Introduction:

Trauma is one of the leading cause of death across the globe including India. Among the numerous causes of death amidst trauma victims; traumatic brain injury (TBI) contributes significantly. TBI involves men, women and children of all age group and injuries can be sustained during a trip or fall, vehicular accident, fall from height, physical assault or a sports related injury.

TBI has been defined in many different ways. A good working definition is that **it is a disruption or alteration of brain structure or function caused by external mechanical forces**. The spectrum of injury to the brain parenchyma may be of variable severity and may be transient or permanent impairment of the normal function. The causative factors are diverse and include rapid acceleration or deceleration, direct compression, penetration and physical disruption of brain tissue, blast and other complex mechanisms, and various combinations of these and other etiologies. Mild TBI may be present with no gross abnormalities on imaging studies. At the other end of the spectrum, more severe injuries may be associated with large contusions, traumatic hematomas, or other immediately life-threatening structural lesions.

Types of Injury:

TBI is classified by mechanism, severity, and morphology.

Primary and secondary Injury:

TBI is a dynamic process. The transfer of energy during the injury is the primary injury, but this injury sets into motion a cascade of molecular, cellular, tissue level, and immune system responses that contribute to secondary injury. These secondary injuries include hypoxemia, ischemia, seizures, fever, hypoglycemia, and other systemic (non-neurologic) events that can directly impact the brain’s ability to respond to a primary insult. Secondary injury does not end, however, with hospital discharge; it can continue to months, years, or perhaps decades as the secondary long-term effects of TBI become manifest. The essential principle is recognition that TBI is a process, not an event.

Approach to a victim with TBI:

Prevention of secondary injury is the primary goal of prehospital and early in-

hospital management. The pre-hospital management of TBI patients by trained paramedical personnel is critical to outcome. Fifty percent of deaths from TBI occur within the first few hours of injury. Basic life support maneuvers are directed at establishing and maintaining a patent airway, achieving adequate ventilation and oxygenation, ensuring adequate circulation, and stabilization of the cervical spine. If any of these are compromised, secondary injury proceeds at an accelerated pace, and the risk of mortality increases.

Airway:

Studies done in the past showed no mortality benefits for victims with TBI who were secured with advance airway by paramedics in the field. Rather supra glottic airway devises are preferred over ET intubation. In the patient who is breathing spontaneously, the airway should be monitored while maintaining spinal stabilization. Supplemental oxygen should be delivered if required by face-mask. If easily tolerated (considering C-spine precautions), insertion of an oral airway device is helpful to prevent the tongue from occluding the airway.

Breathing:

Normal breathing should be maintained with a target goal of pCO2 of 35–40 mmHg. End-tidal CO2 (ETCO2) can be used for monitoring in the pre-hospital environment. If there are clinical signs of herniation, then hyperventilation (ETCO2 28–35 mm Hg) can be used as a temporary measure. If ETCO2 is not available and patient is receiving assisted ventilation goal respiratory rate should be 10–14 breaths/min. Oxygenation via pulse oximetry, should be monitored continuously with a goal SaO2 of >90%. Supplemental oxygen should be used as needed to maintain this parameter.

Circulation:

Cerebral perfusion is linked directly with mean arterial pressure. Targeted BP for adults, is to maintain systolic BP > 100 mmHg. For children, systolic BP should be maintained > 5th percentile for age (70 mmHg + age X 2). Hypotensive patients should be treated with rapid infusion of isotonic fluids (20–40 ml/kg of normal saline). Hypotonic fluids, such as D5W or ½ NS should be avoided, as they may exacerbate brain edema.

During the entire process from extrication of the victim from the site of event till

reaching the Emergency Department there are several factors that has to be monitored concomitantly along with ABC to prevent secondary brain damage.

- In line stabilization of the spine until radiological clearance
- Prevent hypoglycemia
- Assess and reassess GCS
- Pupillary size and reactivity

Initial Emergency Department/Hospital Management:

Once the victim reaches definitive care a quick primary survey followed by a detailed secondary survey should be done to exclude any masked injury. Two large bore peripheral lines should be established to administer drug, I.V fluids or even blood products. Another thing that has to be kept in mind is to prevent hypothermia at all time. Whenever indicated rapid sequence intubation(RSI) to be performed. RSI drugs should be chosen carefully as it can precipitate hypotension and hamper cerebral perfusion. Not very unusual that patient with head trauma are left without any other major injuries. Any fracture has to be immobilize at the earliest to arrest bleeding, pelvic binders for suspected pelvic fractures. A FAST exam can be performed to evaluate for possible internal hemorrhage. Evaluation for coagulopathy should be part of standard laboratory tests, and anticoagulant reversal should be administered if required.

Imaging:

Head computed tomography (CT) remains the emergency neuroimaging modality of choice because of rapidity and its availability in most centers globally. The primary purpose of the initial head CT is to identify any hemorrhagic lesions that require surgery. Usually an extra-axial hematoma (extra- or subdural) >1 cm in thickness, an intra-parenchymal hematoma >20 cc in volume, and a >5 mm midline shift associated with a hematoma are to be considered for surgical intervention.

Decompressive Craniectomy:

Like mechanical injury elsewhere in the body, cerebral edema ensues following TBI from a varied combination of several mechanisms associated with primary and secondary injury. Since brain is enclosed within the rigid skull, as pressure within the cranium increases rapidly, brain tissue displacement can happen which may lead to cerebral herniation, resulting in disability or death. Decompressive

craniotomy is the term that describes a procedure whereby a portion of the skull is removed surgically. Although it sounds to be practical to relive raised ICP but this procedure is reserved for cases with refractory ICH ((>25 mm Hg for 1 to 12 hours) not responding to other modalities for lowering ICP.

Prophylactic Hypothermia:

Hypothermia is a well recognized treatment modality to preserve cellular function following any insult to neuronal tissue. This has also been added in the care bundle in post cardiac arrest care. Added benefit is that hypothermia also reduces ICP. But large studies failed to prove its beneficial in TBI and is therefore not recommended.

Hyperosmolar Therapy:

One of the most consistent part in management of TBI is intravenous administration of hyperosmolar agents for lowering intracranial hypertension and herniation syndromes. Both mannitol and hypertonic saline are routinely employed hyperosmolar agents. However, the optimal agent, their optimal means of administration (i.e., dose and bolus vs. continuous infusion), and their precise mechanisms of action continue to be under investigation. Specific situation demands the use of either agent. Hypertonic saline administration may be hazardous for a hyponatremic patient. Although mannitol can be used as a resuscitation fluid, its eventual diuretic effect is undesirable in hypotensive patients and attention needs to be paid to replacing intravascular volume loss. Both these agents work to reduce intracranial pressure, partly by reducing blood viscosity, leading to improved microcirculatory flow of blood constituents and consequent constriction of the pial arterioles, resulting in decreased cerebral blood volume and intracranial pressure.

Cerebrospinal Fluid Drainage:

Role of external ventricular drainage (EVD) systems in patients with severe traumatic brain injury (TBI) remains a controversial topic. Although EVD can be both therapeutic (CSF drainage) as well as diagnostic (ICP measuring). While in pediatric patient shows marked improvement in outcome such results are not seen in adults because of lack of supportive evidences.

Ventilation Therapies:

Patients with severe traumatic brain injury (TBI) require definitive airway protection as they are vulnerable to pulmonary aspiration and respiratory failure. Under normal conditions, PaCO2 is the most powerful determinant of cerebral blood flow (CBF) and, between a range of 20 mm Hg and 80 mm Hg, CBF is linearly responsive to PaCO2. Cerebral blood flow is important in meeting the brain's metabolic demands. Low PaCO2, therefore, results in low CBF and may result in cerebral ischemia while high PaCO2 levels can result in cerebral hyperemia and high intracranial pressure. Therefore, transient hyperventilation is reserved to treat cerebral herniation and elevated intracranial pressure (ICP). It should be avoided during the first 24 hours after injury when cerebral blood flow (CBF) is often critically reduced. If hyperventilation is used, jugular venous oxygen saturation (SjO2) or brain tissue O2 partial pressure (BtpO2) measurements are recommended to monitor oxygen delivery.

Anesthetics, Analgesics and Sedatives:

Sedation and analgesics plays a pivotal role in management of RBI. It helps to lower ICP, presumably by preventing unnecessary movement, coughing, and straining against tubes as well as suppression of metabolism and alteration of cerebral vascular tone. Agents like barbiturates, Propofol has their own adverse effects and should be used with caution.

Steroids:

Although used previously use of steroids is not recommended in TBI.

Nutrition:

Like any other disease condition, the daily caloric requirement has to be met for favorable outcome. Feeding patients to attain basal caloric replacement at least by the fifth day and, at most, by the seventh day post-injury is recommended to decrease mortality. Trans-gastric jejunal feeding is recommended to reduce the incidence of ventilator-associated pneumonia.

Infection Prophylaxis:

Staying in hospital per se is a risk factor to acquire infection. Over and above TBI

patients are susceptibility to infection because of use mechanical ventilation, urinary catheter, invasive monitoring and EVD. There is insufficient evidence to support the use of prophylactic antibiotics, however there is definitely a benefit in preventing infection by early tracheostomy-to reduce mechanical ventilation days when the overall benefit is felt to outweigh the complications associated with such a procedure and use of antibiotic impregnated catheters.

Deep Vein Thrombosis Prophylaxis:

Patients with TBI are at significant risk for developing venous thromboembolism (VTE). Severe TBI patients can be at significant risk for VTE due to hypercoagulability resulting from the primary brain injury, prolonged periods of immobilization, and focal motor deficits. Low molecular weight heparin (LMWH) or low-dose unfractionated heparin may be used in combination with mechanical prophylaxis. However, there is an increased risk for expansion of intracranial hemorrhage.

Seizure Prophylaxis:

Seizure are not uncommon following TBI. Based upon onset it may be early and late post traumatic seizures (PTS) depending on occurrence within or beyond 7 days post TBI. Post-traumatic epilepsy (PTE) is defined as recurrent seizures more than 7 days following injury. The risk factors for early PTS include: GCS score of ≤10; immediate seizures; post-traumatic amnesia lasting longer than 30 minutes; linear or depressed skull fracture; penetrating head injury; subdural, epidural, or intracerebral hematoma; cortical contusion; age ≤65 years; or chronic alcoholism. Levetiracetam appears to be increasing in use for seizure prophylaxis following TBI. Although phenytoin can be given for early (7 days) PTS it along with valproate is not recommended for late PTS.

Intracranial Pressure Monitoring:

Once stabilized the patient with TBI should be constantly re-evaluated for any rise in ICP. Intracranial pressure should be monitored in all salvageable patients with a severe traumatic brain injury (TBI) (GCS 3-8 after resuscitation) and an abnormal computed tomography (CT) scan. An abnormal CT scan of the head is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns. ICP monitoring is indicated in patients with severe TBI with a normal CT scan if two or more of the following features are noted at admission: age over 40 years,

unilateral or bilateral motor posturing, or systolic blood pressure (BP) <90 mm Hg.

Cerebral Perfusion Pressure Monitoring:

Cerebral perfusion pressure (CPP) is defined as the pressure gradient across the cerebral vascular bed, between blood inflow and outflow. Inflow pressure is taken as mean arterial pressure (MAP), which by convention is calibrated to the level of the right atrium of the heart. In normal physiology the outflow or downstream pressure is the jugular venous pressure (JVP), which is also calibrated to the level of the right atrium. In patients with TBI available data support maintaining CPP 60-70 mmHg in adult to prevent cerebral ischemia.

Conclusion:

Although there are various guidelines regarding management of TBI but the various components addressed by such guidelines needs further evidences and larger study population to cause a paradigm shift in the assessment, diagnosis, treatment, and prognosis of brain trauma.

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