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Decompressive craniectomy in traumatic brain injury Rationale and practice

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Abstract: Despite the growing fund of knowledge on the pathophysiology of traumatic brain injury, the outcomes of severe TBI remain abysmal. While the necrotic and apoptotic cascades triggered off by the primary traumatic insult remain refractory to treatment, the secondary sequelae of increased intracranial pressure remain within the therapeutic realm. The benefit of evacuation of mass producing hematomas is beyond dispute. The management of brain edema however remains suboptimal despite the best medical management. The role of surgical evacuation of edema inducing contusions recalcitrant to osmotherapy is explored in this paper. The role of decompressive craniectomy in the control of refractory hypertension and the rationale and timing of the procedure are discussed.

Keywords: brain edema, decompressive craniectomy.

Severe Traumatic Brain Injury (TBI) is associated with mortality rates nearing 50%[1]. Some of these deaths are due to the primary insult. This includes mechanical neuronal and vascular disruptions. The primary insult is still considered irreversible as paradigms of neuronal replacement and repair remain conceptual[2].

TBI induces excitotoxic surges which facilitate an influx of calcium into the cell. The mitochondrial metabolism fails. Cascades mediated by calpains are triggered which culminate in cell necrosis[3]. Caspases ignite the apoptotic genes which programme further neural loss. Apoptotic cell death occurs in a progressive and delayed manner without the inflammation and swelling that characterizes neuronal cell death. Pharmacological regimes targeting these 'cell death' cascades are even today, beyond the clinicians' reach[4].

Contemporary therapeutic paradigms aim to prevent a second neuronal insult. This second insult is largely mediated by sub-optimal cerebral blood flow and inadequate oxygen delivery. The cerebral perfusion pressure head reflects the difference between the mean arterial and intracranial pressures. The pressure head is lost as the intracranial pressure (ICP) rises. Cerebral vascular reactivity is the phenomenon that regulates autoregulation which functions between mean arterial pressures of 50 to 150 mm Hg in the uninjured brain. The therapeutic armamentarium at our disposal is aimed at averting a calamitous rise in intracranial pressure. Restoration of cerebrovascular reactivity is still not possible.

Various factors contribute to the increase in volume of intracranial contents, which translates into increased pressure within the closed cranial cavity (Monro-Kellie doctrine). The most significant amongst these are mass producing hematoma and brain swelling. Any accumulation of blood outside the vascular channels, including extradural, subdural and intracerbral hematomas and cerebral contusions which reflect intraparenchymal bleeds act as a mass lesion. The indications for surgical evacuation of these mass lesions are relatively standardized.

The second factor contributing to an increase in the ICP is brain swelling. Conventionally this swelling has been attributed to both cytotoxic edema [5] (as a result of membrane permiability and mitochondrial failure) and vasogenic edema consequent to a derangement of the blood brain barrier[6]. The most significant driving force behind the edema accumulation is however, the osmotic load of cellular breakdown products[7].

Management of brain edema is primarily medical. Optimal oxygenation and the maintenance of mild hypocarbia contribute to ICP management by rationalizing intravascular blood volume. Promoting venous return by raising the head end of the bed[8] and avoiding high positive end expiratory pressure during elective ventilation also help by facilitating venous return from the cranium.

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Osmotic agents like 20% mannitol and hypertonic saline act primarily by drawing fluid across the blood brain barrier into the vascular tree, in addition to other benefits like improving cerebral blood flow, Rheogenic properties and antioxidant effects[9]. Neuronal protection strategies including mild hypothermia and barbiturates have been tried with variable benefit[10]. Cerebrospinal fluid (CSF) drainage from a ventricular catheter (often placed to monitor ICP) may be used to tide over a potentially catastrophic ICP rise.

However, despite all those measures, ICP control is often not optimal or adequate. Decompressive craniectomy has been established as an effective modality for the control of intracranial hypertension[11].

History of the procedure: Cushing popularized the concept of temporal decompression for increased intracerebral pressure associated with tumors. However, the resurgence of interest in decompressive craniectomies for intracranial hypertension is a more recent phenomenon[12]. The role of decompressive hemicraniectomies in the management of malignant MCA infarcts is well established[13]. In higher grade Aneurismal subarachnoid hemorrhage too, decompressive craniectomy has resulted in improved outcomes.

In traumatic brain injury, there have been many studies have explored the ICP dynamics of decompressive craniectomy. The intracranial pressure decrease with bone flap removal has been shown to be around 30% which improves to 70% when the dura is laid wide open. There is a radiological reversal of herniation and brain shifts. However cerebrovascular reactivity remains impaired. There have been comparisons of outcomes between patients undergoing decompressive craniectomy and comparable historical data from the traumatic coma data bank. In severe head injuries these comparisons showed good outcomes in 37% of operated patients vs 16% in controls[14].

The Rescue ICP trial[15] is a multicenter randomized trial comparing medical management with surgical treatment, coordinated by the University of Cambridge UK and the European Brain Injury Consortiums. Patients with ICP > 25mm of Hg are randomized into two grades. In one group there is continuation of optimal medical management (including barbiturates) and in the others, the patients undergo decompressive craniectomy.

Timing of surgery : The timing of decompression is crucial to the outcomes[16]. The aim is to expand the available cranial volume before cerebral blood flow impairment occurs due to a failure of the cerebral perfusion pressure, brain shifts and vascular distortions. Crucial to this timing is an understanding of the temporal phase of edema production which is etiology specific.

In traumatic brain injury induced cerebral contusions, the onset of life threatening brain swelling is within two to three hours and is considered to be due to the osmotic load (up to 390 mosmol) exerted by the break down debris of membrane & cytoplasmic structures. There is a second phase of edema which occurs from three to five days due to the breakdown of RBC and resultant inflammatory cascades. In malignant MCA infarcts, on the other hand, the maximum edema occurs from 48 hrs to five days. The optimal timing of surgery, therefore has to be ultra early (within 6 hrs) in TBI and could be delayed up to 24 hrs in MCA infarcts.

Technical Aspects of the Procedure: The aim of decompressive surgery is to decrease the intracranial pressure, to shift the pressure off the midline structures and to reverse herniation. The planning of a decompressive craniectomy depends upon the location of brain swelling. A frontotemporoparietal craniectomy is used in situations where uncal and sub falcine herniation are occurring or imminent. Removal of bone up to the temporal base is desirable in these cases and a minimum bone flap diameter of 12cms is used. There is a definite role for tailoring the craniectomy to suit the pathology. In patients who have a significant mass lesion in the form of a contusion or a sub dura hematoma, the removal of a bone flap of 10cms diameter may be enough, when combined with evacuation of the mass lesion to optimize the ICP. On the other hand – in a malignant MCA main trunk occlusion infarction, it is difficult to differentiate between viable and nonviable brain. There is no mass to be evacuated and ICP control is purely a function of increasing the container volume. Here, a larger craniectomy would be desirable. It has been shown that a 10cm diameter bone flap with durotomy provides an additional volume of around 50ml. Wide opening of the dura is mandatory to achieve volume expansion, as the dura is not distensible. 70% of the volume expansion of a decompressive craniectomy is achieved through wide dural opening and only 30% by bone removal alone. In our experience in decompressive hemi-craniectomy dural opening by radial

dural incisions are preferred over circumferential incisions. The brain bulges more uniformly. Later during reconstruction this facilitates a safer separation of the scalp flap from the brain surface. In bifrontal decompressive craniectomy[17] – the option of disconnecting the superior sagittal sinus at its anterior most end has been described.

Restoration of a dural cover is desirable. The duraplasty aims at interposition of a layer between the brain and the scalp flap. Water tight closures are neither necessary or practical and CSF leak is not a problem in our experience. Duraplasty should reduce the angle of bulge at the craniectomy edge thus reducing the acute kinking of cortical vessels. The need for creating vascular tunnels at the craniectomy to prevent the sequelae of vascular compromise is obviated. We harvest the fascia overlying the temporal muscle for dural reconstruction. By opening out the two layers of temporalis fascia a satisfactory but not watertight cover can be obtained.

Subdural clots beyond the margins of the craniectomy can be washed away with gentle saline irrigation. Grossly contused brain tissue is sucked out through a minimal corticectomy removing only grossly hemorrhagic debris. We desist from excision of potentially viable brain tissue to make space.

We routinely use a sub galeal closed suction drain which is removed after 48 hrs. The galea is closed with interrupted 2-0 polyglactin and the skin with a continuous suture of 2-0 polyglactin 'rapide'. The wound edges are smeared with Povidone Iodine ointment and a head bandage applied –which is removed at the time of drain removal.

Post operatively these patients are electively ventilated overnight. The next morning the GCS is re-evaluated after cessation of the propofol infusion. A decision to extubate the patient or to continue with elective ventilation is then taken. Patients who require elective ventilation for more than 5 days or those whose chest x-ray show features of consolidation by 72 hours post surgery are subjected to a tracheostomy to facilitate pulmonary toilet & easy weaning from the ventilator.

We routinely place the bone flap in the subcutaneous pocket of the anterior abdominal wall. The right upper quadrant is more commonly used by us – but the cosmetic result of a mid lower abdominal placement is superior but can occasionally confound the evaluation of bladder distension.

The bone flap placement is usually done after 6 weeks and before the completion of three months. During reconstruction, the bone flap is fixed with a titanium mini-plate system. We use modified periosteal flaps from the bone adjacent to the cranial defect to cover the screw construct (unpublished data).

Outcomes: There is a significant improvement in favorable outcomes in patients subjected to a decompressive craniectomy[18]. In the pediatric age group, decompressive craniectomy has been shown to reduce the rate of death & unfavorable outcomes. Class I evidence has been provided for this by a prospective randomized study from the Royal children's hospital of Melbourne. The percentage of favorable out comes was improved to 54% in the decompressive craniectomy group as compared to 14% in the standard treatment group.

Amongst adult patients, studies have shown a 61% incidence of favorable outcomes after decompressive craniectomy[19]. The American Brain Trauma Foundation guidelines have included decompressive craniectomy within 48hrs of injury as a treatment option for patients with refractory cerebral edema and intracranial hypertension. However the Cochrane database evaluation results are still equivocal[20].

In our own experience decompressive craniectomies have been associated with a 50% incidence of favorable outcomes.

Conclusion and Recommendations: The inclusion of decompressive craniectomy into the sequence of management of post traumatic brain edema is a useful addition to the therapeutic armamentarium. While managing a case of severe TBI is the initial steps are of resuscitation, with intravenous fluids resuscitation and oxygenation. Osmotic diuretics and antiepileptics are administered and the patient is shifted for a CT scan after ensuring air way protection and adequacy of ventilation. Patients with mass lesions (subdural hematomas, contusions and intracerebral hematomas), who merit surgical decompression are shifted directly to the operation theatre where an appropriately sized decompressive craniectomy and duraplasty are combined with the evacuation of the mass lesion.

Patients without a mass lesion – who merit ventilator support (GCS 8 or <8) are sedated and maximal ICP control measures initiated. ICP monitoring by a sub arachnoid bolt or a parenchymal sensor is initiated. If

the ICP continues to be above 25mm Hg, patients are taken up for a generous and appropriately placed decompressive craniectomy with a duraplasty.

Postoperatively if the flap bulge does not decrease over two to three weeks or if the neurological condition worsens in the absence of metabolic derangements, infection or fever, a CT scan is indicated to the exclude hydrocephalus. Patients who develop delayed hydrocephalus after a decompressive craniectomy merit a ventriculoperitoneal shunt.

In conclusion, we submit that decompressive craniectomy is an effective adjunct in the management of severe post traumatic brain edema. If it is done at the right time, it improves outcomes in patients with severe traumatic brain injury.

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