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Intensive Care Unit Management of Traumatic Brain Injury Patients

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Head injury or traumatic brain injury (TBI) is considered an instant epidemic in the developed world. Head injuries account for one quarter to a third of all trauma-related deaths. According to the Global Status Report on Road Safety 2018, more than 1.35 million people were killed in a single year. Out of these, 90% of the casualties occurred in developing countries, and India contributes about 11% of the total share. Until the last decade, cardiac disease, cancer, and road traffic accidents were considered the leading causes of death in our country. It is predicted that if the incidence of Road Traffic Accidents continues at the present rate, then by the end of 2025, the head injury will become the most common cause of death worldwide. The head injury patients pose a unique challenge to the treating physicians in the early period of hospitalization and the later duration of follow-up. The primary role of intensive care management is preventing and treating intracranial hypertension, which causes derangement in cerebral perfusion pressure (CPP), thereby preventing secondary brain injury. In the present review, we discuss the effective management of TBI in the critical care unit.

Keywords: Critical care; Brain edema; Ischemia; Brain Injuries, traumatic; Thromboembolism

INTRODUCTION

The goal of intensive care unit (ICU) admission is to implement various strategies to prevent secondary injury due to hypoxia, cerebral hypoperfusion, metabolic stress. It involves optimizing general systemic physiology and disease-specific management[1,2]. All traumatic brain injury (TBI) patients must receive daily ICU care similar to other non-TBI patients in intensive care. Head elevation of 30° would improve cerebral perfusion pressure (CPP) by reducing intracranial pressure (ICP). It also reduces the risk of ventilator-associated pneumonia (VAP). A neutral position of the head and neck would maintain cerebral venous drainage. Avoiding any kind of compression around the neck (e.g., tight cervical collar, tight fixation of the endotracheal tube) would reduce cerebral venous drainage. Ensure periodic change in patients' position to avoid bedsore and eye care, oral care & physiotherapy. Standard VAP and central line bundle should be followed to prevent infection[3].

RESPIRATORY CARE

The airway protective reflexes are blunted in TBI patients due to decreased level of consciousness. Patients are at high risk of aspiration and other respiratory complications like pneumonia and acute respiratory distress syndrome (ARDS)[4].

Respiratory system complications in TBI patients may be related to direct thoracic injuries such as pneumothorax, haemothorax, flail chest lung contusion. Associated cervical spine injury is another most common reason for ventilatory support. The goal of mechanical ventilation is to prevent aspiration pneumonia, reduce breathing work, and prevent secondary brain injuries associated with hypoxemia/hypercapnia. However, no specific ventilatory strategies have not been established for TBI patients. Individualised ventilation strategies are essential for good patient outcomes[5,6]. The controlled mode (either volume or pressure control) ventilation may be selected during the initial phase and then changed to an assisted mode/pressure support mode as the patient's clinical conditions improve. Laryngoscopy and tracheal intubation cause sympathetic stimulation, leading to tachycardiac, hypertension, and increased intracranial pressure. The sedative/hypnotic drugs agents selected for intubation should provide optimal intubating conditions with minimum hemodynamic disturbances. The most common drugs are etomidate and propofol. Adequate preoxygenation administration of fluids/ vasopressors during intubation helps prevent hypoxia and severe hypotension[7-9]. Hyperventilation should be used judicially for the treatment of refractory intracranial hypertension. Use any ventilatory setting to achieve normocapnia ($PaCO_2 \approx 35$-mmHg). Both hypercarbia and severe hypocarbia are associated with the potential risk of ischemic side effects on the injured brain[10-12]. Indication and timing for tracheostomy in TBI patients are still debated. Tracheostomy in TBI patients helps to minimise the duration of a ventilator, ICU stay, risk of ventilator-associated lung injuries, requirement for sedation. The TBI patients with one or more failed weaning trials should be considered for tracheostomy placement[5,13,14].

CIRCULATORY SYSTEM CARE

Cardiac dysfunction due to paroxysmal sympathetic hyperactivity is common (8–33%) in moderate to severe TBI patients. This may manifest with ST-T wave, QT prolongation in ECG changes, reduced ventricular function and regional wall motion abnormality. Though there is no specific therapy, non-pharmacological methods include avoiding external stimuli, maintaining hydration, and correcting electrolyte imbalance. Furthermore, pharmacological strategies such as opioid beta-blocker can be used for paroxysmal sympathetic hyperactivity[15,16]. Associated spinal cord injury enhances the risk of cardiovascular disturbances due to impairment of sympathetic tone[17,18]. Adequate cerebral blood flow depends on Cerebral perfusion pressure. Cerebral perfusion pressure (CPP), the difference between mean arterial pressure and intracranial pressure (or Central venous pressure), remains constant over a wide range of systemic blood pressures in a healthy patient. However, hemodynamic disturbances such as hypo/hypertension in TBI patients can compromise cerebral perfusion pressure. Hypotension (SBP < 90 mm Hg or MAP < 65 mm Hg) is an independent predictor for secondary systemic brain insult, thus associated with increased mortality following TBI. Intravascular volume depletion due to haemorrhage, polyuria secondary to diabetes insipidus, spinal cord injury with spinal shock is the common cause of hypotension in TBI patients. Intravenous fluid administration and vasopressors should be used for resuscitation[19,20]. Ideal intravenous fluid for TBI patients is still controversial. Nevertheless, hypotonic fluids should be avoided to prevent volume overload and cerebral oedema[21,22]. Hypertension (SBP > 160 mm Hg or MAP > 110 mm Hg) can aggravate brain oedema and ischemia.

In TBI normotension/normal CPP is preferred , but in several situations¬ there may be hypertension (which is the normal physiological response for maintaining the CPP and it also indicates preserved autoregulation to maintain CPP). This normal physiological raise in blood pressure can be tolerated. Induced hypertension should not be considered for patients without ICP monitoring as aggressive treatment of blood pressure in patients with raised ICP may worsen morbidity or even death. The CPP is the force is the primary determinant of cerebral blood flow. The CPP is defined as the difference between the mean arterial pressure (MAP) and ICP (CPP = MAP – ICP). According to the Brain Trauma Foundation (BTF) guidelines CPP should be maintained at 60-70 mmHg[23].

NUTRITION AND GLUCOSE CONTROL

Early nutrition in TBI patients has shown beneficial effects. It has shown a reduction in mortality, overall poor outcome and infections. Malnutrition increases the mortality rate in TBI patients. Unless contraindicated enteral feeding is preferred in TBI patients, it is physiologic with advantages such as preservation of the gastro-intestinal tract functions, immunological gut barrier, and mucosal integrity[24,25]. TBI patients may have gastric delays due to increased ICP and opiates use for sedation. Prokinetic agents such as metoclopramide improve tolerance. Indirect calorimetry/Harris Benedict's equation/ weight-based formula, i.e., 25–30 Cal per kg

body weight per day, can be used to calculate the caloric requirement. The BTF guidelines support achieving caloric requirements by day seven and trans gastric jejunal feeding[23]. Stress-induced hyperglycaemia is expected after the head injury, leading to secondary brain injury. Hyperglycaemia is associated with an increase in release of pro-inflammatory cytokines, which closely correlate with poor neurological outcome. Similarly, episodes of hypoglycaemia are also detrimental[26]. Blood glucose levels within tight limits (intensive insulin therapy, 81-108 mg/dl) are also controversial because they do not improve neurologic outcome/ mortality/infectious complications. Tight glucose control is associated with increased hypoglycaemic incidences. The blood glucose target in TBI patients is still not defined. A moderate approach to glucose control to 120–180 mg/dL seems to be appropriate [27].

SEDATION & ANALGESIA

The principle of sedation and analgesia is to ensure patient comfort and relieve pain. TBI patients also limit elevations of ICP, discomfort, cough/ pain, and decrease O_2 consumption and CO_2 production. Mechanical ventilation, any ICU procedures/ surgical interventions, nursing care are potential sources of pain[28]. Careful selection of sedation-analgesia drugs for TBI patients is essential for good patient outcomes. TBI patients need daily neurological assessments, frequently confounded by sedation. The ideal sedative/analgesics for TBI patients should have a rapid onset and offset, easy titration and predictable clearance, with no/ minimum effect on ICP and $CMRO_2$ and the cardiovascular system. The most common drugs used are fentanyl, propofol, midazolam and dexmedetomidine. These drugs can be used as a single agent or combination, either continuous infusions or intermittent boluses.

SEIZURE PROPHYLAXIS

Prophylaxis should be considered early post-traumatic seizures (presentation within seven days of injury). As per BTF, phenytoin is recommended for early post-traumatic seizures[23]. The current guidelines do not support antiepileptic drug administration for prevention of late post-traumatic seizures. Though side-effect profiles favour levetiracetam, both levetiracetam and phenytoin are comparable in efficacy, mortality rate and adverse reactions if used for early seizure prophylaxis. Currently, there is insufficient evidence to recommend levetiracetam over phenytoin[29-31].

SUPPORTIVE THERAPY FOR HEAD INJURY PATIENTS

Stress ulcer prophylaxis

An imbalance between mucosal protection and gastric acid production is responsible for stress ulcers in TBI patients. These patients have increased acid secretion due to stress-triggered vagal stimulation of the stomach through the CNS pathway and impaired mucosal protection due to compromised mucosal microcirculation[32,33]. Both proton pump inhibitors and histamine-2 receptor antagonists reduce the incidence of stress ulcers in trauma patients. The superiority of one drug over another is not known[34].

Deep venous thrombosis prophylaxis

Severe TBI patients are at risk of developing venous thromboembolic events (VTE), including deep venous thrombosis (DVT) and pulmonary embolism, venous stasis, hypercoagulability due to tissue injury. Approximately 20% of TBI patients treated in ICU have shown features of VTE. Old age, obesity and severity of TBI increase the risk of VTE in TBI patients[35,36]. TBI patients who receive pharmacologic prophylaxis have a lower incidence of VTE and a lower incidence of worsening haemorrhage. Pharmacologic prophylaxis is also safe in patients with intracranial pressure monitors[37]. Pharmacological DVT prophylaxis and mechanical prophylaxis are recommended for TBI patients if the injury is stable. However, there is insufficient evidence for timing, dose, or agent[23].

Hypothermia

Hypothermia (32°C to 34°C) reduces cerebral metabolic rate, preserves tissues in the face of metabolic challenge and decreases ICP, there is a lack of evidence to support prophylactic hypothermia; though it is not a standard therapy currently , but it can be one of the option in the multimodality approach as supported by TBI guidelines[1]. The use of hypothermia is not a standard recommendation in the management of TBI patients[23]. There is insignificant outcomes benefit in patients with TBI treated with hypothermia. However, fever should be aggressively managed in severe TBI patients[38].

Steroids

Steroids are not recommended to reduce ICP and associated mortality in head injury patients[23]. The CRASH (Corticosteroid Randomization after Significant Head Injury) trial has shown unfavourable outcomes in severe TBI patients if methylprednisolone was used[39].

MANAGEMENT OF INTRACRANIAL PRESSURE

Normal ICP is between 5 mm Hg to 15 mm Hg. Elevated ICP or intracranial hypertension is defined as a sustained ICP > 20 mm Hg. So measures to control ICP should be initiated with intracranial pressure (ICP) thresholds above 20 mm Hg. Ideally, ICP values, clinical findings and brain CT findings should be used to determine the need to treat raised ICP[40]. Intracranial hypertension can reduce cerebral perfusion pressure (CPP). Before initiating ICP targeted therapies the other treatable causes of raised ICP should be addressed. These are position of patient, neck flexion, endotracheal tube block/ventricular dyssynergy, pain, blocked urinary catheter, intracranial hematoma, etc. Controlling raised ICP depends on the underlying cause. Medical treatment for raised ICP includes head-end elevation, IV mannitol [mannitol 20% (0.25–1.5 g/kg IV bolus)], hypertonic saline [3% infusion], transient hyperventilation, barbiturates, and, if ICP remains refractory, sedation, endotracheal intubation, mechanical ventilation, and neuromuscular paralysis. Surgical treatment of choice includes CSF drainage- EVD (External Ventricular Drainage) and decompression of a surgical lesion, like intracranial hematoma (ICH), large infarct or tumor, if the patient's condition is deemed salvageable. Treating ICP without monitoring carries risks where prolonged hyperventilation worsens the outcome. It significantly reduces blood flow to the brain, proven by jugular venous oxygen saturation monitoring. Prophylactic paralysis increases pneumonia and ICU stay[41]. So ICP monitoring helps guide therapy and prognosticating patient outcome.

Guidelines to monitor ICP

According to BTF guidelines. ICP monitoring in Traumatic Brain Injury (TBI) patients can be done if 42);

1. CS is 3-8 after resuscitation with abnormal CT brain findings [ICH (Intracerebral hematoma), Brain edema, Herniation or compressed basal cisterns] [level II evidence] Or
2. GCS is 3-8 who has no abnormal CT findings with two of the three following criteria [level III evidence]
- Unilateral/bilateral posturing (flexor or extensor posturing)
- Age ≥ 40 years
- Hypotension

ICP monitoring devices based on its action mechanism include fluid coupled, non-fluid coupled, fibre optic and pneumatic devices[43]. ICP monitors based on anatomical locations include – intraventricular access transducers, parenchymal transducers, subdural transducers and epidural transducers.

Bratton et al. ranked ICP monitoring technology based on accuracy, reliability and cost[44]. They are as follows-1st – Fluid coupled intraventricular catheter (EVD) with external strain gauge

2nd – Fluid coupled intraventricular catheter with macrostrain gauge at catheter tip (or) fibre optic device
3rd – Parenchymal transducers
4th – subdural devices
5th – subarachnoid fluid coupled devices
6th – Epidural devices

Current concepts in the management of TBI

Major aspects which needs to the addressed in the current era in the management of TBI patients include (a) Moderate hypothermia (32 degree Celsius to 34 degree Celsius) which is attained by cold saline gastric lavage, cooling blankets and prevention of shivering by giving paralytic agents. (b) Barbiturate coma achieved in patients with severe TBI in whom ICP is uncontrollable with available standard treatment modalities. The hemodynamically stability is considered as the prerequisite for the commencement of this therapy. (c) Osmotherapy with hypertonic saline infusion to raise the serum sodium upto 150 mEq/L. The hypertonic saline is responsible for decreasing the cerebral edema by causing the egress of cellular fluid to the extracellular space. (d) In case of clinical signs of herniation with uncontrolled ICP, the immediate decompressive craniectomy is the standard recommendation in order to mitigate the malignant cerebral edema.

Management of specific post traumatic conditions

Surgical management of specific brain injuries like an extradural hematoma, subdural hematoma, parenchymal lesion and depressed cranial fractures depends on proper indication and timing of intervention[45].

Extradural hematoma

An Extradural hematoma is an acute emergency in neurosurgical practice. The indication of surgery depends on the volume of hematoma (> 30cc) regardless of GCS (Fig. 1). If the volume is less than 30 cc or midline shift less than 5mm or maximum thickness less than 15 mm or GCS greater than 8, patients may be managed with close neuromonitoring. Patients with an acute extradural hematoma or GCS < 9 or pupillary asymmetry should undergo surgical intervention as soon as possible[46].

Subdural hematoma

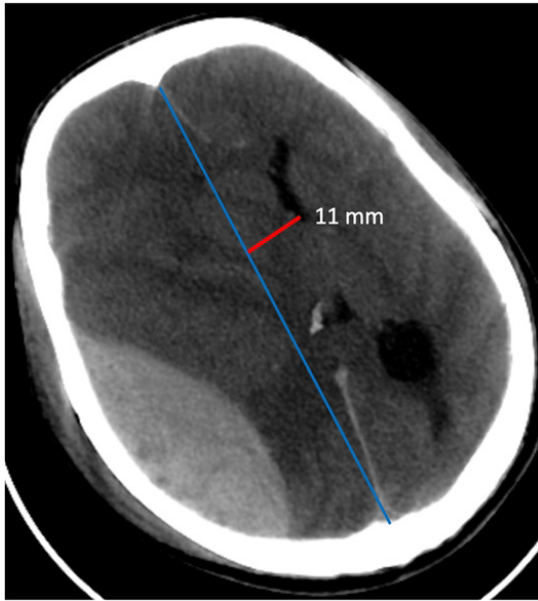The indication for surgery in subdural hematoma depends on

Fig. 1. Computerised tomography scan of an individual suggestive of right parietal biconvex hyperdense collection of acute blood suggestive of acute extradural hematoma. The blood clot is causing significant midline shift as indicated by the red line (11 mm).
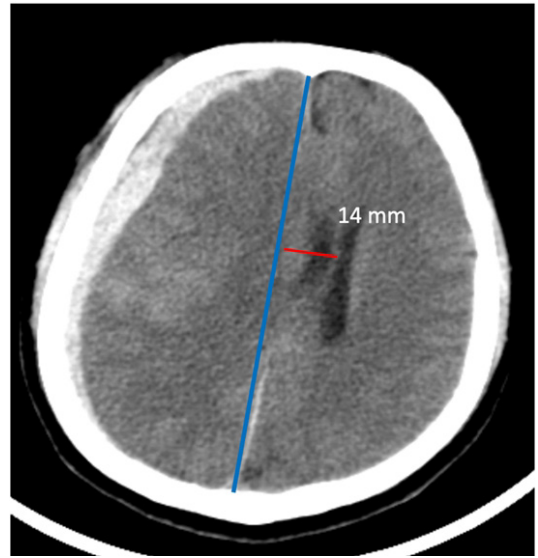

Fig. 2. Computerised tomography scan suggestive of convexo concave hyper density in the right fronto parietal region suggestive of acute subdural hematoma. The lesion is causing a significant midline shift of 14 mm as pointed out with red line.

Fig. 3. The axial cut of computerised tomography scan suggestive of significant hyper density in the bilateral frontal lobes with surrounding hypodensity suggestive of edema. This bleed in the brain parenchyma reflects cerebral contusion (indicated by blue stars) with obvious subgaleal hematoma collection.

the thickness of the blood clot, and it should be more than 10 mm with a midline shift of more than 5 mm irrespective of GCS (Fig. 2). The other clinical scenario, like acute subdural hematoma with GCS less than 9, should be monitored with ICP monitoring. If the subdural thickness is less than 10 mm or midline shift less than 5 mm, then the indication of surgery is two or more than two score drops in GCS from the admission GCS with or without pupillary asymmetry. The surgical procedure opens the bone flap (craniotomy) with hematoma evacuation with or without duroplasty.

Post-traumatic hematoma or contusion

The indication of surgery in case of primary brain parenchymal hematoma or contusion depends on volume (> 20 cc) in the frontal or temporal location with midline shift of more than 5 mm46) (Fig. 3). If the volume is more than 50 cc, the individual must be considered for surgical evacuation of the contusion irrespective of the GCS score. The principle of surgery is to evacuate the hematoma or contusion with lax duroplasty. Patients with hematoma < 20 cc without focal neurological deficits and controlled ICP can be managed conservatively with anti-edema measures. In a case of herniation, either trans tentorial or uncal, decompressive craniectomy is beneficial to control the ICP.

Depressed cranial fractures

The surgical indication for depressed fracture is open compound wound with depression of bone fragment more than the thickness
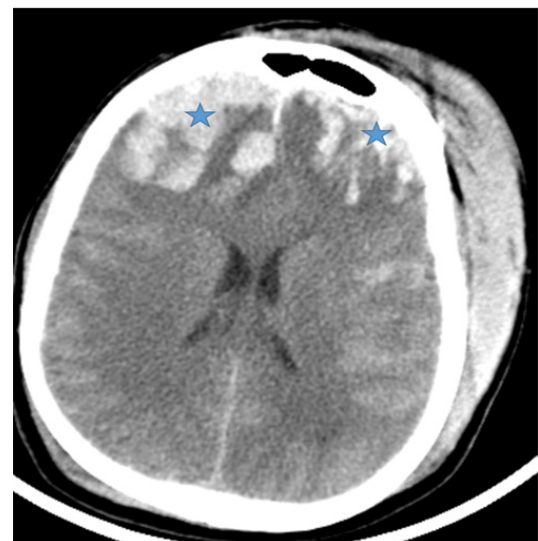
of skull with or without CSF leak or brain herniation or frontal sinus involvement (Fig. 4). The purpose of surgery in depressed fracture is to prevent infection or repair the dural tear. Early surgery is indicated in the case of a CSF leak or brain herniation. Elevation of the bone fragment and debridement is the principles of surgery[46]. The evacuation of the contused portion of the brain elimi-

Fig. 4. The axial cut of computerised tomography scan suggestive of a segment of skull bone getting depressed due to fracture at two points (marked with arrows) giving the impression of right fronto parietal depressed fracture. The fracture is causing significant compression over the underlying brain parenchyma with overlying subgaleal hematoma.

nates the chances of releasing inflammatory mediators and brain swelling.

CONCLUSION

TBI is devastating as it is detrimental for the individual, family, and society. These patients often require monitoring and treatment in the intensive care unit with a multimodal and multidisciplinary approach to prevent secondary brain injury. Multimodal treatment can improve patient outcomes by promoting customized treatment strategies for each patient rather than using the same standard treatment practice in all the patients.

NOTES

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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