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Clinical Outcome of Decompressive Craniectomy Operation for the Management of Acute Traumatic Brain Injury

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Abstract

Objectives: To evaluate the efficacy of Decompressive Craniectomy (DC) on the postoperative clinical state of the patient to define a line of management of these cases. Take in considerations the surrounding circumstances of the patient till he reaches the ER in Egypt and the hospital resources. **Methods:** 200 patients suffering from acute traumatic brain injury causing DCL resulted from different pathologies causing increased ICP. In group A, patients with acute TBI were managed by surgical intervention in the form of Decompressive Craniectomy and in the control group B, patients were managed by medical treatment. The age range was from 8 to 65 with no history of associated medical disorders with exclusion criteria of non-traumatic causes of increased ICP. **Results:** Data collected showed: male to female ratio of 3:1. The most common mode of injury was falling from height. Mean time from injury to operative intervention was 4 hours. The leading initial symptoms were DCL. In group A the overall mortality was 60%, functional recovery rate was 30%, and left severely disabled or vegetative was 10%. 50% of the cases had associated injury. 20% suffered from post-operative complications. **Conclusion:** DC is the ideal solution for the management of acute TBI with persistent increased ICP when the other medical management fails, given an early intervention and taking into consideration other factors affecting surgical outcome.

Keywords

Decompressive Craniectomy "DC", Traumatic Brain Injury "TBI", Intracranial Pressure "ICP"

1. Introduction

There has been more acceptance and interest in the idea of Decompressive Cra-

niectomy for the past two decades in the management of severe traumatic brain injury and stroke [1] [2] [3]. While, we are living in a world with continuous increase of traumatic brain injury rates especially with the increase of using motor vehicles in low and middle-income countries [4]. Decompressive Craniectomy has been proven effective in the treatment of TBI in many retrospective studies [3] [5] [6] [7]. In a recent study, it was proven that a large wide craniectomy significantly improves the outcome of the operation and the impact on lowering the ICP compared to a limited craniectomy [5] [8]. Although there're many studies that favor decompressive craniectomy in the treatment of refractory post-traumatic intracranial hypertension, it remains controversial because of the lack of randomized and controlled trials [9].

We have to put into consideration many other factors that may affect the results as other systemic injuries, timing of trauma, timing of arrival at ER, timing of intervention, vital signs and post-operative complications.

The purpose of this study was to evaluate the efficacy of decompressive craniectomy in decreasing ICP by reviewing 200 patients with acute traumatic brain injury with different underlying brain pathologies. All the cases in Group A were treated with Decompressive craniectomy and duroplasty after failed medical treatment. Group B was randomized to no surgical intervention and was medically managed.

GOS was used in this study to determine the outcomes in all patients which defines:

1) Grade I as death,
2) Grade II as persistent vegetative state,
3) Grade III as severe disability (being conscious but disabled),
4) Grade IV as moderate disability (being disabled but independent), and
5) Grade V as good recovery.

-Poor outcome was defined as GOS 1 - 3 and good as GOS 4 and 5 [10].

Abbreviations used in this paper: CPP = cerebral perfusion pressure; ICP = intracranial pressure; TBI = traumatic brain injury; DC = decompressive craniectomy; GCS = Glasgow coma score; GOS = Glasgow outcome scale; DCL = disturbed conscious level.

2. Methods

The study was done prospectively in the Neurosurgery Department, Trauma Causality Unit, Cairo University on patient admitted in the period from June 2014 to June 2016, suffering from acute traumatic brain injury causing DCL resulting from different pathologies causing increased ICP.

2.1. Inclusion and Exclusion Criteria

2.1.1. The Inclusion Criteria for the Patients Participating in Both Groups of This Study

Ages from 8 - 65, there was no preference for males or females.

Acute traumatic brain injury with persistent increasing intracranial pressure

and deterioration of the conscious level; GCS from 3 - 9,

Failure of therapeutic measures as hyperventilation, deep sedation and diuretics,

Therapeutic measures are not suitable for the management (e.g. Acute subdural hematoma with rapid deterioration of conscious level),

No history of associated medical disorders as cardiac and pulmonary disorders.

2.1.2. Exclusion Criteria Were

Non-traumatic causes of increased ICP as vascular infarction,

Surgically unfit patients and brain stem dead patients on admission (absence of neurological reflexes as dilated fixed pupils which don't respond to light, absent oculovestibular reflexes, absent corneal reflex and motor response to supraorbital pressure. Apnea and GCS 3),

Devastating injury not expected to survive 24 hours,

Bleeding diathesis (genetic or acquired).

2.2. Clinical Material

This randomized controlled study had two groups, group A with a hundred patients suffering from acute TBI and they were managed surgically by DC after failure of other therapeutic measures as (hyperventilation, deep sedation, diuretics).

Group B with the same criteria but it was managed only with the therapeutic measures in the form of:

- Mechanical ventilation and hyperventilation in the intensive care unit with close observation.
- Deep sedation and paralysis mostly with morphine and lorazepam.
- Hyperosmolar therapy with Mannitol, hypertonic saline also has been used in some cases. Intravenous bolus of mannitol (0.25 g/kg to 1 g/kg body weight) administered first at the time of admission and (0.25 to 0.5 g/kg) of mannitol can be repeated every 2 to 6 hours with close monitoring of the arterial blood pressure and replacement of the fluids that has been lost because of mannitol-induced diuresis.
- Hypothermia.

PREHOSPITAL REPORT (Table 1):

All patients were treated with similar prehospital emergency treatment and routine brain CT scan was performed, Mannitol (0.5 - 1 mg/kg), Lasix (0.25 mg/kg), were given preoperatively regarding there is no hypotension.

All the surgically treated patients included in this study underwent a fronto-tempro-parietal DC either unilateral hemicraniectomy or bilateral wide bone flaps with augmentative duroplasty. We scheduled the cranioplasty for the survivors after 6 months from date of discharge from the hospital.

In group A, the following were assessed (postoperatively after follow up CT scan):

Table 1. Prehospital report.

Mechanism	How did injury occur? Presence of drugs or alcohol Deaths at scene Confounding issues
Injury	Primary survey Glasgow Coma Scale
Vital data	Heart rate Blood pressure Respiratory rate Oxygen saturation Temperature (if applicable)
Treatment	Airway (airway management) Breathing (oxygen administration, needle or tube thoracotomy) Circulation (intravenous access established and fluids administered) Disability—neurologic (spine precautions) Extra information (medications administered, procedures performed)

- Residual of any pathology—need for re-operation.
- Postoperative treatment: Based on the conditions of intracranial pressure after operation, Mannitol (0.5 mg/kg), Lasix (0.25 mg/kg), were given. Antibiotics, hemostatic, and neurotropic drugs were routinely used for all the patients.

In group B, all patients were put under close monitoring of systemic parameters, including ventilation, oxygenation, electrocardiogram, heart rate, blood pressure, temperature, blood glucose, and fluid intake and output. All patients received the above-mentioned therapeutic measures.

2.3. Follow Up and Outcome

*Group A:

- Postoperatively patients were admitted to the ICU and had at least one CT scan performed within 72 hours after operation. All patients were followed up after decompression with CT scan and neurological examination including GCS until discharge. The average stay of the patients was 24 days. After discharge frequently (weekly) for the first 6 months and scheduled for cranioplasty operation and then less frequent for another 6 months.

*Group B:

- All patients were admitted to the ICU with close observation of GCS until discharge, follow up CT scan was done after 24 hours from discharge and if there's any change in the GCS of the patient. The average stay of the patients at the hospital was 14 days. After discharge each patient scheduled for follow up date each week for the first month and the less frequent for the rest of the

year.

All patients in both groups were assessed by the Glasgow Coma Scale (GCS) and the outcome was graded using the Glasgow Outcome Score (GOS).

3. Results

- The data collected from 200 cases of traumatic brain injury patients in group A and B in this study was evaluated; the study included 100 patients in each group, group A had 70 males and 30 females and age range was between 8 and 65 years and the other group had 82 males and 18 females with the same age range.

Mechanism of injury:

- Mechanism of injury was road traffic accidents in 110 cases 55%, fall from height in 80 cases 40% and blunt head trauma in ten cases 5%.

Outcome:

- In group A, the overall mortality (Grade I) was 60%, the functional recovery rate (Grades IV and V) was 15% and 15% respectively, and (Grades II and III) was 5% both and in group B (Grade I) was 90% and (Grades II and III) was 10%.

Injury to operation:

- In group A, time from injury to operation was subdivided to <2 hrs, 2 - 4 hrs, 4 - 6 hrs and >6 hrs. after injury. Time was (<2 hrs) in (5 case—5%), (2 - 4 hrs) in (25 cases—25%), (4 - 6 hrs) in (40 cases—40%) and (>6 hrs) in (30 cases—30%) of cases. The mean time from injury to operative intervention was 4 hours and 54 minutes.

Clinical presentation:

- The leading initial symptoms were deterioration of level of consciousness for all patients.
- The associated injuries were present in (100 cases—50%) mainly orthopedic fractures of long bones in (50 cases—25%), fracture ribs in (20 cases—10%), hemothorax with fracture ribs in (20 cases—10%) and mild abdominal collection in (10 cases—5%). The 20 cases with hemothorax and fracture ribs died indicating more severe trauma to the patients.
- The main pathology was ASDH causing progressive increase in the ICP in 160 cases 80% and brain contusions in 40 cases 20%.

Complications:

- Post-operative complications in group A were; residual SDH was in 10 cases 10% and in group A, the incidence of wound infection was in 15 cases 15% and the cases responded to antibiotics and repeated dressing without the need for wound debridement.

Summary of Demographics for both patient groups (**Table 2**).

Summary of Clinical Characteristics for both patient groups (**Table 3**).

Predictors of outcome after decompressive craniectomy in patients with diffuse injury due to TBI (**Table 4**).

Table 2. Summary of demographics for both patient groups.

	Group A	Group B
Age (Mean)	40.96	40.2
Sex (M/F)	App. 2:1	App. 4:1
Race	Caucasian	Caucasian

Table 3. Summary of clinical characteristics for both patient groups.

	Group A	Group B
Mechanism of injury	Road traffic accident in 110 cases—fall from height in 80 cases—blunt head trauma in 10 cases	
Leading symptom	DCL	
Main pathology	ASDH in 160 cases (80%) of all cases and brain contusions in 40 cases (20%) of all patients	
Injury to operation	Less than 2 hrs. in 5 cases (5%) 2 - 4 hrs. in 25 cases (25%) 4 - 6 hrs. in 40 cases (30%) More than 6 hrs. in 30 cases (30%) * mean time from injury to operative intervention was 4 hours and 54 minutes.	-
Outcome	Grade I 60% Grade II, III 5% Grade IV, V 15%	Grade I 90% Grade II, III 10%
Associated injuries	Orthopedic injuries in 50 cases (25%) of all patients—fracture ribs in 20 cases (10%)—hemothorax in 20 cases (10%)—mild abdominal collection in 10 cases (5%)	
Post-operative complications	Residual hematoma in 10 cases (10%)—superficial wound infection in 15 cases (15%)	-

Table 4. Predictors of outcome after decompressive craniectomy in patients with diffuse injury due to TBI.

Variable	No. of patients	outcome
Timing of DC		
Early (>2 - 4 hr.)	30	Better—survival rate 60%
Late (4 - 6 hr.)	70	Worse—death rate 83%
Admission GOS		
Grade 1	60	Worse—mortality in 60%
Grade 2 - 3	5 - 5	Left severely disabled 5%
Grade 4 - 5	15 - 15	Better—mortality in 15%
Age		
>40 y.	35	Mortality in 90%
<40 y.	65	Mortality in 22%

*Mean age of survival was 30.7 and non-survival was 49.1.

4. Discussion

The concept of Decompressive Craniectomy is by no means novel; it can be defined as the removal of a large area of skull to increase the potential volume of the cranial cavity.

Our collected results demonstrated:

1) Drastic rapid decrease in ICP.
2) Sustained reduction of the intracranial hypertension.
3) Decompressive Craniectomy can save lives by controlling brain edema.
4) But also, could shift outcome to vegetative state and severe disability.

With close observation to the patients and series of follow up CT scans; brain edema was the lowest right after the operation and increased gradually over the first two days but always remained stable and significantly lower than pre-operative state. P.S. we did not have enough resources to provide bedside ICP monitoring to all patients. A decrease in both the number and duration of high ICP episodes was studied and discussed in some previous papers [11] [12].

Severe traumatic head injury might cause brain edema, intracranial hypertension, decrease the cerebral blood flow and ischemia which lead to more brain edema. Decompressive craniectomy with wide bone flap and duroplasty increase the space around the brain which gives the cerebral hemisphere more chance to expand than the normal limits to avoid herniation [13]. This results in improving the cerebral compliance, decrease ICP, increase CCP and rise in cerebral blood flow [3] [9] [12] [14] [15] [16] [17]. On the contrary there are some studies indicate that decompressive craniectomy might worsen brain edema and cause adverse outcomes [3] [12] [18] [19] [20].

Study Limitations:

The lack of ICP monitoring devices (e.g. Miniature strain-gauge or fiber-optic transducers) to evaluate the relief of elevated ICP after surgery which was judged upon only radio-logically.

The follow up period is short due to shortage of resources and the poor database and archival in the hospital and the failure to contact some patients after discharge. Many patients are lost to follow up, especially those who have to travel to this tertiary care facility. An extended period of follow up more than 48 months to 5 years at least is required for analysis of rehabilitation and return to work to be included in the final judgment during comparison of these surgical techniques.

An accurate hospital stay cost per patient could not be calculated as the Cairo University hospital is a teaching hospital affiliated to Cairo University and operates on governmental funds and provides care free of charge to its patients. Patients are not billed for neither ER nor operative costs.

In our study:

Focusing on group A, male: female ratio was almost (2:1), we found increasing age is a strong predictor of poor outcome, and the earlier the surgical decompression if indicated, the better the results. In our study the mortality rate within

2 hours from the trauma was 0%, while in (2 - 4 hrs.) was 40% of the cases in this group. In (4 - 6 hrs.) mortality rose to 83%. The mechanism of injury had no significant effect on mortality rate, fifty percent of patients had associated injuries mainly orthopedic. The rise in mortality may be attributed to the more severe injuries the patients were subjected to and the delay of arrival to the hospital (70% admitted after 4 hours from trauma) due to traffic jams and lack of large Neuro-trauma centers nearby for referral.

Intraoperative blood loss was not accurately measured however the amount of blood transfused is a rough indicator as most patients had two units of blood.

This study showed worse results compared to the international studies. However, the results in group A were significantly better than those observed in group B which had a 90% mortality rate and 10% of the patients were in a vegetative state or severely disabled, while in group A; death rate was 60% (60 patients), five patients remained in a vegetative state, five severely disabled and 30% of patients (30 patients) had favorable outcome.

Some factors may have contributed negatively to the results mainly long time to the OR as 70% of the patients were operated on after 4 hours from the trauma. Age is another contributing factor. But in comparison to the control group these results were much better regarding the survival rates and functional outcomes in those surviving.

In the study made by **Hamel, *et al.* 2014** [21]:

It was conducted on 60 patients after TBI; operated upon by Decompressive Craniectomy. Regarding the GOS; in that study Grade I (28.3%). Grade II, III (21.7%) and Grade IV, V (50%) however in our study Grade I (60%), Grade II & III (10%) and Grade IV (30%).

In the study Traumatic Acute Subdural Hematoma—Major Mortality Reduction in Comatose Patients Treated within Four Hours made by **Seelig, *et al.*, 1981** [22] there were 82 patients suffering from ASDH after TBI; all treated by Decompressive Craniectomy. In the first 4 hrs the mortality was 30% and 90% in those who had surgery after 4 hrs from injury as well as in our study it was 0% in the <2 hrs group and 40% in the 2 - 4 hrs group and 83% in the 4 - 6 hrs group.

Summary of characteristics in the clinical studies on decompressive craniectomy (**Table 5**).

5. Conclusions

Acute traumatic brain injury (TBI) is one of the most common and most morbid traumatic neurosurgical emergencies with high rates of mortalit and morbidity. Improving the outcome of patients with TBI as ASDH is a difficyult task.

Decompressive Craniectomy can decrease ICP and increase CPP. Apart from initial GCS and the pupillary status, time elapsed between trauma and treatment is the most important and can be improved by rapid transportation after trauma. Treatment in a Specialized Center is a major contributing factor. This means

Table 5. Summary of characteristics in the clinical studies on decompressive craniectomy.

Author and year	Study design	No. of patients	Timing of operation	Pre-op ICP (mmHg)	Post-op ICP (mmHg)	Surgical technique	Outcome
Yoo *et al.*, 1999	P	6	NR	31.7	3.5	Bifrontal craniectomy	Mean GOS 3.3; 1 patient died
Munch *et al.*, 2000	R	9	4.5 ± 3.8 (63.3% of cases) 56.2 ± 57 (36.7% of cases)	22.1 ± 11.1	19.7 ± 10.8	Hemicraniectomy	Good 41%; poor 59%
Taylor *et al.*, 2001	P	13	17.3 (range 605 - 27.5)	26.4 ± 7.9	17.4 ± 3.4	Bitemporal craniectomy with no duroplasty	Reduced risk of death, PVS and severe disability a 6 mos postop: 0.54 (95% CI 0.29 - 1.07)
Howard *et al.*, 2008	R	40	28.2 ± 9.6	35 ± 13.5	14.6 ± 8.7	Hemicraniectomy	Good 30%, survival 67% w/good outcome; mortality 55%
Kim *et al.*, 2009	R	28	9.6 ± 6.9	37.9 ± 15.9	9.2 ± 5.9	Hemicraniectomy (unilateral or bilateral)	Good 57.1%, poor 42.9%, mortality 21.4

that all comatose patients with an acute subdural hematoma should be treated in centers where neurosurgical facilities and appropriate monitoring are available. Other factors determining prognosis are: age, hematoma thickness, midline shift and associated injuries.

Regardless of the result of this study and the high mortality rate, we concluded that Decompressive Craniectomy operation is the ideal solution for the management of acute TBI with persistent increased ICP when the other medical management fails. Decompressive craniectomy operation has a novel role in lowering the increasing ICP provided that there is early surgical intervention and keeping in mind the relatively good Glasgow Coma Scale. Decompressive Craniectomy is considered for TBI patients the last chance to save the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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