The Role of Surgical Intervention in TBI

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OUTLINE

TBI EPIDEMIOLOGY AND CRANIAL SURGERY RATES

In the United States, there were 2.5 million emergency department (ED) visits, hospitalizations, and deaths attributed to traumatic brain injuries (TBI) in 2010 alone, either as an isolated injury or in combination with extra-cranial injuries.[1] Approximately 2% of those patients (>50,000) died, this accounts for approximately 40% of all deaths from acute injuries in the United States.[1] The major causes of TBI-related hospitalizations were falls, assaults, and motor-vehicle traffic incidents.[1, 2] TBI also remains the most common cause of disability amongst people under the age of 40. An estimated 3.2 million–5.3 million persons in the United States are living with disability acquired from a TBI-related event.[3-5] Since 2007 the number of TBI-related ED visits have increased by 56%.[1] This increase did not apply for TBI-related hospitalizations and deaths. TBI-related crude mortality rates slightly decreased from 18.2 to 17.1 per 100,000 persons from 2007 to 2010.[1] Although the exact cause for this decrease has not been established, it is thought to follow a continued reduction in motor-vehicle traffic incidents. In addition, there have been advances in pre-hospital and neuro-intensive care in specialized trauma centers, all leading to improved care quality and health outcomes for TBI patients.[6] A study conducted using the National Trauma Data Bank (NTDB) demonstrated that craniotomies were performed in 3.6% of all head-injured patients.[7] Over 95% of head-injuries patients in the NTDB received conservative/non-operative management. However, the NTDB included patients with both mild and moderate head injuries, and the absolute number of emergency cranial surgical procedures has not been established firmly.[7] It remains important to track these rates in order to assess practice patterns, implementations of guidelines, and its impact on patient outcome.[7-9]

INVASIVE BRAIN MONITORING

Monitoring of intracranial pressure (ICP), clinical neurological examination and CT scanning, are currently the primary methods to guide treatment for patients with TBI during neurointensive care.[10, 11] Unconscious or unstable patients are often sedated, therefore limiting the utility of clinical examinations. In these cases, ICP monitoring has been traditionally used to guide management to maintain adequate cerebral perfusion and oxygenation, and avoid secondary injuries.[11, 12] The BEST TRIP study provided evidence that patients may be managed without ICP monitoring.[13] However, the Brain Trauma Foundation guidelines and a recent consensus conference held in Milan recommend ICP monitoring in salvageable severe TBI patients with abnormal CT finding (mass lesions, swelling, herniation, or compressed basal cisterns).[11, 14]

Measurement of ICP can be done in several ways. Many consider intraventricular catheters as the "gold standard" method of ICP monitoring.[11] It allows both measurement of ICP, and the possibility to treat raised ICP via drainage of CSF. Intraventricular catheters can be connected with fluid-coupled catheter to an external strain gauge, or available with an integrated micro strain gauge or fiber-optic tipped catheter. As with all intraventricular catheters, there is chance of drain-related infections that increases the longer a catheter is in place.[15-17] In addition, in a trauma setting it can be technically challenging to insert an intraventricular catheter in a patient with cerebral edema, midline shift, or small/compressed ventricles.[18]

Intraparenchymal ICP monitoring devices use fiber-optic catheters to measure the ICP without CSF diversion. Compared to intraventricular catheters, parenchymal monitors are a less invasive alternative to measure ICP and carry a lower risk of infection and hemorrhage.[19-21] However, this

method does not allow CSF drainage for therapeutic purposes. There are varying reports on the drift of parenchymal monitors, although this is not deemed to be a clinical concern[11, 22-24]. Subdural, subarachnoid and epidural monitors have also been describing and currently considered less accurate than intraventricular or intraparenchymal devices.[11] In TBI cases with mass lesions, it has been known that ICP is not transmitted equally throughout the intracranial space. Studies have suggested that expanding mass lesions are associated with ICP gradients, in particular acute subdural hematomas.[25] Differences have described greater that 10mmHg between hemispheres. Further research is needed to define the optimal ICP measurement location to guide ICP management for these cases.

Multimodality neuro-monitoring, including ICP, partial pressure of oxygen (PbtO2), and cerebral microdialysis can provide a more comprehensive monitoring of the injured brain than ICP monitoring alone.[10, 12, 26] It allows individualized management of secondary cerebral insults targeting patient-specific pathophysiology. Current cranial access devices enable multiple catheters and sensors to be transmitted into the brain parenchyma, to allow for ICP, cerebral microdialysis (monitoring of chemistry of the extracellular space), and PbtO2 (monitoring of cerebral oxygen metabolism) catheters to be monitored continuously at the bed-side.[27]

EVACUATION OF INTRACRANIAL HEMATOMAS

The role of surgery in traumatic intracranial hematomas is to prevent irreversible brain injury or death caused by hematoma expansion, raised ICP, and herniation of the brain.[28-30] An initial assessment of neurologic deficits, pupil abnormalities, degree of midline shift, hematoma volume, and the presence/severity of associated trauma is required to determine the necessity for

emergency cranial surgery. For neurosurgeons one of the most complicated decisions to make is whether or not moderate-sized mass lesions should be evacuated or observed. On the one hand surgical intervention might be unnecessary; on the other hand, neurological deterioration with possible secondary insults to the brain may negatively impact the patient's outcome. Current guidelines and recommendations are available, but principally drawn up by experts and the (limited) evidence that is available.[28-30]

EPIDURAL HEMATOMAS

Epidural hematomas (EDH) usually develop in young adults following traffic-related accidents, falls, and assaults.[29] In TBI patients the incidence of surgical and nonsurgical EDH cases has been estimated between 2.7 to 4%.[29] EDH are thought to result from a direct blow to head and are usually found on the same side impacted by the blow. Typically, the source of bleeding is arterial, following a trauma to the sphenoid or temporal bone with subsequent tearing of the middle meningeal artery and hematoma formation in the middle cranial fossa. Extradural hematomas may also occur in the frontal, occipital and vertex regions and are usually associated with the anterior ethmoidal artery, transverse or sigmoid sinuses, and superior sagittal sinus, respectively. EDHs originating from venous sources are thought to expand more slowly compared to their arterial counterparts.[31] EDH specific mortality has been described to be around 10 percent in adult patients.[29] The role of surgery is to prevent irreversible brain injury or death caused by hematoma expansion, raised ICP, and herniation of the brain. Patients presenting with (progressive) focal neurologic signs or symptoms and/or hematoma growth, have to be considered as an emergency case. Evidence and expert-based recommendations for evacuations of EDH recommend surgery for all adult patients with a hematoma volume >30 cm3 (>30 mL)

regardless of the GCS score, and comatose patients (GCS <9) with pupillary abnormalities.[29] Evacuation should be performed through a craniotomy window fashioned according to the location of the hematoma, providing adequate access to the hematoma margins. If the brain appears tight it is important to inspect the subdural space for additional clots. When the bone flap is replaced several tenting sutures should be placed to minimize the epidural space. Bone flaps are not generally left out for isolated EDH with no parenchymal injuries. Close observation and conservative management is appropriate for patients with no focal neurologic deficits, with a small hematoma (<30 cm3), a clot thickness <15 mm, and midline shift <5 mm on imaging.[29]

ACUTE SUBDURAL HEMATOMAS

Acute subdural hematomas (aSDH) usually develop following motor vehicle accidents (MVA), falls, or assaults.[28] In younger patients (18–40 yr) 56% of the ASDH were caused by MVA and only 12% were caused by falls. Unlike the young patients, those aged 65 years and older had in 22% of the cases an MVA and 56% had a fall.[28] Unlike EDH, the source of bleeding is usually venous caused by torn bridging veins under acceleration conditions, with arterial bleeding sources reported for approximately 20% to 30% of aSDH cases.[32, 33] Mortality for aSDH patient requiring surgery has been described to be between 15 to 60 percent.[28, 34-39]. Evidence and expert-based recommendations for evacuations of aSDH recommend surgery for all adult patients with a hematoma thickness >10 mm, midline shift >5 mm, GCS score decreased by ≥2 points from injury to hospital admission, and/or patients presenting with pupillary abnormalities.[28] As advanced age has been associated with increased rates of adverse outcome, age should be taken into account when deciding to perform surgery. TBI patients presenting with an aSDH are frequently accompanied by significant parenchymal injury and swelling on

imaging.[28, 38, 40] Patients with aSDHs that require surgery to remove the clot are treated either with a craniotomy or a decompressive craniectomy (DC). However, there is often uncertainty as to whether the bone flap should be replaced or not.[41] The RESCUE-ASDH trial is currently recruiting aSDH patients and aims to compare craniotomy versus DC for adult patients undergoing evacuation of an aSDH.[42] The results of this trial will inform surgical decision making in the management of aSDH patients. Close observation and conservative management is appropriate for patients that are neurologically stable, hematoma thickness <10 mm, midline shift <5 mm, no pupillary abnormalities, and no intracranial hypertension on ICP monitoring.[28] Conservatively managed aSDHs resolve gradually and are usually absorbed over weeks, although in elderly patients may turn into a chronic subdural hematoma.

TRAUMATIC INTRACEREBRAL HEMORRHAGE

Traumatic intracerebral hemorrhage (ICH) is also referred to as traumatic intraparenchymal hemorrhage and (hemorrhagic) contusion. Post-traumatic contusions are usually multiple and are located in the basal surface of the frontal and temporal lobes.[30] In the acute stages, the ICH consists of a (semi-)liquid mass of blood with surrounding edema. These mass lesions evolve over days and change consistency while edema begins to recede. Mortality secondary to traumatic ICH is related to the location and size of the lesion(s).[30] Surgical interventions are aimed at preventing secondary damage, brainstem compression and herniation of the brain. Unfortunately, the only trial investigating the role of early surgery versus initial conservative treatment to anticipate and prevent secondary damage in traumatic ICH was halted early.[43, 44] Although the evidence is limited due to the low sample size resulting from premature termination, it appears that the STITCH(Trauma) Trial observed reduced mortality with early

surgery.[44] However, it is important to note that patients in this trial were mostly recruited in resource-limited settings where ICP monitoring was not usually available. Current evidence and expert-based recommendations for evacuations of traumatic ICH involving the cerebral hemispheres recommend surgery for patients with focal lesions and the following indications; progressive neurological deterioration, medically refractory raised ICP, a hematoma volume >50 cm3 (>50ml), GCS score of 6 to 8 in a patient with a frontal or temporal hemorrhage >20 cm3 (>20ml) with either midline shift of > 5 mm and/or cisternal compression on CT scan.[30] Patients with diffuse injuries developing medically refractory post-traumatic cerebral edema and intracranial hypertension may be considered for a bifrontal DC within 48 hours of injury.[30] DCs may also be considered for patients with refractory intracranial hypertension and diffuse injuries with clinical and radiographic evidence for transtentorial herniation. Evacuation of a traumatic ICH in the posterior fossa is recommended when there is evidence of neurologic dysfunction/deterioration and significant mass effect on the basal cisterns, 4th ventricle, or signs of obstructive hydrocephalus.[45] Intensive monitoring and serial imaging is appropriate for patients with no focal neurologic deficits, and non-significant mass effect on imaging.

DECOMPRESSIVE CRANIECTOMY IN TBI

Management of refractory raised ICP following severe TBI consist of medical and surgical treatments.[46, 47] DC is generally considered a surgical treatment for patients with diffuse brain swelling or expanding contusions/hematomas refractory to medical treatment and impending herniation.[48, 49] In recent years, the role of DC has been discussed as a primary treatment in the acute phase, leaving out the bone flap after evacuation of a mass lesion, or as a second- or third-tier therapeutic measure for diffuse brain injury and edema, commonly named secondary or

protocol-driven DC. The expansion of a swollen brain outside the skull, can potentially lead to a reduction in ICP and risk of herniation. The physiological improvements described in severe TBI patients after DC, include improvement in brain tissue oxygenation, cerebral perfusion, and neurochemistry.[50-55] The risk of complications should also be considered as early or delayed complications can occur after DC.[48] Expansion of (contralateral) mass lesions, wound infections and healing problems, subdural or subgaleal collections, hydrocephalus, syndrome of the trephined, and complication related to the subsequent cranioplasty have been recognized as DC related complications.[56-58] As the risk of severe disability and death in severe TBI remains relatively high, a number of trials have explored the use of DC to improve patient outcomes.[42, 59, 60] However, defining the indications, timing, techniques, and optimal outcome measures for DC has proven to be difficult, and good quality evidence linking efficacy to outcome is lacking.[48]

DECOMPRESSIVE CRANIECTOMY METHODS

Decompressive craniectomy is an umbrella term for a group of procedures in which part of the skull is removed. In severe TBI the most frequently described DC procedures in adults are bifrontal DC and unilateral frontotemporoparietal craniectomy, also termed hemi(spheric)-craniectomy or unilateral DC.[48, 61, 62] For unilateral pathologies with midline shift and (potential) swelling, e.g. aSDH with parenchymal injuries, a hemi-craniectomy can be useful. Evidence and expert-based recommendations for adequate hemi-craniectomies suggest that the bone flap should be large with a minimum anteroposterior diameter of 11–12 cm,[40, 63] in order to achieve an adequate reduction of ICP and also reduce the risk of transcalvarial herniation that is associated with parenchymal injuries at the bone edge.[64, 65] Bifrontal DC is a treatment option for diffuse (bi-hemispheric) injuries with medically refractory intracranial hypertension.[48] A

bifrontal DC extends from the floor of the anterior cranial fossa to the coronal suture posteriorly and to the temporal floor bilaterally. A widely opened dura mater is required to allow the brain to sufficiently expand. Different techniques have been described for the dura (left open with onlay of hemostatic material, pericranium, or temporalis fascia, or closure with dural expansion grafts)[66-68] and sagittal sinus sectioning or sparing.[49] Studies have also described bilateral hemi-craniectomies as an approach for patients with diffuse injuries, although an improvement over the bifrontal DC approach has not been investigated.[62] For patients with temporal lesions or edema causing brainstem compression, extension of the DC to the floor of the middle cranial fossa is essential.

EVIDENCE BASE FOR DC IN TBI

The DECRA trial failed to found an improvement in functional outcome by performing early bifrontal DC over medical management for patients with diffuse TBI.[59] The study showed that patients treated with DC had shorter duration of ventilation and length of stay in the intensive care unit. The RESCUEicp trial aimed to examine the clinical and cost effectiveness of secondary DC (unilateral or bifrontal DC) for severe TBI patients with refractory intracranial hypertension as a last-tier therapy. The target sample size of 400 patients was achieved and the study is currently in the analysis / write-up phase, results are expected in 2016.[60] In contrast to the aforementioned trials, the RESCUE-ASDH trial is an ongoing randomized trial comparing primary unilateral DC to craniotomy (bone flap out versus bone flap replaced) for patients with aSDH.[42] Information from these studies will define the role of secondary and primary DC in future TBI treatment guidelines. On the basis of the current available evidence, neurosurgeons

and neuro-intensivists must weigh the potential risks and benefits faced by their individual patients when deciding to perform a DC.

EXTERNAL VENTRICULAR DRAINAGE IN TBI

External ventricular drainage does not only allow for measurement of ICP, but also drainage of CSF at the bedside to control raised ICP.[11] This method is relatively fast, minimally invasive, and effective, for patients with intracranial hypertension even without hydrocephalus.[69] However, this procedure also carries certain risks and potential complications.[15-17] Complications of EVD placement include the risk of infection, increasing with the length of drainage, and the risk of hemorrhages, increased by post-traumatic clotting derangements. Image guidance can facilitate safe placement of the catheter in TBI patients with diffuse brain swelling that often have small ventricles. Optical and electromagnetic neuro-navigation systems can be used, with the latter not requiring rigid pinning cranial fixation.[70] There is no class I evidence on the use of EVD as a first-tier or second-tier intervention in severe TBI patients, and there is also clinical uncertainty regarding continuous drainage of CSF (open EVD system) versus intermittent opening as necessary to drain CSF (closed EVD system).[71] While the latter method would allow for real-time measurement of ICP, it can potentially expose patients to elevations of ICP between drainage periods. The theoretical benefit of an open EVD system is tighter and stable ICP control, however, there is a risk of overdraining with potential subsequent collapse of the ventricles.

SURGICAL MANAGEMENT OF SKULL FRACTURES

Skull fractures most frequently involve the parietal bone, followed by the temporal, occipital, and frontal bones.[72] TBI patients usually present with linear fractures, and less frequently with depressed and skull base fractures. The force of the trauma to the skull required to cause fractures is significant, therefore patients are at significant risk of underlying brain injury. Evidence and expert-based recommendations for skull fractures recommend elevation and washout for patients with open skull fractures depressed more than the thickness of the cranium or more than 5 mm below the adjacent inner table.[73] The rationale is reducing the risk of infection for these cases with early surgery, especially in the presence of dural tears, pneumocephalus, frontal sinus involvement, or contaminated wounds. Emergent surgery is also indicated for fractures with an underlying (expanding) hematoma. Elevation of the fracture will also improve cosmesis for cases with significant displacement of the bone. Reconstruction can usually be achieved by using the bone fragments; if this is not feasible, implants can be used to cover the skull defect.[74] Antibiotics are usually administered to patients with open skull fractures; currently routine prophylaxis for all skull fractures is not supported by the available evidence.[73, 75, 76]

POST-TRAUMATIC CSF LEAKS

In TBI patients it is estimated that CSF leaks occur in approximately 2% of patients.[77-79] Fractures carrying a high risk of CSF leaks are those involving the frontal or ethmoidal sinuses, and the temporal bone.[78, 80] The majority of CSF leaks are self-limiting and resolve

spontaneously within days.[77, 81, 82] Surgical interventions are aimed at reducing the symptoms and risk of infection in cases with persistent CSF leaks/ fistulas. Studies have reported infection rates between 7% and 30% for TBI patients with CSF leaks, with each day of leakage increasing the risk of ascending intracranial infection.[77, 83-87] Lumbar drainage has been recognized as a treatment option for TBI patient with CSF leaks, while also facilitating a route to administer intrathecal fluorescein for diagnostic purposes.[85, 88] In addition, lumbar drains are also used to relieve CSF pressure after surgical repairs to increase the success rate. The relative importance of antibiotic prophylaxis as well as the selection of the "true" high-risk candidates remain to be defined.[89] However, the Center for Disease Control and Prevention recommends either the pneumococcal conjugate vaccine (PCV13) or the pneumococcal polysaccharide vaccine (PPSV23) for patients with CSF leaks.[90]

POST-TRAUMATIC HYDROCEPHALUS

Post-traumatic hydrocephalus (PTH) is a complication of TBI and studies have reported clinical improvement after permanent CSF diversion.[91] Patients who suffered TBI and under active follow-up are usually monitored for PTH, which can present as worsening neurological status or lack of improvement. It is important to recognize and treat PTH, since it could both impact morbidity and mortality if left untreated.[92-94] The incidence of PTH has been reported to range between 0.7–51.4%.[91, 95, 96] However, it is often difficult to determine whether ventriculomegaly observed post-TBI is related to atrophy or hydrocephalus; computerized CSF infusion studies have been reported to be useful in distinguishing between the two different processes.[97, 98] Selection of patient benefiting from permanent CSF diversion is important, since shunting is also associated with significant complications. No clear guidelines exist for PTH treatment, however

adjustable or flow-regulated ventriculo-peritoneal (VP) shunts are most commonly described as the preferred choice of shunting to reduce the risk of overdrainage. PTH has been reported as a a relative contraindication to endoscopic third-ventriculostomy (ETV) [99], however this notion has been challenged by others.[100] It is difficult to predict the response of CSF diversion in PTH, since these patients often have comorbidities and significant underlying brain injury. Studies have also suggested that DC is a risk factor for hydrocephalus[48, 96] whereas others do not support this hypothesis.[101] Hydrocephalus has been described in TBI patients undergoing DC, ranging in case series between 0-88.2%.[102] It is thought that CSF malabsorption or obstructed flow are the cause of post-DC hydrocephalus. However, the current case series are limited by their design and heterogeneity of criteria used to diagnose hydrocephalus.

CRANIOPLASTY

Cranioplasty is the surgical reconstruction of a bone defect after a previous operation, usually DC, or due to the skull injury. A cranioplasty is usually recommended for protection of the underlying brain that is left vulnerable to damage with a skull defect and also for cosmetic issues that might have psychological and social consequences for the patient.[48] The cranioplasty can also facilitate neurological rehabilitation and may also improve neurological function, as observed in patients with syndrome of the trephined.[103-106] However, this procedure is associated with challenging complications; the most discussed are wound-healing problems and implant related infections.[107-109] Complications can also be specific to the type of material chosen, e.g. resorption of the bone with autologous bone. The wide range of techniques, graft materials (autologous bone, metal or synthetic) and timing of reconstruction (1 – 12 months) discussed in

the literature reflects the lack of consensus and good quality evidence to guide neurosurgeons when planning cranial reconstruction.[106, 110-115] There is also controversy in relation to timing, performing the cranioplasty too early can risk infections or the development of devitalized autograft. In cases of an (suspected) infected area it is important to wait a certain amount of time (can be as long as one year) to avoid infection of the implant. Also, it is not clear if early reconstruction can improve the neurological recovery of TBI patients .[106, 110-112, 114, 115]

SUMMARY

The general consensus to optimize the care for severe TBI patients is management at specialized neurotrauma centers with neurosurgical and neurocritical care support and the use of guidelines-based standardized protocols. Over the past decade significant efforts have been made to define neurotrauma treatment guidelines. This progress reflects the effort that has been made to examine the evidence that is available to us together with widely held expert opinions. It is however important to recognize the heterogeneity of TBI and that the 'one size fits all approach' may not always be appropriate for these patients. Knowledge synthesis activities in neurotrauma are important to define future research agendas. Clinical and research advances have influenced the field of neurotrauma as it continues to mature into a distinct subspecialty of neurosurgery.

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