The available data on DC to treat elevated ICP and brain shifts associated with STBI shows that the mortality has decreased 20-30% in the last two decades in the patients undergoing DC<sup>9,12,13</sup>.

to improved outcome<sup>14</sup>. Massive brain swelling ensues within 2-3 hours after STBI. Second surge of brain swelling occurs within 2-5 days due to blood cell break down products and activated inflammatory cascade, therefore surgical intervention should be done as early as possible to prevent secondary brain injury<sup>15</sup>. The timing of DC is an important factor, especially for STBI with complex ASDH. Our study also confirmed that diffuse brain edema can coexist with ASDH as mentioned in previous studies<sup>16,17,18</sup>. If DC is done within 48 hours of the time of injury, the outcome is favorable<sup>19</sup>. Our centre is not equipped with ICP monitors so radiological and clinical parameters are our best guide for decision on DC. The raised ICP is one of the major deteriorating factors in patients with STBI though it may be in the form of thin layer ASDH, therefore prevention of intracranial hypertension by decompression plays a key role with respect to secondary brain injury<sup>12,16,20</sup>. In our study, patients who underwent DC within 24 hours had favourable outcome as consistent with QiuW et al<sup>19</sup>. Although a straightforward comparison among studies relating to DC in STBI is not possible due to the different they considered, variables they all demonstrated a favourable outcome on patient's survival after DC, ranging from 16% to 69%12,13,17,21. In this study 53.84% patients had favourable outcome and 23.07% had unfavourable outcome consistant with QiuW et al 56.8% and 16.2% respectively<sup>19</sup>. Chibbaro S et al reported favourable outcome in 89 (67%, GOS 4 or 5), and was not favorable in 25 (19%, GOS 2 and 3), and 19 patients (14%) died comparable with our results 25. The mortality rate in our study after DC was 23% as compared to QiuW etal who reported mortality of 27% after unilateral DC19. Authors have reported that there is a correlation between pre-op GCS and DC outcome<sup>13,17,22</sup>. Outcome was favorable in 73.07% patients having pre-op GCS 6-8 and unfavourable in 84.61% patients having GCS 4-5, comparable to Bunc G and colleagues<sup>10</sup>. Our experience also supports the report by Howard et al that pupillary reaction irrespective of the contributes towards the favourable size outcome of DC<sup>22</sup>. Additionally young age is a favourable prognostic factor for DC as reported by many authors similar to our study in which the patients are less than 50 years of age<sup>10,17,23,24</sup>. Associated injuries have negative prognostic impact in the development of secondary brain injury thereby influencing poor outcome of these patients<sup>22</sup>.

The DC is not free of complications and we found wound dehiscence/infection in 2.56%, hydrocephalus in 5.1%, CSF leak in 2.56%, expansion of contusion in 10.2%, external brain herniation in 5.1%, subdural hygromas in 10.2%, syndrome of the trephined in 17.94% in this study and similar complications have been reported in literature as well<sup>10,12,19,22,25</sup>. These complications were managed conservatively except for the patient with CSF leak through small wound dehiscence, but was resolved by simple suture placement. Syndrome of the trephined responded to cranioplasty. External cerebral herniation is more than 1.5 cm of herniated brain tissue through the craniectomy in the center of the skull defect. Craniectomies

larger than 12 cm as in our study less likely to cause effects of external cerebral herniation such as cortical vein compression and cortical laceration resulting in venous infarction of herniated brain tissue and cortical damage. Additionally external cerebral herniation disappeared in all these patients with time without any surgical intervention<sup>26</sup>.

The DC is becoming an increasingly



Figure: (A) Right complex ASDH (B) Tense dura (C) Massive swollen brain (D) Bone flap (E) Contusion expansion (F) Sinking flap (G) Left ASDH (H) Burr holes (I) Contused brain (J) Post DC (K) 3-D Calvarial defect (L) Subdural Hygroma.

preferred method for the treatment of massive brain swelling after STBI, at our neurosurgical unit since 2011. There are still many controversies regarding the use of DC in STBI, further studies are required so that we obtain additional valuable information for treatment protocol of patients with STBI. The limitations of our study included single centre experience, sample bias, lack of ICP monitoring, lost to regular follow up and lack of long-term followgu data. А prospective, multi-centre, randomized controlled trial with extended follow-up duration will be more helpful to fully investigate the prognostic factors and formulate an ideal treatment protocol for STBI.

## CONCLUSION

Worldwide STBI is the leading cause of mortality/morbidity in trauma individuals, however to date there is no standard method to address this issue of preventing secondary brain injury due to post-traumatic raised intracranial pressure. However DC is an effective option to lower the refractory intracranial hypertension with favourable surgical outcome, provided the patients are resuscitated promptly at the earliest, stabilized, transported properly by fastest means to the neurosurgical centre to receive intensive care support with ICP monitoring and prompt neurosurgical care.

#### CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

### REFERENCES

- 1. Kolias AG, Guilfoyle MR, Helmy A, Allanson J, Hutchinson PJ.
- Traumatic brain injury in adults. Pract Neurol 2013; (13): 228–35. 2. Kay A, Teasdale G. Head injury in the United Kingdom.World J Surg
- 2001; 25: 1210-20.
  Kolias AG, Peter J. Kirkpatrick, Hutchinson PJ. Decompressive craniectomy: past, present and future.Nature Reviews Neurology. 2013; (9): 405-15.
- Sahuquillo J, Arikan F. Decompressive craniectomy for the treatment of refratory high intracranial pressure in traumatic brain injury.Cochrane Database Syst Rev 2006; CD003983.
- Hutchinson PJ, Timofeev I, Kolias AG, Corteen EA, Czosnyka M. Decompressive craniectomy for traumatic brain injury: the jury is still out. British Journal of Neurosurgery 2011; 25(3): 441–2.
- Quinn T, Taylor J, Magarik J, Vought E, Kindy M. Decompressive craniectomy: technical note. Acta Neurologica Scandinavica.2011; 123(4): 239–44.
- Mathai KI, Sudumbrekar SM, Segupta SK, Rappai TJ. Decompressive craniectomy in traumatic brain injury - Rationale and practice. Indian Journal of Neurotrauma 2010; 1: 9-12.
- Das S, Alam MJ, Islam KMT, Elahi F, Mahmud E. Decompressive craniectomy in severe traumatic brain injury - a study of 20 cases. Bangladesh Med J 2014; 43 (2): 100-2.
- Cushing H. The establishment of cerebral hernia as adecompressive measure for inaccessible brain tumors. Surg Gynecol Obstet 1905; 1: 297-314.
- Bunc G, J Ravnik J, Klobucar R, Velnar T. Decompressive craniectomy following traumatic brain injury:our experience and review of literature. Acta Medica Mediterranea.2014; 30: 673.
- 11. Patel HC, Menon DK, Tebbs S. Specialist neurocritial care and outcome from head injury.Intens Care Med. 2002; 28: 547-53.
- 12. Albanese J, Leone M, Alliez JR, Kaya JM, Antonini F. Decompressive

craniectomy for severe traumatic brain injury: evaluation of the effects at one year. N Crit Care Med 2003; 31: 2535-8.

- 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.
- Shah M, Khan MM, Nabi A, Nauman MA, Aman A, Nasir M. The role of decompressive craniectomy in traumatic cerebral contusions; A prospective observational study. Pak J of neurol surg.2014; 18(2): 116-24.
- Timofeev I, Kirkpatrick PJ, Corteen E. Decompressive craniectomy in traumatic brain injury: outcome following protocol-driven therapy. Acta Neurochir Suppl 2006 ; 96 : 11-6.
- 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: 187–196.
- Ucar T, Akyuz M, Kazan S, Tuncer R. Role of decompressive surgery in the management of severe head injuries: prognostic factors and patient selection. J Neurotrauma. 2005; 22:1311–8.
- Skoglund TS, Eriksson-Ritzen C, Jensen C, Rydenhag B. Aspects on decompressive craniectomy in patients with traumatic head injuries. J Neurotrauma 2006; 23: 1502–9.
- QiuW, Guo C, Shen H, Chen K, Wen L, Huang H. Effects of unilateral decompressive craniectomy on patients with unilateral acute post-traumatic brain swelling after severe traumatic brain injury. Crit Care 2009; 13(6):185.
- Pompucci A, De Bonis P, Pettorini B, Petrella G, Di Chirico A, Anile C. Decompressive craniectomy for traumatic brain injury: patient age and outcome. J Neurotrauma. 2007; 24:1182–8.
- Whitfield PC, Patel H, Hutchinson PJA, Czosnyka M, Parry D. Bifrontal decompressive craniectomy in the management of posttraumatic intracranial hypertension.Brit J of Neurosurg 2001; 15: 500-7.
- Howard JL, Cipolle MD, Anderson M, Sabella V, Shollenberger D. Outcome after decompressivecraniectomy for the treatment of severe traumatic braininjury. J Trauma 2008; 65: 380-6.
- Schneider GH, Bardt T, Lanksch WR, Unterberg A. Decompressive craniectomy following traumatic brain injury: ICP, CPP and neurological outcome. Acta Neurochir 2002; 81: 77-9.
- Ziai WC, Port JD, Cowan JA, Garonzik IM, BhardwajA. Decompressive cranietomy for intractable cerebral edema: experience of a single center. J Neurosurg Anesth 2003; 15: 25-32.
- Chibbaro S, Di Rocco F, Mirone G, Fricia M, Makiese O, Di Emidio P. Decompressive craniectomy and early cranioplasty for the management of severe head injury: A prospective multicenter study on 147 patients. World Neurosurg 2011; 75: 558–62.
- Ban SP, Son Y, Yang H, Chung YS, Lee SH, Han DH. Analysis of complications following decompressive craniectomy for traumatic brain injury J Korean Neurosurg Soc. 2010; 48(3):244–50.

#### **AUTHORS CONTRIBUTION**

Shahzad Ahmed Qasmi, conception/design, Abdul Ghaffar, analysis, Zahid Hussain, interpretation of data, Maqsood Akram, analysis.