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Decompressive Craniectomy in a case of Severe Traumatic Brain Injury: To do or not to do- Two Case Reports

Author
Dr (Col) Gurmeet Singh Sarla
Classified Specialist Surgery, Military hospital Devlali,
Devlali Cantt, Nasik, Maharashtra, Pin 422401
Phone 9882562223

Abstract
Decompressive craniectomy as a surgical procedure to relieve raised hypertension has been performed in the early 20th century. However even after a century and despite numerous studies in this regard, the decision to conduct decompressive craniectomy still remains a dilemma. In our case report we discuss two cases of traumatic brain injury where the surgical team was faced with the challenge of performing the surgery in a peripheral hospital with a favorable post-operative outcome.

Introduction

Traumatic brain injury (TBI) is among the major causes of mortality and morbidity in the developing as well as the developed countries. Though the exact incidence is not known various studies quote the annual incidence at 2% of global population.[1] Mortality in case of severe TBI has significantly dropped in the last decade as revealed on analyzing International Mission for Prognosis and Clinical Trials (IMPACT) database. However 50% of those affected still continue to die or have non functional outcome[2]. The management of severe TBI has further been streamlined by the development of Brain Trauma Foundation Guidelines which enlists medical as well as surgical treatment modalities available along with level of evidence[3]. However the decision whether to perform decompressive craniectomy (DC) is still a point to debate. In our case report we present a case of severe head injury where the surgical team was faced with such a challenge, whether to perform DC or not.

Case Report

Case 1

51 years old, male, with no known comorbidities, was brought to our hospital in an unconscious state. Individual had sustained head injury following fall from two wheeler after a high velocity impact. The individual was not wearing a helmet. Initial management was done at a peripheral clinic where the patient was intubated and neuroimaging in form of NCCT head done. Patient was then shifted to our centre which is a 280 bedded hospital.

On initial evaluation GCS was $E_1V_TM_1$. Pupillary reaction was abnormal in that right pupil was dilated and fixed while left was normal size and reacting normally. Plantar reflex was bilaterally mute. There was a visible deformity in right arm

with abnormal mobility. NCCT Head showed depressed fracture of left parietal bone along with hemorrhagic contusion in right temporal and left parietal region. There was sub-arachnoid hemorrhage in left sylvian fissure and left fronto-temporo-parietal sulci and acute thin subdural hemorrhage in left frontal and right temporal convexities. There were fractures of lateral wall of left orbit, left greater wing of sphenoid, left zygomatic arch and temporal bone and fracture of nasal bone and bony nasal septum (Fig 1). Radiograph of the right arm showed supracondylar fracture of right humerus. Hematologic and biochemical parameters were within normal limits.

Patient was initially managed conservatively with cerebral decongestants, antiepileptics, antibiotics, ventilator support and inotropic support. He was clinically monitored with six hourly GCS, Vitals and pupillary reaction. Patient was also closely monitored for any hematologic or biochemical derangement. In72 hrs he showed no significant clinical improvement despite optimal medical management. Decompressive craniectomy was planned for the patient

A large right hemi-craniectomy scalp flap was marked and raised. Multiple fragments of skull fracture were visualized. There was massive brain swelling with a large extra dural and sub dural hematoma in right temporo-parietal region with multiple cerebral contusions. Dura was torn at multiple sites. Loose bone fragments were excised and skull defect enlarged widely to provide adequate decompression. Contused brain and subdural hematoma were removed, hemostasis ensured and gel foam sheet was placed on the dural defect. Brain was pulsating well at the end of the procedure. Scalp wound was closed in a single layer with a subgaleal drain. (Fig 2)

Post operatively patient was continued on ventilator support, cerebral decongestants, anti-epileptic and broad spectrum antibiotics. In view of the need for continued ventilator support, elective tracheostomy was performed. Patient showed steady recovery post operatively and the vitals remained stable and there was no evidence of immediate or early post operative complication. By seventh post op day patient's GCS improved to $E_2V_TM_4$ and he was subsequently placed on T-piece. Once patient had stabilized he was transferred to tertiary care centre for management by neuro surgical team. Patient was followed up after a month when his tracheostomy was closed and he was ambulant on wheel chair and alimentation was by means of Ryle's tube. Presently patient is planned for feeding jejunostomy and the closure of the cranial defect by means of prosthesis. His current Glasgow Outcome Score Extended is assessed to be 4 at the end of 03 months and he is on continuous follow up.

Case 2

25 years old male, sustained head injury following road traffic accident. There was associated history of loss of consciousness of five minutes. There was no history of vomiting, nose bleed or bleeding from ears. On examination patient was drowsy and vital parameters were within normal limits. His GCS was 13/15 (E3V4M6) and pupils were normal in size and reaction on both sides. Motor examination revealed Grade IV power in all four limbs. Rest systemic examination was within normal limits. NCCT head revealed right frontal epidural hematoma with significant midline shift. There was sharply demarcated biconvex heterogeneously hyper dense lesion in the right frontal region. The lesion exerts mass effect as evidenced by buckling of the subjacent cerebral cortex with midline shift. There is chinking of the frontal horn of the right lateral ventricle (Fig 3). His biochemical and hematological profile was within normal limits.

In view of the setting of neurological deficit in form of quadriparesis and radiographic evidence of midline shift, frontal craniotomy was planned for the patient. Intra operatively there was a large extra dural hematoma in the right frontal region (Fig 4). The dura was intact. Hematoma evacuated and bone flap reposed. Scalp flap was closed over

drain. Patient was put on broad spectrum antibiotics, cerebral decongestants and antiepileptic. His GCS score showed in 24 hours and was 15/15 (E4V5M6). His motor deficit improved within 48hrs. NCCT head was repeated post operatively which revealed evidence of right fronto- parietal craniotomy with minimal midline shift (Fig 5). Patient showed steady post op recovery. Drain was removed and cerebral decongestants tapered over one week. At the end of one month there has been no post-operative complication or any residual neurologic deficit and he is kept on regular follow up.

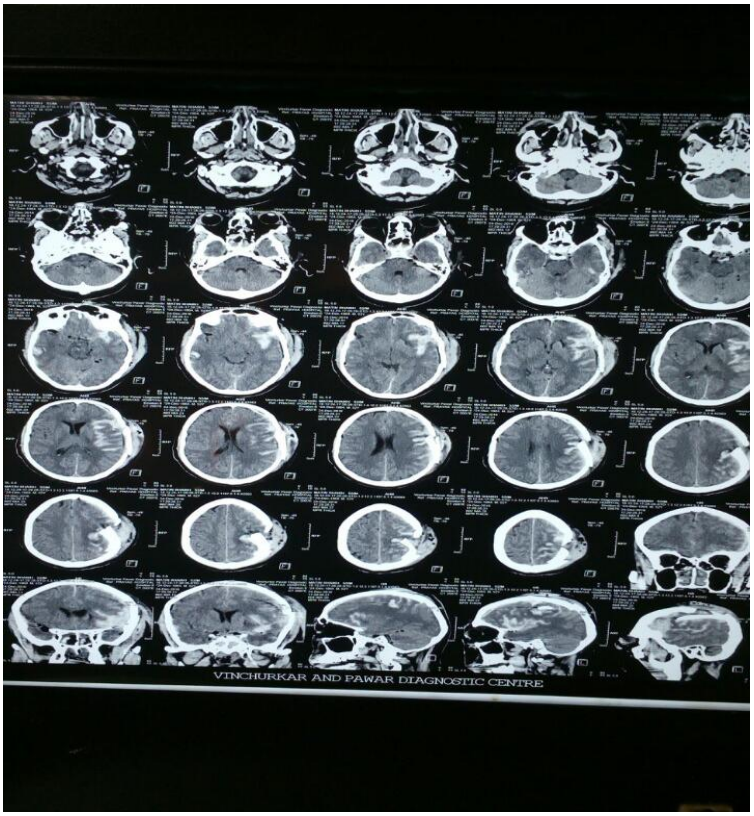

Fig 1 NCCT Head showing depressed fracture left parietal bone along with hemorrhagic contusions in right temporal and parietal region

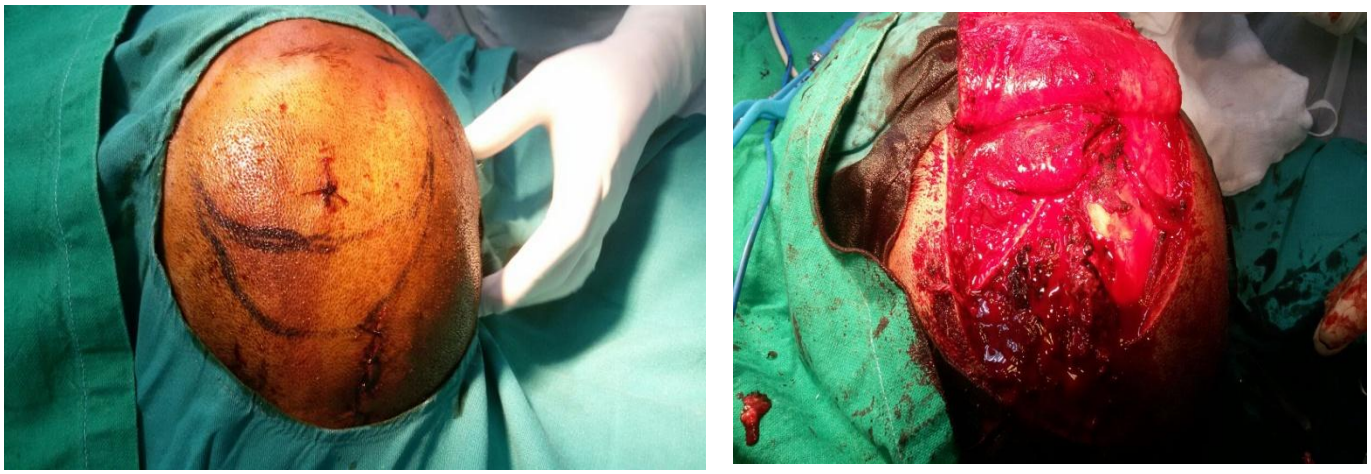

Fig 2 Intraopertive images showing skull fracture and cerebral contusion

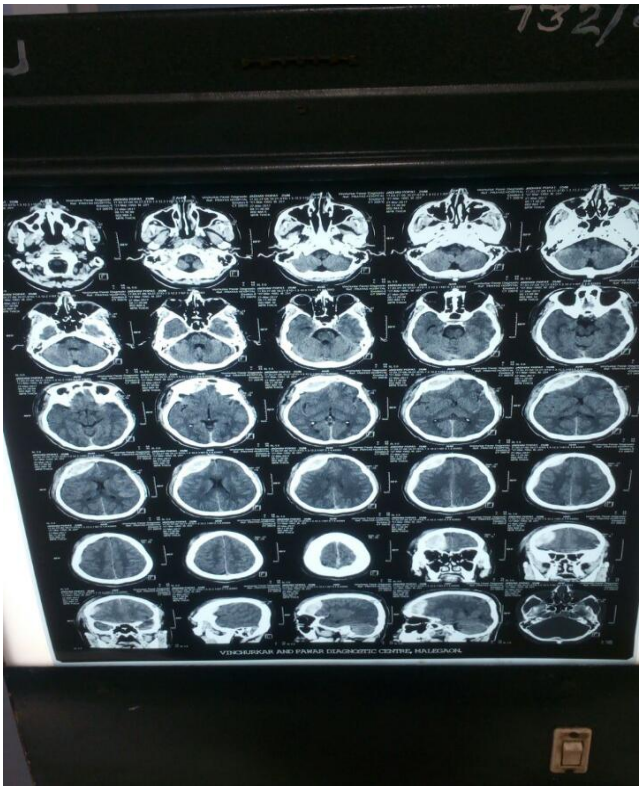

Fig 3 NCCT head showing Right Frontal Epidural Hematoma with Midline Shift

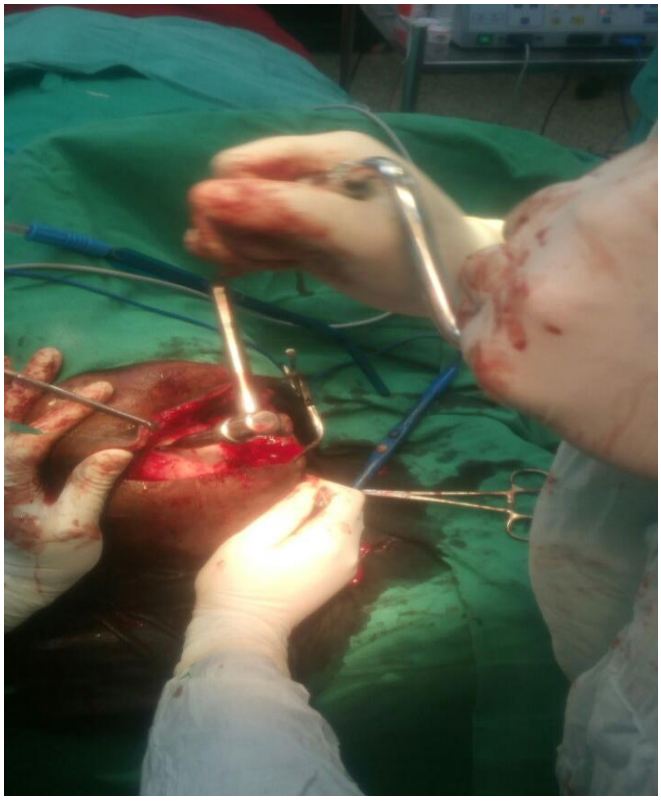

Fig 4 Intraoperative image- Frontal (Burr Hole) Craniotomy

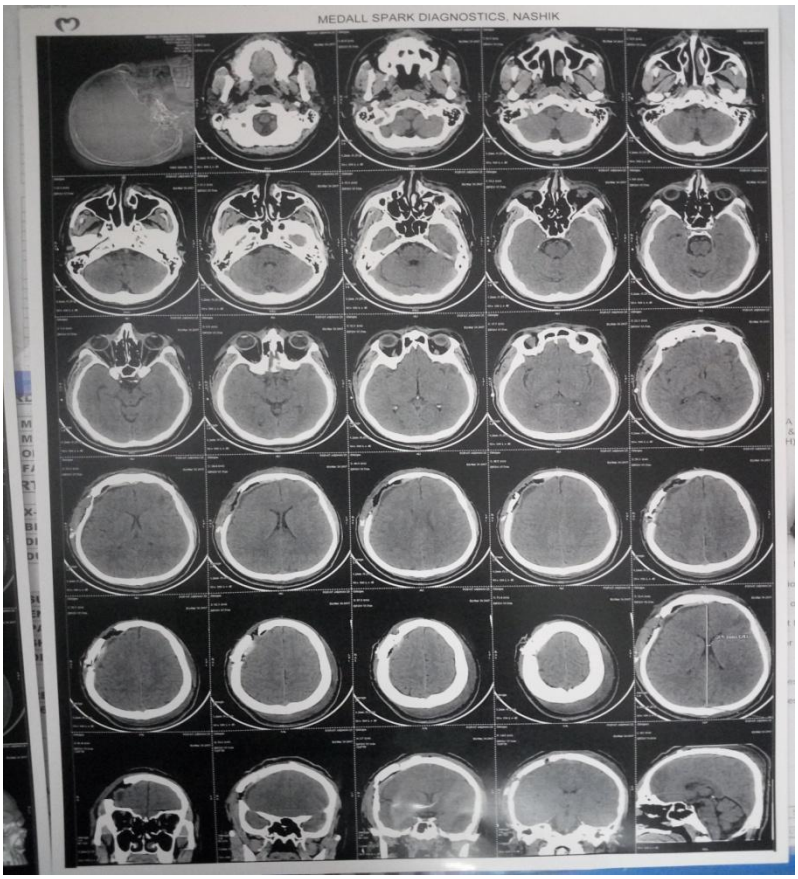

Fig 5 Post Operative NCCT of Case 2 showing resolution of midline shift compared to Fig 3

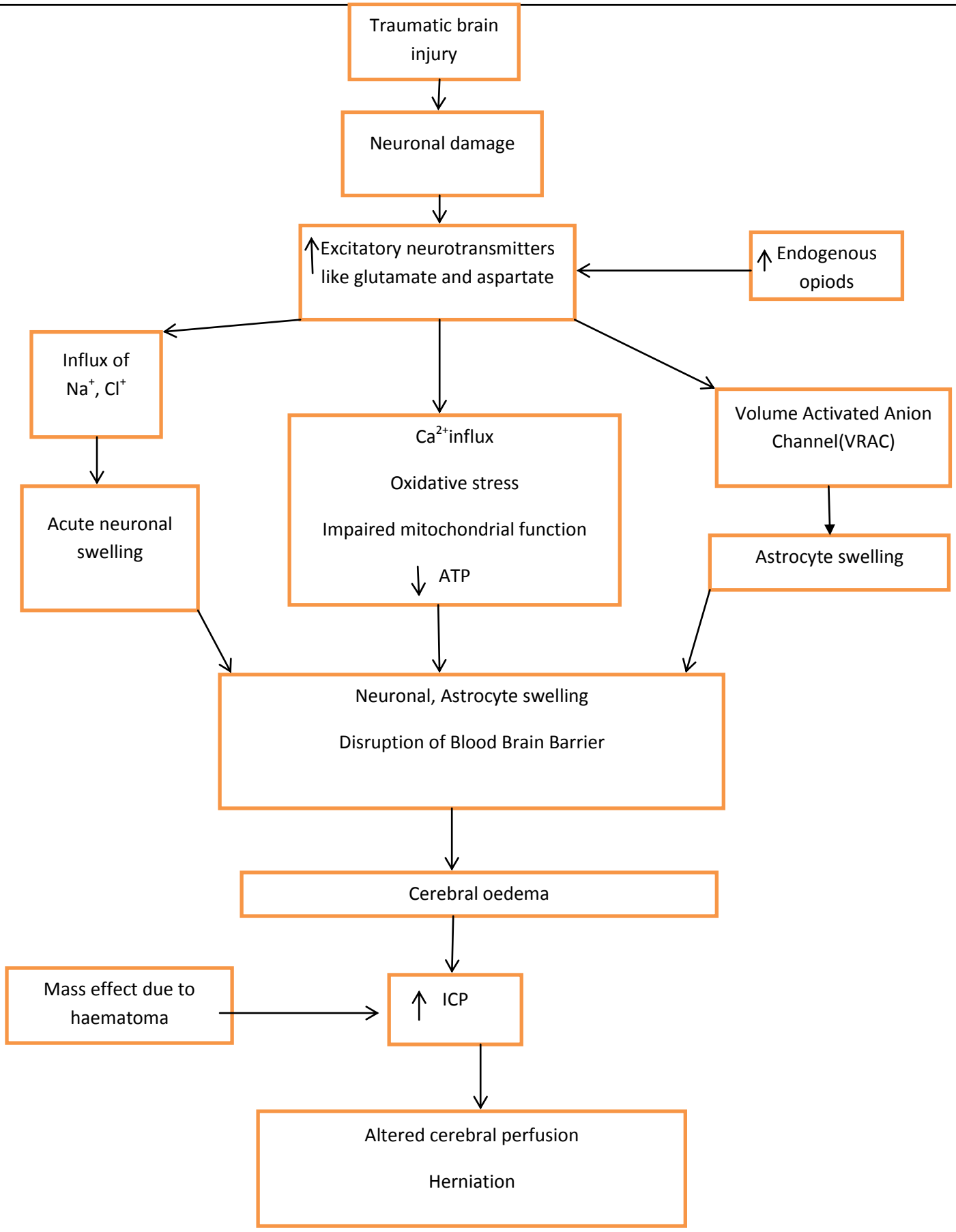

Fig 6 Pathophysiology of Secondary Brain Injury

Traumatic Brain Injury (TBI) is the outcome of an external insult to the cranium and its contents leading to temporary or permanent impairment, functional disability or psychosocial maladjustment. It can be primary injury or secondary injury. Primary injury is the one occurring at the time of impact and can be – skull fractures, intracranial bleed, brain contusion, diffuse axonal injury. Secondary injuries are those that are attributable to cellular damage set off by the primary injury. The sequence of event leading to secondary damage is elucidated in Fig 6.

The Monroe Kelly doctrine states that the cranium being of a fixed capacity the sum of the intracranial contents i.e. brain tissue, blood and CSF remains constant, implying that if there is increase in one of the components then there will be compensatory reduction in volume of other component. So in the setting of cerebral edema and raised intra cranial pressure in TBI there is fall in the cerebral perfusion as given by the equation below leading to further secondary damage.

CPP= MAP –ICP

CPP= Cerebral Perfusion Pressure

MAP = Mean Arterial Pressure

ICP= Intra Cranial Pressure

Malignant cerebral edema following TBI is a primary cause of poor neurological outcome. Juul et al, in a post hoc analysis of data from multicenter Selfotel trial showed that raised ICP is the most powerful predictor of neurologic worsening[2]. As given in Fig 6, raised ICP may have vascular effect (leading to impaired perfusion) as well as mechanical effect (herniation). Therefore control of traumatic cerebral hypertension is the principal target of brain protective therapies as per the brain trauma foundation guidelines. This can be achieved by conservative as well by surgical methods.

Conservative approach includes-

a) Prophylactic hypothermia
b) Hyperosmolar therapy- 20% mannitol, 3% hypertonic saline
c) Hyperventilation
d) Head end elevation to 30-45^{o}

Surgical approach include-

a) Decompressive craniectomy
b) CSF drainage

Treatment recommendation as per the brain trauma foundation guidelines is given in Table 1[(5)]. Hemicraniectomy as a therapeutic measure for control of raised ICP was first described more than a century ago (Kocher 1901) and it was only in 1971 that it was first introduced as a treatment option for traumatic subdural haematoma[3]. The rationale behind DC is that it allows for the edematous cerebral hemisphere to "decompress" through the craniectomy defect, thus preventing the rise in ICP as also enhancing the cerebral perfusion. DC can be primary or prophylactic wherein the surgeon guided by the intra-operative findings decides to leave the bone defect open, anticipating a rise in ICP. Secondary DC is performed secondary to failure of first line interventions to reduce ICP[2].

Benefits of DC have been a topic of debate and various studies have shown conflicting results. Cochrane collaboration literature review in 2009 did not recommend DC in adult trauma population for primary treatment; however it still concluded that DC can be a valid treatment modality for refractory intracranial hypertension [3]. The DECRA trial was a multicentre RCT conducted in 15 hospitals in Australia, New Zealand and Saudi Arabia to test the efficacy of DC. The main conclusion of the study was that DC decreased ICP and length of hospital stay but was associated with more unfavorable outcome. However the surgical technique used in DECRA trials as well as the ICP threshold and the evaluation of the results has been debated [2]. On the other hand retrospective studies have been published that show significant percentage of patients ranging from 16-69% experiencing favorable outcome [4].

Conclusion

Management and outcome of traumatic brain injury has undergone paradigm shift in the past century which has further been facilitated by

Brain Trauma Foundation guidelines, though decompressive craniectomy still remains a topic for debate. Despite numerous studies there has been no conclusive consensus regarding the benefits of DC. However in the appropriate clinical setting the benefits of DC outweigh the risks. In our case report we have tried to highlight two such cases where DC was performed as a life saving measure and has shown favourable outcome.

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