Analysis of Prognostic Factors and Complications Following Decompressive Craniectomy in Severe Traumatic Brain Injury

Dinesh Shukla

1Associate Professor, Department of Neurosurgery, MLB Medical College, Jhansi, Uttar Pradesh.

ABSTRACT

BACKGROUND
The aim of the study was to investigate the therapeutics effects, complications and factors associated with prognosis of patients with severe Traumatic Brain Injury (TBI) in whom decompressive craniectomy (DC) was performed.

METHODS
A retrospective study was conducted between 2015 and 2019 and included patients with severe TBI who underwent decompressive craniectomy. The parameters assessed were clinical state using the Glasgow Coma Score (GCS), CT Scan findings, details of DC, complications, factors associated with mortality and neurological outcome upon discharge from hospital using Glasgow Outcome Scale (GOS).

RESULTS
A total of 83 patients were included in the study. The mean age of the patients was 32 ± 14 years. There were 68 (81.9%) males and 15 (18.1%) females. Most common cause for injury was road traffic accident (62%). The average Glasgow coma score at admission was 7 ± 3.44 (53%). Complications included contralateral hematoma (4.8%), external cerebral herniation (28.9%), seizures (6.0%), hydrocephalus (8.4%), postoperative infection (30.1%) etc.

CONCLUSIONS
Decompressive craniectomy is an important procedure to save the life of patients with severe TBI. However some complications associated with the procedure have to be kept in mind before using it in a generalised manner. Low GCS at admission, development of hydrocephalus and old age were factors associated with poor outcome.

KEYWORDS
Decompressive Craniectomy, Severe Traumatic Brain Injury, Complications
BACKGROUND

Traumatic brain injury (TBI) is one of important causes of raised intracranial tension and death among young people in the industrialised countries. Acute post traumatic brain swelling can be one of the severe consequences of traumatic brain injury. It has been described as the increase of brain size with in the several hours following (TBI) without haematoma. The pathophysiological basis of posttraumatic brain swelling is not clearly understood. It is believed that cytotoxic oedema could be the main contributing factor to this phenomenon rather than vasogenic oedema or cerebral vasocongestion. Increased intracranial pressure (ICP) is one of the main reasons associated with poor outcome. In refractory intracranial hypertension (ICH), ICP cannot be controlled with first tier therapeutic measures such as sedation, CSF drainage and osmotherapy. It is present in 10 to 15% of patients. In such cases second tier measures such as therapeutic hypothermia or decompressive craniectomy (DC) needs to be considered.

Decompressive craniectomy is supposed to reduce ICP, to prevent herniation and vicious cycle of brain swelling and to counter the deleterious effects of TBI. It has been suggested that in few subgroups of patients with post-traumatic brain swelling, DC should be routinely performed before irreversible brain damage occurs. Decompressive craniectomy (DC) is usually indicated in cases where CT scan shows a midline shift of more than 5 mm, especially with thin layered subdural hematoma. Although Decompressive craniectomy (DC) appears to efficient therapeutic strategy is few situations, there is no class 1 level of evidence to support or refute the effectiveness of its utilization in adults. Also the criteria and safety of DC has not been fully established.

The objective of this study was to further investigate the therapeutics effects, complications and factors associated with prognosis of patients with severe TBI in whom DC was performed.

METHODS

It was a retrospective study conducted between 2015 to 2019 at Hospital associated with MLB Medical College, Jhansi. It is the only major trauma centre of the region and many patients with head injuries and polytrauma are referred here from peripheral health care facilities. All adult patients above 18 years of age with severe TBI (GCS 5-8) who underwent DC in our hospital during the study period were included in the study. This study was approved by ethical committee. An informed consent was taken from a close relative of the patient. The parameters which were assessed were clinical state using the Glasgow Coma Score (GCS), CT Scan findings, details of DC, complications, factors associated with mortality and neurological outcome upon discharge from hospital using Glasgow Outcome Scale (GOS). Patients who were excluded from the study those below the age 18 or above 65 years, patients who had multiple injuries, if any previous disabling neurological disease was present, bleeding diathesis, those with very severe injuries who did not survive more than 24 hours, any previous cranial surgeries and spinal cord trauma. The clinical outcomes of studied patients were divided into good and bad outcomes. Good outcome was defined by GOS scores of 4 or 5 i.e., without disability or light disability respectively whereas poor outcome was defined by GOS scores of 1, 2 or 3 was death, vegetative state or severe disability respectively. Severe TBI was defined as GCS equal to or less than 8 after initial review or with an initial GCS score greater than 8 but which requires neurosurgery for the evacuation of a space occupying intracranial lesion.

Definition of the following parameters was as follows:

**Shock:** as mean blood pressure less than 70 mmHg and/or clinical signs of peripheral hypoperfusion or lactic acidosis greater than 2 mm; ICH as ICP greater than 20 mmHg; Post-surgical central nervous system infection included meningitis, ventriculitis, cerebral abscess and subdural empyema.; Subdural collection as a hypodense collection greater than 1 cm; Hydrocephalus as dilatation of the ventricular system which is accompanied by signs or clinical symptoms requiring treatment; External brain herniation was considered to be cerebral protrusion greater than 1.5 cm thorouhg the bone defect. Ventilator associated pneumonia (VAP) was defined as association of fever or leukocytosis, purulent tracheobronchitis, new of persistent chest x-ray images and cultures of positive tracheal secretions or bronchial alveolar lavages greater than 10^4 colony forming units (CFU/mL). Severe sepsis was defined as the presence of an infectious focus associated with systemic inflammatory syndrome and multiple organ failure.

Refractory ICH (Intracranial Hypertension) was defined as ICP greater than 20-25 mmHg that was maintained for at least 30 minutes and there was no response to first-tier therapeutic measures. Decompressive craniectomy was divided into 2 types: 1) Primary- in which the operating surgeon performs DC after evacuation of a subdural hematoma and also in a condition where based on CT scan findings, diffuse cerebral oedema without hematoma was found. 2) Secondary- in which DC is performed for the treatment of refractory ICH during medical treatment.

Surgical Procedure

According to the guidelines set forth by the Joint Section on Neurotrauma and Critical Care of the Brain Trauma Foundation and the American Association of Neurological Surgeons for management of severe head injury, all patients underwent DC within 24 hours after injury along with the first-tier measures to reduce the ICP. During surgery, bone window of 15 cm was created with craniotomy drill, dura was opened in C shape manner with a base towards superior sagittal sinus and duraplasty was done using expanded dura substitute. Unilateral DC was done for lesions confined to one cerebral hemisphere. In patients with bifrontal or anterior cranial fossa lesions, bifrontal craniectomy was done from anterior cranial fossa to the coronal suture. Following craniectomy, EDH or SDH were evacuated when...
present. Brain intraparenchymal hematomas were removed in cases with persistent brain swelling following craniectomy and hematoma evacuation.

RESULTS

There were a total of 83 patients who underwent DC craniectomy during the study period. The baseline clinical characteristics of patients are shown in Table 1. The mean age of the patients was 32±14 years. There were 68 (81.9%) males and 15 (18.1%) females. Most common cause for injury was road traffic accident (62%) followed by fall from height (14%). Less common causes were assault injuries, fall of heavy object overhead, workplace accidents etc. The average Glasgow coma score at admission was 7±3. On examination, pupils were bilaterally unreactive in 36 (43.3%) patients, Unilaterally unreactive in 16 (19.3%) patients and bilaterally reactive in 31 (37.3%) patients. CT scan examination revealed a midline shift of ≤10 mm in 21 (25.03%) patients and >10 mm in 62 (74.7%) patients. Among the documented intracranial pathologies most common was SDH in found in 50 (60.2%) patients followed by contusions in 22 (26.5%) patients. DC was performed as a primary intervention in 66 (79.5%) and as a secondary intervention in 17 (20.5%) patients.

Complications following DC occurred in 44 (53%) patients. Few patients developed more than one complications. Contralateral hematoma developed in 4 (4.8%) patients and was one of the earliest complications to develop for which reoperation had to performed in 2 patients. External cerebral herniation occurred in 24 (28.9%) patients. 8 (6.0%) developed seizures postoperatively in spite of prophylactic antiepileptic drugs. 7 (8.4%) patients developed hydrocephalus for which ventriculoperitoneal shunt was done for all. 25 (30.1%) patients had postoperative infection including meningitis and ventriculitis. Appropriate antibiotics were administered in them based on culture and sensitivity report. 3 (3.6%) patients had CSF leak through the surgical wound for which resuturing was done. 4 (4.8%) patients developed venous infarction. Non craniotomy related complications were Ventilator associated pneumonia (VAP) in 32 (38.5%) patients, 2 (2.4%) patients developed severe sepsis, 10 (12.1%) patients had electrolyte disturbance and 8 (9.6%) had renal dysfunction.

The clinical outcome was determined using Glasgow outcome score (GOS). Overall mortality in the study was 35 (42.1%). 30 patients had good outcome with GOS 5 in 16 (19.3%) patients and GOS 4 in 14 (16.8%) patients. 53 patients had poor outcome- GOS 3 in 12 (14.5%) patients, GOS 4 in 6 (7.2%) patients and 35 (42.1%) patients died. On analysis of factors which could have found to have impact on outcome it was found that lower GCS at admission and occurrence of hydrocephalus were associated with poor outcome. Also, old aged patients didn’t have a good outcome.

DISCUSSION

Traumatic brain injuries are one of the most common causes of death in industrialised nations.9 In past, patients with severe head injuries not associated with obvious surgical lesion and with significant cerebral oedema were managed with external ventriculostomy and kept in ICU for non-surgical treatment of ICH such as CSF drainage, sedation and paralysis, hyperosmolar solutions such as mannitol and hypertonic saline, hyperventilation, barbiturates and hypothermia. But the mortality in these subgroups of patients was markedly high. So it was thought that markedly raised ICP which leads to cerebral infarction, hypoperfusion and hypoxia. But the mortality in these subgroups of patients was markedly high. So it was thought that markedly raised ICP which leads to cerebral oedema and secondary brain damage is not treatable by medical measures alone. In these cases, decompressive craniectomy (DC) emerged as an alternative therapy. DC helps in reduction of ICP and improving the clinical outcome.10,11 How it is also associated with controversies such as the unclear precise indications, timing and long term functional outcome. Although DC is a relatively simple surgical procedure, complications are known to occur which sometimes may have significant impact on the clinical outcome.12 A majority of these
Complications develop from normal pathophysiologic changes in ICP, CSF circulation and cerebral blood flow (CBF) following removal of large portion of cranial vault. Also, early DC is mired with controversy and few studies have reported that early DC can increase the cerebral oedema (increase the transmural gradient of hydrostatic pressure in the capillary bed) and can lead to infarcts with haemorrhagic transformation until cortical necrosis. In present study we aimed to find the prognostic factors associated with outcomes in patients with decompressive craniectomy.

In present study, primary DC was performed in 66 (79.5%) patients. It was based on cerebral characteristic findings intraoperatively and also according to the initial CT scan findings. The most important complication reported in previous studies is subdural hygroma and in majority of cases it resolved spontaneously. In present study, subdural hygroma occurred in 6 (7.2%) patients and resolved gradually in all the patients following conservative management. Contusion expansion or development of new intracranial hematomas occurs contralateral or remote to the decompressed hemisphere. The reason for this complication may be reduction or loss of the tamponade effect of calvarium. It can develop early after decompression and management includes keeping as close monitoring and early detection of any further deterioration.

The reason for external cerebral herniation is brain oedema. External cerebral herniation may lead to cortical vein compression and cortical laceration resulting in venous infarction of herniated brain tissue and cortical damage. To prevent this large craniectomies with augmentative duraplasty is advised to allow the brain to expand outward without constriction and decreased risk of venous infarction. On analysis of factors which could have found to have impact on outcome it was found that lower GCS at admission and occurrence of hydrocephalus were associated with poor outcome. Also, old aged patients didn’t fare well and had a poor outcome.

Limitation of present study is its retrospective nature done in a single centre and small number of patients. This is the same reason why statistical analysis could not be performed.

CONCLUSIONS

Decompressive craniectomy is an important procedure to save the life of patients with severe TBI. It decreases the raised ICP and hence improves the cerebral blood flow and viability of cerebral tissue. However, some important complications associated with the procedure have to be kept in mind before using it in a generalised manner. Also, it was observed that low GCS at admission, development of hydrocephalus and old age were factors associated with poor outcome.

REFERENCES

[1] Chibbaro S, Tacconi L. Role of decompressive craniectomy in the management of severe head injury with refractory cerebral edema and intractable intracranial pressure. Our experience with 48 cases. Surg Neurol 2007;68 (6):632-638.
[2] Martins ET, Linhares MN, Sousa DS, et al. Mortality in severe traumatic brain injury: a multivariated analysis of 748 Brazilian patients from Florianópolis City. J Trauma 2009;67 (1):85-90.
[3] Liu WG, Qiu WS, Shen H, et al. Management of postruama brain swelling based on clinical typing. Chin J Traumatol 2004;7 (3):175-178.
[4] Sahuquillo J, Biestro A, Mena MP, et al. First tier measures in the treatment of intracranial hypertension in the patient with severe craniocerebral trauma. Proposal and justification of a protocol. Neurocirugia (Astru) 2002;13 (2):78-100.
[5] Guerra WK, Gaab MR, Dietz H, et al. Surgical decompression for traumatic brain swelling: indications and results. J Neurosurg 1999;90 (2):187-196.
[6] Le Galle JR, Lemeshow S, Saulnier F. A new Simplified Acute Physiology Score (SAPS II) based on a European/North American multicenter study. JAMA 1993;270 (24):2957-2963.
[7] Teasdale GM, Pettigrew LE, Wilson JT, et al. Analyzing outcome of treatment of severe head injury: a review and update on advancing the use of the Glasgow Outcome Scale. J Neurotrauma 1998;15 (8):587-597.
[8] Bratton SL, Chestnut RM, Ghajar J, et al. Guidelines for the management of severe traumatic brain injury. J Neurotrauma 2007;24 Suppl 1:51-95.
[9] Martins ET, Linhares MN, Sousa DS, et al. Mortality in severe traumatic brain injury: a multivariated analysis of 748 Brazilian patients from Florianópolis City. J Trauma 2009;67 (1):85-90.
[10] Aarabi B, Hesdorffer DC, Ahn ES, et al. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. J Neurosurg 2006;104 (4):469-479.
[11] Gaab MR, Rittierodt M, Lorenz M, et al. Traumatic brain swelling and operative decompression: a prospective investigation. Acta Neurochir Suppl (Wien) 1990;51:326-328.
[12] Flint AC, Manley GT, Gean AD, et al. Post-operative expansion of hemorrhagic contusions after unilateral decompressive hemicraniectomy in severe traumatic brain injury. J Neurotrauma 2008;25 (5):503-512.
[13] Moody RA, Ruamsuke S, Mullan SF. An evaluation of decompression in experimental head injury. J Neurosurg 1968;29 (6):586-590.
[14] Stiver SI. Complications of decompressive craniectomy for traumatic brain injury. Neurosurg Focus 2009;26 (6):E7.