



RESEARCH ARTICLE

RADIOLOGICAL INDICATORS OF POST OPERATIVE OUTCOME IN DECOMPRESSIVE
CRANIECTOMY FOR HEAD INJURY PATIENTS

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ARTICLE INFO

Article History:

Received 14th September, 2017
Received in revised form
26th October, 2017
Accepted 09th November, 2017
Published online 27th December, 2017

Key words:

Acute SDH,
Lobar contusions,
Decompressive craniectomy (DC),
Post-op improvement,
Post-op midline shift,
Postop basal cisterns

ABSTRACT

Introduction: Prediction of outcome following decompressive craniectomy has a huge impact on the productivity of the patient after surgery. The prediction tool should be easily accessible even in rural areas. In this study we analyze the impact of radiological indicators like post operative reduction in mid line shift and post operative opening of basal cisterns in CT brain on the final outcome of the patients.

Materials and Methods: It is a prospective analytical study conducted at Institute of Neurosurgery, Madras Medical College. A total of 136 patients who underwent decompression craniectomy for moderate and severe brain injury were included in the study. All patients had CT scan with evidence of Acute SDH, unilobar or multilobar contusions with diffuse cerebral edema, midline shift >5mm, and effacement of basal cisterns. CT brain was used to study the postoperative mid line shift and postoperative basal cisterns. The GCS and GOCS (Glasgow outcome score) at discharge were noted and outcomes were analyzed. Statistical analysis was performed by using MANOVA test. A statistically significant difference was indicated by a p-value of less than 0.05.

Results: The patients with Basal cisterns opening post decompressive craniectomy had statistically significant improvement in survival rates. Patients in whom there was reduction in the mid line shift post surgery did not have statistically significant improvement in the survival rates.

Conclusion: The opening of the basal cisterns can be considered as a predictor of favorable outcome following decompression craniectomy. Whereas the reduction in midline shift alone cannot be considered as a predictor of favorable post operative outcome. The availability of CT scans even in rural areas makes this an easily accessible prediction tool.

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Citation: Dr. Jothi Kumar Sethuraman, 2017. "Radiological indicators of post operative outcome in decompressive craniectomy for head injury patients", International Journal of Current Research, 9, (12), 62690-62695.

INTRODUCTION

Traumatic brain injury (TBI) affects up to 2% of the population per year and constitutes the major cause of death and severe disability among young people (Ross Bullock *et al.*, 2006). Road traffic injuries account for 2.1% of global mortality. The developing countries bear a large share of burden and account for about 85% of the deaths as a result of road traffic crashes. India accounts for about 10% of road accident fatalities worldwide (Arvind Kumar *et al.*, 2008). An injury to the brain may cause edema and produce swelling of brain. Pressure within the skull then increases as the brain has no room to expand; this excess pressure, known as high intracranial pressure, can cause further secondary brain injury. High intracranial pressure is the most frequent cause of death and disability in brain-injured patients. The management of increased intracranial pressure is common clinical scenario in neurosurgery. If high intracranial pressure (ICP) cannot be

controlled using general or first-line therapeutic measures, second-line treatments are initiated, one of these procedures is decompressive craniectomy (DC) and also performed while intracranial hematoma evacuation. DC involves the removal of a section of skull so that the brain has room to expand and the pressure decreases. Removal of a section of skull bone after a severe traumatic brain injury in patients with persistent raised intracranial pressure that has not responded to conventional medical treatments. Strategy for management of ICP by decompressive craniectomy is to remove the mechanical constraints imposed by the cranial vault. There is however still clinical uncertainty regarding the use of DC and a lack of consensus on the optimal management of traumatic brain injury (Juan Sahuquillo, 2009). Moreover, there is a need for modalities easily available even in rural areas to predict the post operative outcome in decompressive craniectomy for head injury patients. One such easily available tool is CT brain. The factors like post operative reduction in mid line shift and post operative opening of basal cisterns can be assessed using CT brain.

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Aim of study

The present study was undertaken to analyze the factors that affects the patient’s outcome in our setup, and to analyze the role of decompressive craniectomy and also the radiological indicators predicting the outcome. In this study we analyze the impact of factors like post operative reduction in mid line shift and post operative opening of basal cisterns on the final outcome of the patients.

MATERIALS AND METHODS

It is a prospective analytical study conducted at Institute of Neurosurgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. All Patients admitted in our hospital trauma ward with moderate to severe head injury who are undergoing primary decompressive craniectomy according to brain trauma foundation guidelines are included in this study. Categorization of head injury severity is based on Glasgow coma scale (GCS) score, GCS 9-12=moderate, GCS 3-8=severe.

Inclusion criteria

- Age 12-70 years and within first 48 hrs. from time of injury.
- Only traumatic causes.
- Post resuscitation GCS 4-12.
- CT scan with evidence of Acute SDH, unilobar or multilobar contusions with diffuse cerebral edema, midline shift >5mm, and effacement of basal cisterns.

Exclusion criteria

- Age less than 12 years and more than 70 years.
- Non-traumatic causes like infarct, spontaneous ICH or aneurysmal bleed.
- Post resuscitation GCS 3.
- Bilateral fixed and dilated pupils.
- Absent brain stem reflexes.
- Devastating injury not expected to survive for 24 hrs.
- Patients who are not willing for surgery or study.

All patients were initially seen in our emergency services. Hemodynamic stabilization and intubation was done where necessary and the post resuscitation GCS was noted. A CT scan was done as soon as possible. Patients with moderate to severe head injury requiring decompressive craniectomy considered for this trail, entry will be determined using the above inclusion and exclusion criteria after resuscitation, and data were entered in proforma. Consent for surgery and study was obtained from next of kin after detail explanation about the study. Approval for the study was obtained from the ethics committee. After the surgery patient treated in head injury ICU, then CT scan brain was done with in 24hr to 48 hrs. and compared to pre op CT scan. The postoperative GCS and GOCS (Glasgow outcome score) at discharge from the hospital were noted, primary and secondary outcomes were analyzed.

Primary outcome measures:

- Proportion (%) of favorable outcomes (GOCS4&5), unfavorable outcome (GOCS1,2&3)

Secondary outcome measures:

- Assessing post op GCS, adequacy of bone removal, reduction of mid line shift, basal cisterns compression, residual hematomas in post op CT scan and complications.

The variables analyzed on CT scan were the midline shift, status of basal cisterns, presence of residual hematomas and adequacy of bone removal. The midline shift was measured as the largest perpendicular distance between an imaginary reference line joining the frontal crest and internal occipital protuberance and the most shifted point of the septum pellucidum. Suprasellar and perimesencephalic cisterns were taken for basal cistern assessment. The extent of craniotomy and the details of duraplasty were noted. Adequacy of bone removal was measured by the margins of craniectomy in CT scan: anteriorly to the superior border of orbital roof; posteriorly to at least 2cm lateral to the external auditory meatus; medially to 2cm lateral to midline; and inferiorly to the floor of middle cranial fossa.

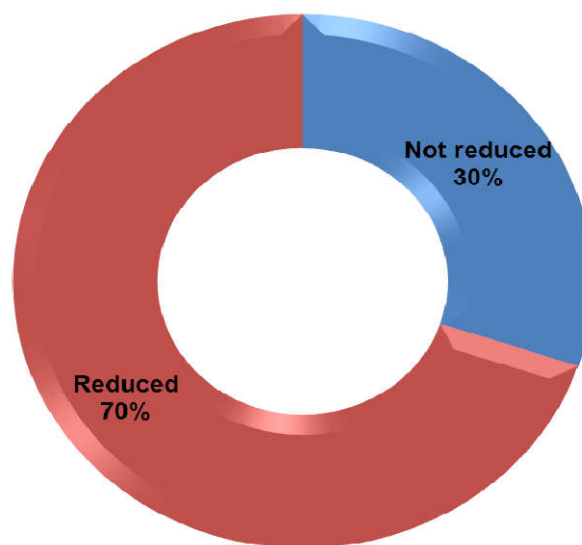
Statistical analysis

Statistical analysis was performed by using MANOVA test. Multivariate analysis of variance (MANOVA) is a statistical test procedure for comparing multivariate means of several groups. A statistically significant difference was indicated by a p-value of less than 0.05.

RESULTS

Post op Shift

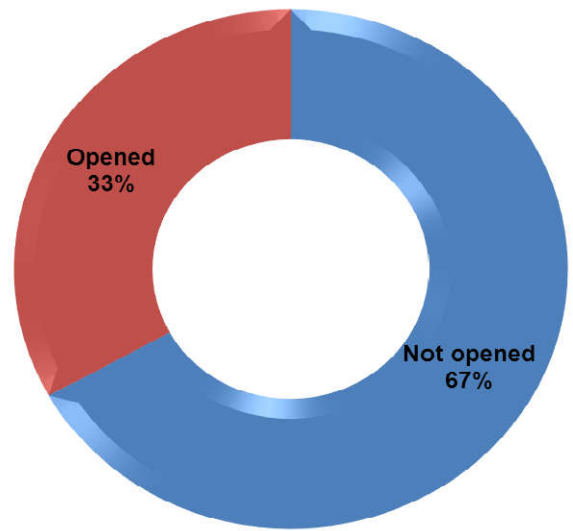
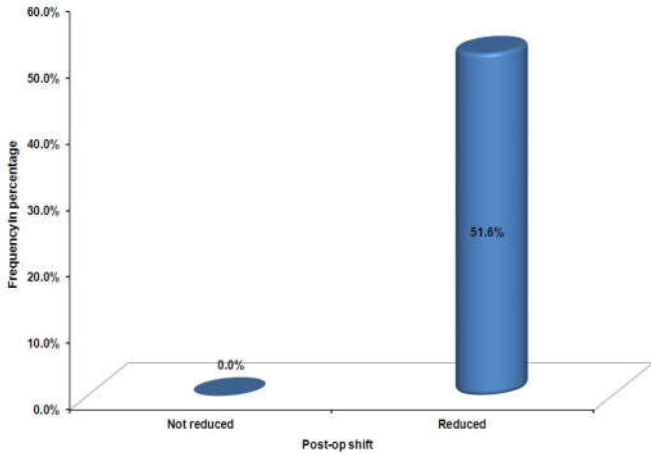
Post op Shift	No of patients
Not reduced	41
Reduced	95
Total	136



Post op shift vs outcome

Post-op shift	No of patients		Total
	Favourable Outcome	Unfavourable Outcome	
Not reduced	0	41	41
Reduced	49	46	95
Total	49	87	136

Percentage of favourable outcome

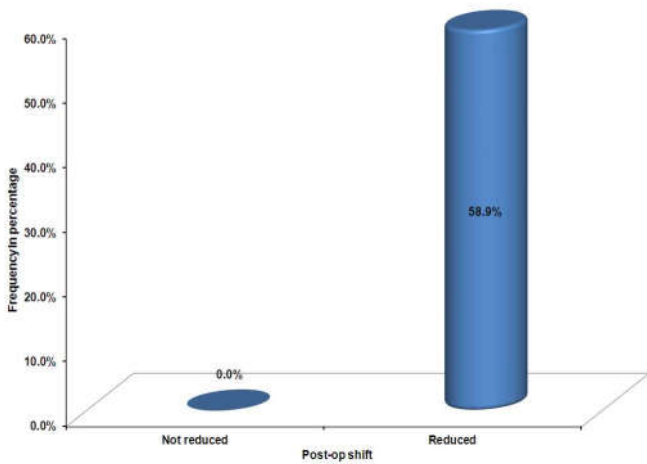


There is no statistical significance between post operative Shift and outcome (P-value – 0.062 > 0.05)

Post op shift vs Survival / Death

Post-op shift	No of patients		Total
	Survival	Death	
Not reduced	0	41	41
Reduced	56	39	95
Total	56	80	136

Percentage of survival



There is no statistical significance between post op Shift and survival (P-value – 0.166 > 0.05)

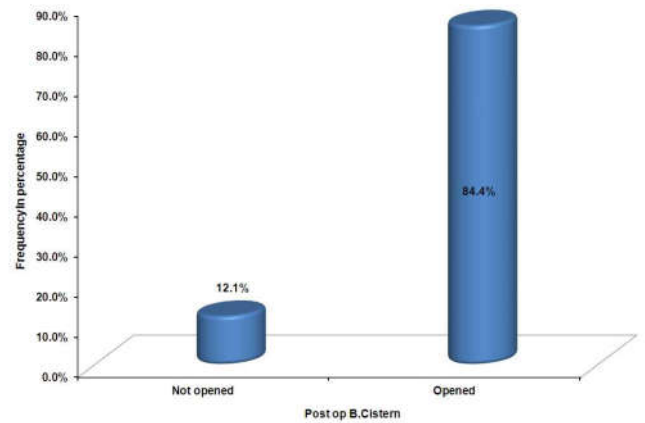
Post op B.Cisterns

B.Cisterns	No of patients
Not opened	91
Opened	45
Total	136

Post op B.CisternVs. Outcome

B.Cistern	No of patients		Total
	Favourable Outcome	Unfavourable Outcome	
Not opened	11	80	91
Opened	38	7	45
Total	49	87	136

Percentage of favourable outcome

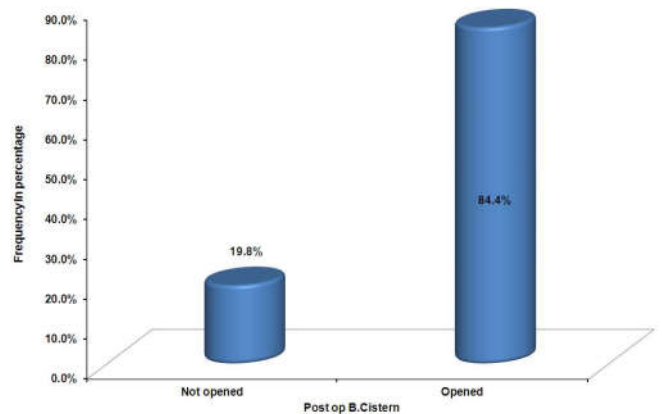


There is statistical significance between post op B.Cistern opening and outcome (P-value – 0.000 < 0.05) – MANOVA

Basalcistern vs Survival / Death

B.Cistern	No of patients		Total
	Survival	Death	
Not opened	18	73	91
opened	38	7	45
Total	56	80	136

Percentage of survival



There is statistical difference between post op Basal Cisternand survival (P-value – 0.000 < 0.05)

DISCUSSION

Pathophysiology of TBI

The tissue damage at the moment of brain trauma is the primary injury, whereas secondary mechanisms lead to brain edema. Disruption of the BBB is the most important prerequisite for edema formation (Abhishek Patro and Sureswar Mohanty, 2009). Both vasogenic and cytotoxic edema results in increased intracranial pressure and eventually decreases cerebral perfusion pressure. This is in line with the Monroe-Kellie hypothesis which states that 'the sum of the intracranial volumes of blood, brain, CSF and other components is constant and that an increase in any one of these must be offset by an equal decrease in another. Elevated ICP diminished cerebral perfusion and can lead to tissue ischemia. Ischemia in turn may lead to vasodilatation via auto regulatory mechanisms designed to restore cerebral perfusion. However, vasodilatation increases cerebral blood volume, which in turn then increases ICP, lower CPP and provokes further ischemia (Rosner and Rosner, 1995). After Traumatic brain injury, CBF auto regulation is impaired or absent in most patients. When pressure auto regulation is impaired or absent, ICP decreases and increases with change in cerebral perfusion pressure (CPP) (Enevoldsen and Jensen, 1978). Also, auto regulatory vasoconstriction seems to be more resistant compared with auto regulatory vasodilatation which indicates that patients are more sensitive to damage from low rather than high CPP.

Cascade of events in the pathophysiology of TBI

1. Initially there is direct tissue damage and impaired regulation of cerebral blood flow and metabolism.
2. Decreased CBF leads to accumulation of lactic acid due to anaerobic glycolysis, increased membrane permeability and consecutive edema formation.
3. Anaerobic glycolysis leads to depleted ATP stores and failure of energy dependent brain ion pumps.
4. Hypoxia leads to release of excitatory neurotransmitters like glutamate and aspartate.
5. These and other neurotransmitters activate the inotropic (NMDA) and metabotropic receptors
6. Consequently, Ca⁺⁺ and Na⁺ influx with K⁺ efflux
7. Ca⁺⁺ also activates lipid peroxidase, resulting in accumulation of free fatty acids and oxygen free radicals.
8. Prostaglandins and kinins initiate an inflammatory response.
9. Further activations of caspases, translocases and endonuclease initiate progressive structural changes of biological membranes and nucleosomal DNA.
10. There is a depression of metabolic activity of neural tissue resulting in suppressed neuronal activity.
11. Role of aquaporin-4 channels, decreased Mg⁺⁺ levels and vassopressor-2 receptor channels and erythropoietin in the pathophysiology of post traumatic brain edema is being studied.

Collectively these events lead to BBB disruption and degradation of cellular structures and ultimately necrotic or programmed cell death (Abhishek Patro and Sureswar Mohanty, 2009).

Historical background of De

Kocher was the first to propose decompressive craniectomy for patients with clinical symptoms of persistent elevated ICP in 1901 (Juan Sahuquillo, 2009; Abrar A Wani *et al.*, 2009; Kocher, 1901). Later, in 1905, Harvey Cushing made a detailed report on sub temporal and sub occipital decompression procedure to relieve high ICP in patients with inoperable brain tumors (Juan Sahuquillo, 2009; Abrar A Wani *et al.*, 2009; Kocher, 1901). A comprehensive historical review of the first few patients who underwent DC was published by Spiller and Frazier in 1906. Decompressive craniectomy in TBI was initially described by Miyazaki in 1966 and later popularized by Kjellberg and Prieto in 1971 (Kjellberg and Prieto, 1971).

The mechanism by which decompressive craniectomy provides reliefs in raised ICP are (Abrar A Wani *et al.*, 2009):

1. It lowers the ICP immediately.
2. It adds vector of expansion to cerebral hemispheres which relieves brain herniation.
3. Allows exploration of subdural space.
4. In addition, it provides quick tapering of medical treatment, in order to avoid potential complications.

Effects of decompressive craniectomy

1. Improving cerebral perfusion
2. Preventing ischemic damage
3. Avoiding mechanical compression of the brain (brain herniation)

The overall effects of decompressive craniectomies are to increase volume-buffering capacity of the cranial vault by allowing for centripetal herniation. The centripetal herniation in turn minimizes centrifugal compression of the brain stem structures (Clark Chen *et al.*, 2006). Decompressive craniectomy reduces intracranial pressure by 50%, duratomy further enhances intracranial pressure reduction by an additional 35% (Yoo *et al.*, 1999). The rationale for decompressive surgery is based on the Monroe-Kellie law. According to this theory intracranial volume should remain constant and volumetric compensations should be achieved by shifts in CSF, cerebral blood volume, or brain herniation. Removing a variable amount of bone, with or without leaving the dura open or augmented by a duraplasty, is a fast and effective means of increasing intracranial volume; reducing elevated intracranial pressure and increasing the compliance of the intracranial space. In the Aarabi *et al* study, mean ICP decreased from 24 to 14.6 mm Hg after decompressive craniectomy (Aarabi *et al.*, 2006).

Aans Recommendations

The American Association of Neurological Surgeons has recommended decompressive craniectomy for patients with traumatic brain injury and refractory intracranial hypertension if some or all of the following criteria were met (Abrar A Wani *et al.*, 2009):

1. Diffuse cerebral swelling on CT imaging.
2. Within 48 hrs of injury.
3. No episodes of sustained intracranial hypertension (ICP) > 40 mm Hg before surgery.

4. GCS > 3 at some point subsequent to injury.
5. Secondary clinical deterioration, and
6. Evolving cerebral herniation syndrome.

Indications for decompressive craniectomy

1. DC has most commonly been performed in patients with traumatic brain injury and cerebral infarction associated with intractable intracranial hypertension.
2. Other indications, which have mostly been described in single case reports or small case series includes aneurysmal SAH, ICH, palliation for brain tumors, meningitis, subdural empyema, encephalitis, acute disseminated encephalomyelitis, encephalopathy due to Reye syndrome, toxoplasmosis, and cerebral venous and dural sinus thrombosis (Peter Hutchinson *et al.*, 2007; Clark chen *et al.*, 2006).

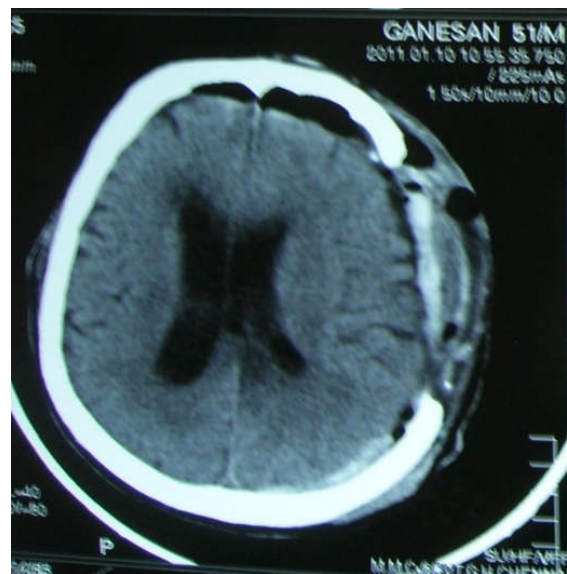
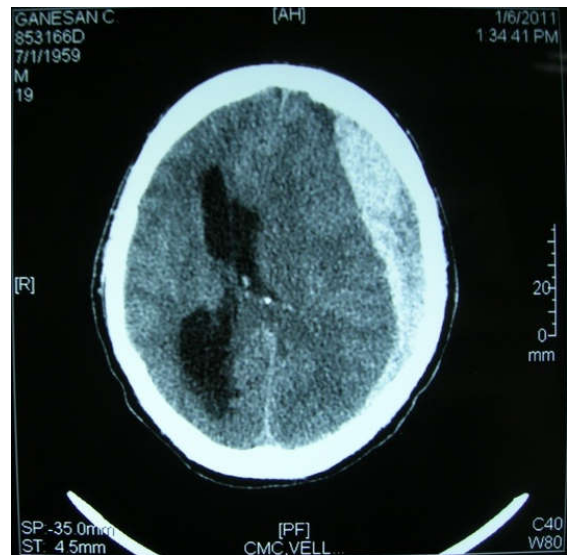
Surgical technique

Wide variability has been reported in the surgical procedures for performing decompressive surgery. Nine different types of craniectomies were reported. These variations include small to massive amounts of bone removal, unilateral or bilateral bone decompression, opening the duramater or leaving it closed, scarifying the duramater to decrease its rigidity, and sectioning of the falx among others. Localization of bone removal can be unilateral, bilateral, bi-frontal, or sub temporal; or it can be expanded to what has been called 'circumferential decompression'. In general, these decompression techniques can be divided into three approaches (Clark chen *et al.*, 2006): Frontaltemporo-parietal approach, frontal approach and temporal approach. All the three approaches can be performed unilaterally or bilaterally.

Frontaltemporo-parietal approach

The patient is placed in supine with head elevated and rotated 30 to 45 degrees. Vertex of the head is directed downwards to bring the zygomatic arch to the uppermost plane. The skin incision can be in the form of trauma flap, with the goal of exposing the following margins of craniectomy: anteriorly to the superior border of orbital roof (avoiding entry into frontal sinus); posteriorly to at least 2cm lateral to the external auditory meatus; medially to 2cm lateral to midline (avoiding sagittal sinus); and inferiorly to the floor of middle cranial fossa. Temporalis muscle is reflected anteriorly. Burr holes are placed at the keyhole, the root of the zygoma and along the planned craniectomy margin, and these are connected. The sphenoid wing is fractured and removed to the superior orbital fissure. The dural edges are tacked up to bony margin and dura is opened in a stellate manner.

Duraplasty is crucial that dural closure be non-constraining and loose to allow for further expansion of intra cranial contents. The recommendation of dimension of cranial vault removal is 10 x 15 cm craniectomy, with the lower margin extending to less than 1cm from middle cranial fossa. The lower margin of the craniectomy, relative to middle cranial fossa floor, directly correlates to the state of mesencephalic cisternal decompression (Munch *et al.*, 2000). Bi-frontal craniectomy is most widely used approach in decompression of diffuse traumatic brain injury as described by Polin and colleagues (Polin *et al.*, 1997).



Pre op mid line shift Post op reduction in shift (24hrs)

Outcome following decompressive craniectomy for TBI

Early reported results of DC performed on TBI were not very encouraging. However, recently, the use of DC has regained popularity as a treatment modality of TBI with associated increased ICP, refractory to medical treatment. Furthermore, some authors advocate that DC could be performed prophylactically, especially in developing countries, where neurointensive care resources and ICP monitoring may not be readily available (Austin *et al.*). There is no Class I evidence to support the use of DC, and prospective studies are being organized by both the European and American Brain Injury Consortiums. There are many studies in the literature with Class II and III evidence that have shown that DC might play a role in severe brain injury refractory to medical therapy. Our understanding of the different factors that determine prognosis after severe brain injury has allowed for improvement in the management of brain injury. In 2001, a small randomized study originating from the Royal Children's Hospital in Melbourne was published (Taylor *et al.*, 2001). Patients were randomized to standard treatment alone or with decompression. Those in the standard treatment group had a mean ICP reduction of 3.7 mm Hg and a favorable outcome (normal or mild disability) in 14%; patients in the standard treatment plus decompression (performed at 19 hours post injury) group had a mean ICP

reduction of 8.9 mm Hg and a favorable outcome rate of 54%. (Peter Hutchinson *et al.*, 2007) In our present study there were 136 cases underwent decompressive craniectomy, of these 90% were males and the remaining 10% were females. The most common mode of injury was road traffic accident (76%). Out of 136 patients 56 patients were survived (41%), of whom 30 patients had good recovery (22.1%), 19 patients had mild disability (14%), 4 patients had moderate disability (2.9%) and 3 patients were in vegetative state (2.2%) at the time of discharge. 80 patients died accounting for a mortality rate of 58.8%. Favorable outcome rate was 36.1% (GOS 4&5), unfavorable outcome rate was 63.9% (GOS1,2&3).

CT scan parameters

Eisenberg *et al* reported that mid line shift is very strong predictor of persistent raised ICP. Munch *et al* reviewed the effect of DC on computed tomography parameters and noted reduction of shift from 9.7 to 6.2 mm and a reduction in basal cistern compression, both known to predict poor outcome. In our present study post operative mid line shift reduction was seen in 78% of patients and post operative basal cistern opening was seen in 33% of patients, it indicates decompressive craniectomy decreases the ICP.

Conclusion

The management of post traumatic uncontrollable brain swelling remains a challenge for neurosurgeons. Primary decompressive craniectomy is a therapeutic option for patients who had moderate to severe head injury with the clinical and radiological features of increased ICP. The opening of the basal cisterns can be considered as a predictor of favorable outcome following decompression craniectomy. Whereas the reduction in midline shift alone cannot be considered as a predictor of favorable post operative outcome. The availability of CT brain even in Rural areas makes it an easily accessible tool to assess the post operative opening of basal cisterns.

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