Early Decompressive Craniectomy after Severe Traumatic Brain Injury

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Abstract: Background: The role of decompressive craniectomy in the management of severe traumatic brain injury is a subject of debate in the present era. Increased intracranial pressure (ICP) can cause brain ischemia and compromised brain oxygen after severe traumatic brain injury (TBI). Decompressive craniectomy (DC) is applied to treat post-traumatic intracranial hypertension (IC H). The purpose of this study is to prove the efficacy of early decompressive craniectomy (DC) in the treatment of elevated ICP and good clinical outcome after severe head injury. **Methods**: A retrospective study was conducted of 21 patients who underwent cranial decompression after severe head injury. Baseline demographics, neurological examination results, and underlying pathology were reviewed. Clinical outcome was assessed by length of intensive care unit stay, length of hospital stay, and survival at discharge. Control of intracranial hypertension was assessed by intracranial pressure (ICP) monitoring and an ICP therapeutic intensity index. Radiographic outcomes were assessed by comparing preoperative and postoperative CT scans for: 1) postoperative volume of cerebral expansion and brain edema; 2) presence of uncal herniation; 3) intracerebral hemorrhage; 4) Subdural collections, and 5) brain ischemia. The patient's age, six, trauma etiology, GCS and focal neurological deficit at admittance, and associated other injuries were reviewed as well. According to GCS results, the patients were grouped as severe TBI (GCS=3–8), moderate TBI (GCS=9–13) or mild TBI (GCS=14 and 15). Only Severe TBI group are reviewed in this study while other groups are excluded**. Results:** DC was performed on average 2- 12 hrs after admission. DC was found to immediately reduce ICP about 6-11 mm Hg from 20, 27mmhg to 14-16mmHg). TIL, continued to improve within the postsurgical monitoring period. The duration and severity of CIB were significantly reduced as an effect of DC. The overall mortality rate in the patients was lower than predicted at the time of admission. **Conclusion**: These results suggest that a DC for increased ICP can reduce the CIB of the brain after severe TBI. We suggest that DC be considered early in a patient's clinical course, particularly when the TIL and ICP are increased. Also large decompressive craniectomy (frontotemproparietal) combined with enlargement of the dura by duraplasty seems to have the most favorable results beside it decrease the risk of several secondary surgical complications.
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Abbreviations: CBF= cerebral blood flow. CPP= cerebral perfusion pressure. DC= decompressive craniectomy. ICP= intracranial pressure. TBI= traumatic brain injury. TIL= therapeutic intensity level. CSF= cerebrospinal fluid. ICH= Intracranial hypertension. GCS= Glasgow come scale. GOS = Glasgow Outcome Scale

1. Introduction

Traumatic brain injury (TBI) is a leading cause of death and disability among people of all ages. Damage following TBI is the result of primary and secondary injuries on the brain. Primary injury is largely due to shearing forces between brain tissue of different densities, specifically between skull and dura mater, dura mater and gray matter, white matter and gray matter. (1) Secondary physiological injury, however, is also responsible for a large amount of morbidity and death. Secondary injury can result from impaired cerebral blood flow, regional edema, hemorrhage, elevated ICP and therefore reduced CPP, dysfunction of ion pumps, excessive release of neurotransmitters, cascade of cellular destruction via reactive oxygen species, proteolysis, and inflammation. (2) Intracranial hypertension, particularly when it does not respond to maximal medical management, increases the risk of mortality and poor outcome (3). Marmarou and his colleagues (4) observed a significant association between poor outcome and the number of hourly intracranial pressure (ICP) values that were > 20 mm Hg. Consequently, ICP control specifically treatment to maintain ICP <= 20 mm Hg, to optimize cerebral perfusion pressure (CPP), and to prevent secondary cerebral injury. Today, many neurointensive care units also use multimodality monitoring, eg, brain oxygen (PbtO2), continuous electroencephalogram, or microdialysis, to help prevent secondary brain injury (5).

Decompressive craniectomy is performed in the treatment of uncontrollable cerebral edema from trauma and other conditions.(6) yet much study data

suggest the procedure is beneficial for some conditions, particularly head trauma.(7)

2. Material and Methods

Between March 2010 and March 2012, the study was performed of 21 patients with severe traumatic brain injury after motor care accident who underwent decompressive craniectomy operation were reviewed retrospectively from our database General Clinical Management.

Each patient was fully resuscitated according to advanced trauma life support guidelines, intubated, and mechanically ventilated with the head of bed initially elevated approximately 30° to 45°. FIO_2 and minute ventilation were adjusted to maintain SaO_2 > 93%, PaO_2 of 95 to 100 mm Hg, and $PaCO_2$ of 34 to 38 mm Hg. Volume resuscitation was achieved with 0.9% normal saline and albumin for a target central venous pressure of 6 to 10 cm H_2O. Therapeutic targets were adjusted to avoid ICP > 20 mm Hg and CPP <= 60 mm Hg. After adequate fluid resuscitation, phenylephrine (10–100 µg/min) was administered when CPP was <= 60 mm Hg and ICP was normal. A standard stair step approach was used to treat intracranial hypertension. Initial management consisted of head of bed elevation, sedation (lorazepam), analgesia (fentanyl), neuromuscular blockade (vecuronium),and osmotherapy (mannitol) was started, provided that serum osmolarity was <= 320 mosm/L and serum sodium was <= 145 mmol/L. Optimized hyperventilation also used.

Different methods of decompressive craniectomy have been developed for, decompression of the brain at risk for the sequelae of traumatically elevated ICP. These include subtemporal decompressive craniectomy, (8) fronto- or temporoparietal decompressive craniectomy, (9) large fronto-temporoparietal decompressive craniectomy, hemisphere craniectomy, and bifrontal decompressive craniectomy. (10)

Two main methods for duroplasty: the dura is enlarged with the patient's own tissue, such as temporal fascia, temporal muscle, or galea aponeurotica, or this is performed with artificial dura substitute. (9) In our study the unilateral frontotemproparietal or bifrontal decompressive craniectomy with artificial duroplasty were used.

In general, DC done in patients with shift of midline more than 5 mm on CT brain or when there is suspicion of increase ICP as in patient with multiple brain contusions with progressing brain edema and increase ICP. Medically refractory elevated ICP was defined as an ICP of > 20 mm Hg for > 15 minutes in a 1-hour period. Patients under went unilateral DC with preservation of bone flab on bone bank not more than 4 months in all patients, the dura mater was opened as part of the operation, and the dural defect was covered with artificial dural graft. A subgaleal drain was placed. The same intensive care management protocol was followed after DC, and therapy was tailored to achieve the same ICP and CPP targets.

The patients are evaluated clinically and radiologically. Clinically: by Glasgow outcome Scale, and ICU monitoring. All patients were improved within 12-72 hours post craniectomy but the long stay in the ICU within 4 to 35 days postoperative due to other body injuries or chest problems, except one patient died because of bleeding disorders. Radiological: based on the initial head CT scan followed by repeated CT brain post DC operation.

The TIL modified from was calculated every 6-12 hours for 2-4 days after DC. The number of calculated TILs in some patients therefore depended on the interval between admission and DC. There are 6 medical management categories including hyperventilation, pressor administration, hyperosmolar therapy, ventricular drainage, paralysis, and sedation.

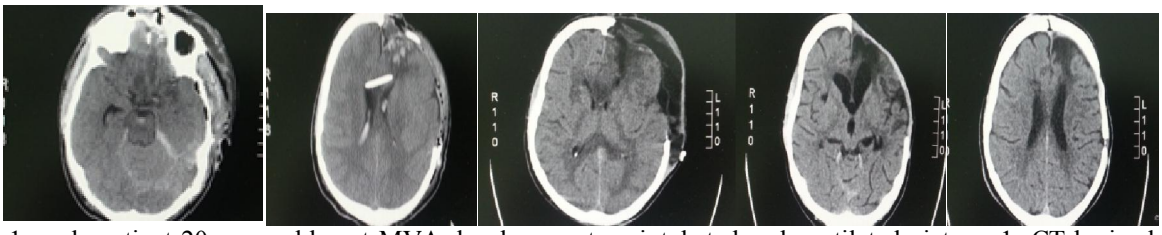

Fig. 1; male patient 20 years old post MVA deeply comatose intubated and ventilated picture: 1- CT brain shows intracerebral contusions SAH and depressed skull fracture, picture: 2- shows left frontotemproparietal craniectomy with EVD, brain contusions and IVH, picture: 3-CT brain 2 week later shows collection of left subdural hygroma, picture: 4 -4 weeks later: shows CT brain with resolved subdural hygroma, picture: 5- CT brain after 3 months shows replacement of bone flab

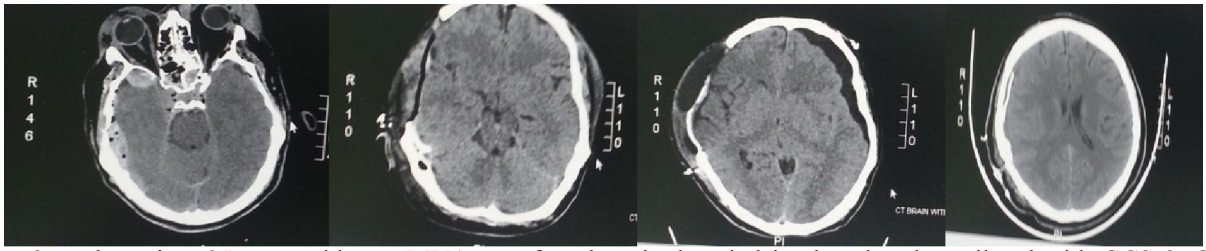

Fig. 2: male patient 35 years old post MVA transferred to the hospital intubated and ventilated with GCS 8, CT brain picture1: shows right extradural hematoma pneumocephlous fracture base and SAH. Picture 2: shows post right frontotemproparietal craniectomy with evacuation of hematomas and release of pressure within3hours. Picture 3: CT brain after 2 weeks shows collection of subdural hygroma bilaterally picture 4: CT brain after 3 months shows bone flab replacement with resolved hygroma

3. Results

During the specified 2-years period 21 patients met the inclusion criteria for this study underwent decompressive craniectomy. The age ranged from 9-57 years old. Individual patient clinical and radiological characteristics, outcome, and expected outcome are listed in the Table 1. All patients had an admission Glasgow Coma Scale <= 8. All patient were after motor car accident, all patient underwent same medical treatment pre and post-operative, all patient post-operative sent to rehabilitation, therapeutic intensity index in all patient improved post-operative, all CT brain after rehab were normal except for brain malicia or subdural collection which resolved after time, 90 percent of patient survive without deficits 10 percent of patient died due to bleeding disorder. The TIL reflects the amount of medical therapy (e.g, hyperventilation, osmotherapy, sedatives, muscle blockades, and pressers) delivered to the patient to control ICP. Therapeutic values were calculated before and after the DC. A reduction in TIL was observed after DC.

Table 1: Patients master table

	Age	Sex	Phy. Exam.	CT finding preoperative	Time and type of operation from admission	Duration of ICU stay	Duration of hospital stay	complications	Time of bone flab replacement	Outcome and complications
1	39	M	Facial laceration, raccoon eye bilateral, sluggish reaction, CSF leake pupil GCS 8	Brain contusions, right SDH, 7mm midline shift, tentorial herniation, basal skull fractures, with facial fracture	5 hours, bifrontal craniectomy	35 days	3 months tell transfer to rehab	Right side weakness G3	40 days	After I year intact neurologicall y no deficits
2	41	M	Transferred after 6 hours from trauma, GCS 6 intubated	Frontal contusions brain edema, pupil no reaction to light	4 hours bifrontal craniectomy	20 days	30days tell transfer to rehab	Vision problems improved after maxillofacial reconstruction	2 months	After 12months intact neurologicall y no deficits
3	57	M	Deeply comatose sedated intubated, bilateral dilated pupil, GCS4	Diffuse axonal injury, Left brain contusions	3hour left frontotemproparietal craniectomy with EVD	4 days				Died, Blood disese
4	21	M	Intubated, sedated, GCS 5, dilated left pupil	Left frontal contusions SDH, SAH, depressed skull fracture	3hours Left frontotemproparietal craniectomy with EVD	10 days	2 months tell transfer to rehab	non	35days	After 16 months intact neurologicall y no deficits
5	9	F	Intubated and sedated GCS 5, pupil sluggish reaction bilateral	Brain contusion, right SDH, pneumocephalous basal skull fracture	3h. right frontotemproparietal craniectomy with EVD	30 DAYS	45 DAYS tell transfer to rehab	NON	95 DAYS	After13 months intact neurologicall y no deficits
6	33	M	Intubated, sedated pupil bilateral sluggish, GCS 8	Brain contusions, brain edema right SDH	4h. right frontotemproparietal craniectomy with EVD	12 days	33days tell transfer to rehab	non	60days	After 12 months intact neurologicall y no deficits
7	35	M	Intubated,sedated , bilateral raccoon eye, GCS8	right extradural hematoma pneumocephlous fracture base and	6h. right frontotemproparietal craniectomy with evacuation of	7 days	28days tell transfer to rehab	Bilateral SDH and resolved after 2 months	3 months	After 12 months intact neurologicall y no deficits

				SAH. Picture	hematomas within 3 hours.					
8	15	M	Intubated, sedated, GCS 6, pupil, sluggish	Brain contusions, Brain edema, shift of midline to the left side about 5mm	right frontotemproparietal craniectomy	12 days	30days tell transfer to rehab	non	2 months	After 12 months intact neurologicall y no deficits
9	41	F	Intubated, sedated, GCS 8	Left SDH; Pneumocephalous, fracture base	Left frontotemproparietal craniectomy	8 days	28days tell transfer to rehab	non	28 days	After 12 months intact neurologicall y no deficits
1o	22	M	Intubated, sedated,GCS 8,pupil sluggish	brain contusions with effacement of right lateral ventricle	right frontotemproparietal craniectomy	12 days	40days tell transfer to rehab	non	38days	After 12 months intact neurologicall y no deficits
11	25	f	Intubated, sedated, GCS 8,pupil sluggish	Scattered brain contusions with shift of midline to left	right frontotemproparietal craniectomy	18	40days tell transfer to rehab	Left side weakness	35 days	After 12 months left side weaknes S G 4 little improvement
12	49	M	Intubated, sedated, GCS 8, pupil sluggish	Right frontotemproparieta l brain contusions, SAH with shift of midline to left	right frontotemproparietal craniectomy	21	35days tell transfer to rehab	NON	40 DAYS	After 10 months intact neurologicall y no deficits
13	14	M	Intubated, sedated, GCS 8, pupil sluggish	Left brain contusion,brain oedema and frontoparietal subdural hematoma	leftfrontotemproparieta l craniectomy	35 days	60 days tell transfer to rehab	Right side weakness	45 days	After 12 months intact neurologicall y no deficits
14	33	M	Intubated, sedated, GCS 8, pupil sluggish	Left brain contusion, brain oedema	leftfrontotemproparieta l craniectomy	33 days	41days tell transfer to rehab	non	30	After 14 months intact neurologicall y no deficits
15	18	M	Intubated, sedated,GCS 8, pupil sluggish	Brain contusion, right SDH, basal skull fracture	Right frontotemproparietal craniectomy	18 days	29days tell transfer to rehab	non	21 days	After 12 months intact neurologicall y no deficits
16	22	F	Intubated, sedated, GCS 8	Left brain contusion, brain oedema	leftfrontotemproparieta l craniectomy	20 days	32days tell transfer to rehab	non	24 days	After 12 right side weakness G4
17	44	M	Intubated, sedated, GCS 8	Left brain contusion, brain oedema	leftfrontotemproparieta l craniectomy	22 days	32days tell transfer to rehab	non	30 days	After 9 months intact neurologicall y no deficits
18	35		Deeply comatose sedated intubated, bilateral dilated pupil,	Diffuse axonal injury Left and right brain contusions with shift to midline to the left	Right frontotemproparietal craniectomy	15 days	44days tell transfer to rehab	non	30 days	After 12 months intact neurologicall y no deficits
19	44	M	Intubated, sedated, GCS 8, pupil sluggish	Left brain contusion, brain oedema, left SDH	leftfrontotemproparieta l craniectomy	10 days	36days tell transfer to rehab	NON	21 DAYS	After 12 months intact neurologicall y no deficits
20	33	F	Deeply comatose sedated intubated, bilateral dilated pupil,	Diffuse axonal injury; Left and right brain contusions with shift to midline to the left	Right frontotemproparietal craniectomy	19 days	50days tell transfer to rehab	Right hemiparesis	40 days	After 12 months intact neurologicall y no deficits
21	22	F	Deeply comatose sedated intubated, bilateral dilated pupil	Diffuse axonal injury; Left SDH with shift to midline to the right	leftfrontotemproparieta l craniectomy	17 days	35days tell transfer to rehab	Right hemiparesis	22 days	After 12 months right hemiparesis G4

4. Discussion

Intracranial hypertension that does not respond to maximal medical management occurs in 10 to 15% of patients with severe head injury and contributes to increased morbidity and mortality rates. (12)

Research on severe head injury has revealed no easy pharmaceutical therapy for severe head injury and cerebral swelling**.** (13)

A negative attitude toward decompressive craniectomy may stem from the results of earlier outcome studies of prophylactic decompressive

craniectomy, which indicated unacceptably high morbidity and mortality rates. (14)

The effectiveness of decompressive craniectomy in improving outcome following severe head injury is challenged by the paucity of any strong scientific evidence.(15) The effectiveness of decompressive craniectomy in reducing ICP has been proven in all recently reported clinical investigations.(16) including improvement in a variety of physiological parameters, like ICP, compliance, ICP indexes such as cerebrospinal compensatory reserve and cerebrovascular reactivity, brain oxygen, and metabolic parameters measured by cerebral microdialysis, (18,19,20) These physiological improvements are often greater in those patients who subsequently have a favorable outcome. Our findings are consistent with and extend these observations. Our results suggest that DC can reduce TIL and the CIB of the brain. These findings imply that DC should be considered early in a patient's course, particularly when the TIL is elevated.

More recent data have revealed survival rates from 60 to 95%, with two thirds of those survivors having favorable outcomes. (17)

Recently, Ho and colleagues (18) studied 16 TBI patients who had DC and observed a significant improvement in PbtO2 and an 85% reduction in episodes of cerebral ischemia among patients who subsequently had a favorable outcome. This effect was not present in those who had an unfavorable outcome. In addition, abnormal brain neurochemistry, including glutamate, glycerol, and lactate measured with microdialysis, also improved when a favorable outcome occurred. Together, these data suggest that multimodality monitoring may help guide treatment and DC selection or, at the very least, indicate when management, even after DC, is futile.

Based on this retrospective study for most parameters of severe head injury assessment, individuals undergoing decompressive craniectomy has good outcome than those who did not. This is reflected by the fact that good GOS scores.

Prior to the performance of a decompressive craniectomy, several points should be considered to avoid complications.The following recommendations are based on the problems encountered during definitive management of our patients.

First: Surgical planning starts with early patient selection. The patients who received early decompressive craniectomy in this study have a good outcome than others. It is also important to remember that the concomitant presence of traumatic chest, abdominal, pelvic, or extremities injuries may affect the total outcome

Second: If the decision has been made to perform a decompressive craniectomy, every effort should be made to make the bone removal as large as possible. The recommendation is that the size of the decompression be, at a minimum, 14 cm (anteroposterior) by 12 cm (superoinferior) if the intention is to perform a frontotemproparietal craniotomy.

Conclusion

Early decompressive craniectomy, although an aggressive surgical procedure, is indicated for patients with severe TBIs who exhibit signs of increased ICP and declining neurological examination results. It produces an increase in the volumetric compensatory capacity of the intracranial cavity. Based on our experience and results, we believe that patients can safely undergo decompressive craniectomy with reasonable neurological recovery, despite poor initial findings on preoperative examination.

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