Emergency Anaesthesia for the head injured patient for non head injury procedures

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Introduction:

Traumatic Brain Injury is one of the leading cause of mortality and morbidity worldwide. One of the most important concepts that has evolved recently is that the this morbidity and mortality is not due to the initial primary injury but due to to secondary injury which happens in the ensuing period. So if steps are taken to prevent secondary brain injury the outcome becomes much better. One of the most important aspect in the management of a patient with TBI is treatment considerations of the associated injuries. But, in the emergencey management of a patient with TBI the principles remain the same and the central goal is to prevent secondary brain injury. In this lecture we will enumerate the principles of management of a patient with TBI for non head injury procedure.

Intraoperative management of head injury patients:

The anaesthetic approach is the same for the patients coming for surgery irrespective of the type of the lesion. The goals are to avoid secondary brain injury, optimize cerebral perfusion and oxygenation and provide adequate operating conditions for the surgeon. Most of the patients with severe head injury who are to undergo surgery for are usually intubated in the emergency department because they are more likely to benefit from early intubation and controlled ventilation. The patients who are not already intubated and with a GCS < 9, or those who are unable to protect the airway should be treated with immediate oxygenation and securing of airway without any delay. Nevertheless, one should be aware that these patients often have a full stomach, decreased intravascular volume and potential cervical spine injury. Rapid sequence induction would be ideal in an operating room set up in view of difficulty in assessing the fasting status of a comatose patient and it would minimize the ICP elevation following intubation.

Performing a direct laryngoscopy with aggressive atlanto occipital extension should be avoided in a patient with an uncleared cervical spine. Nasal intubation is also not an ideal option either as it may be contraindicated in patients with basal fractures, Le Forte's fracture and might not be easy to negotiate the tube anteriorly or posteriorly without movement of the neck. Fibreoptic guided intubation though being theoretically ideal, have the limitations of availability of equipment, expertise in an emergency situation and might not be of much help in the presence of blood and secretions and in an uncooperative patient. Direct laryngoscopic intubation with manual inline neck stabilization if effectively applied is the most practically feasible option nevertheless, there will be difficulty with the visibility of the larynx. Use of McCoy laryngoscope, gum elastic bougie and intubation stylets may be of help. The equipment and trained personnel to perform surgical airway access should be readily available during the rapid sequence induction because occasional difficulty with intubation is inevitable and hypoxia and hypercapnia may have deleterious effects on the outcome of the severely head injured patient.

Induction agents like thiopentone, propofol and etomidate have been used for rapid sequence induction in emergency situations. They have the beneficial effects of reduction of CMRO2, ICP and minimizing the intubation response but hemodynamic effects of propofol and thiopentone might not be tolerated by hypovolemic patients and might necessitate dose modification. Etomidate is gaining popularity as an ideal sedative–hypnotic for RSI in the emergency department by virtue of its protective effects on myocardial and cerebral perfusion. Appraisal of the available evidence in a review of several studies suggests that etomidate is an effective induction agent for emergency department RSI; it has a rapid onset

of anaesthesia and results in haemodynamic stability, even in hypovolaemic patients or those with limited cardiac reserve. There is significant evidence demonstrating that etomidate, when used as an induction agent to facilitate RSI, causes transient adrenal insufficiency of uncertain clinical effect. However, the clinical relevance in head injured patients receiving this agent for induction of RSI in the ED is uncertain. Ketamine is the only intravenous anesthetics which has the unique ability to increases both CBF and CMR. studies indicate that the changes in CMR are regionally variable especially frontal and anterior cingulate cortex. PET studies in humans have demonstrated that even a subanesthetic doses of ketamine (0.2 to 0.3 mg/kg) can increase global CMR by about 25%. The cerebral autoregulation and CO_2 responsiveness is preserved during ketamine anesthesia and the anticipated ICP correlate of the increase in CBF has been confirmed to occur in humans. However, some studies report no increase in ICP with controlled ventilation or when diazepam, midazolam, thiopenthal or propofol sedation is given concurrently. Administration of 3 different doses of ketamine up to 5mg/kg showed a decrease in the ICP and no significant differences in the CPP, $SJVO_2$ and MCA blood flow velocity in 8 mechanically ventilated head injury patients who received propofol sedation(3mg/kg/hr). Hence, ketamine although is not ideal as a sole anaesthetic agent, cannot be deemed absolutely contraindicated in traumatic brain injury patients.

Studies about succinyl choline in humans suggest that (a) it can causes an increase in ICP in lightly anesthetized patients; (b) this increase is abolished by IV lidocaine, "deep" anesthesia, or a defasciculating dose of nondepolarizing blockers; (c) this response is significantly attenuated in neurologically injured patients; (d) the influence of laryngoscopy and tracheal intubation on ICP far outweighs that of succinylcholine. Although succinyl choline can produce increases in ICP due to the increased afferent traffic from the muscle spindles, it cannot be absolutely contraindicated in circumstances where rapid paralysis and intubation is thought to be necessary. Rocuronium is an effective alternative for rapid sequence induction in these patients because of rapid onset of action and no effect on intracranial hemodynamics.

Nasal gastric tubes are avoided because of the potential presence of a basilar skull fracture. A large-bore oral gastric tube can be inserted after intubation in patients with full stomach and gastric contents are initially aspirated and then passively drained during the operation.

All inhalational agents are consistently associated with the increase in CBF despite the decrease in $CMRO_2$. The order of vasodilating potency is approximately halothane $\gg$ enflurane > isoflurane > desflurane > sevoflurane. Although, the use of these agents for maintenance is appropriate with acceptable increases in ICP, but if the ICP is raised than these agents may be discontinued and a totally intravenous anesthetic or an opioid relaxant technique can be used.

Role of Regional Anaesthesia :
Centrineuraxial block is contraindicated in a patient with TBI for fear of brain herniation in patients with raised ICP. However, the role of plexus block and continuous perineural block seem to be an excellent adjunct to general anaesthesia whenever possible. For instance if a patient with polytrauma has fracture of femur along with severe TBI, a continuous femoral block will aid in stable haemodynamics in the intraoperative period as the haemodynamic surges that occur during the process of bone reaming is attenuated in the presence of good analgesia provided by the femoral block. Similarly a thoracic paravertebral block would be of great use in a patient with TBI having chest trauma with fracture ribs. The role of regional anaesthesia in attenuating the stress response to surgery further supports more extensive use of these techniques.

Prevention of secondary brain injury in the intraoperative period:

In severe traumatic brain injury, the secondary insults occur frequently and profoundly influence the outcome. This influence appears to differ markedly from the hypoxic and hypotensive episodes of similar magnitude from that occurs with other trauma patients without any neurologic injury. The deleterious influence of hypotension and hypoxemia in the outcome of severe traumatic brain injury was analysed from a large (717 patients) prospectively collected dataset from traumatic coma data base

(TCDB). The TCDB study showed that hypotension and **hypoxaemia(PaO2 < 60mmHg)** were the major independent predictors of poor outcome. A single episode of hypotension **(Systolic BP of < 90mm Hg)** was associated with doubling of the mortality and increased morbidity when compared with matched group of patients without hypotension. Retrospective data suggest that intra-operative hypotension is also important, with a three-fold increase in mortality in those patients experiencing intra-operative hypotension.

Maintenance of blood pressure

Fluid resuscitation which should be ideally started during the prehospital management of these patients should be continued in the emergency department and the operating room with the goal of optimizing cerebral perfusion pressure. The brain trauma foundation advocates the avoidance of hypotension and recommends the systolic pressure to be maintained > 90 mm Hg.

There are three cerebral perfusion pressure (CPP) management strategies driven by differing beliefs about common pathophysiologic derangements. The most commonly held, the "Edinburgh" concept, emphasizes low postinjury CBF, impaired autoregulation and the necessity to support CPP (mean arterial pressure [MAP] — intracranial pressure [ICP]) to 70 mm Hg. The "Birmingham" concept from the University of Alabama, entails pharmacologically induced hypertension. This approach is based on the belief that autoregulation is largely intact and that hypertension will result in cerebral vasoconstriction with concomitantly reduced CBV and ICP. The "Lund concept" emphasizes the contribution of hyperemia to the occurrence of elevated ICP. That approach uses antihypertensive agents to reduce blood pressure while maintaining CPP over 50 mm Hg, over time the Lund proponents have modified their approach, and now a CPP of 60–70 is considered optimal and normovolemia is the clinical objective.

The controversy over choosing crystalloids or colloids for fluid resuscitation has existed for over five decades. For more than half a century a debate has continued over whether solutions that alter oncotic pressure or osmolarity contribute to a better end result following traumatic brain injury. The current ATLS guidelines call for an aggressive fluid resuscitation regimen that starts with a 2-L bolus of crystalloids in adults, preferably lactated Ringer's (LR) solution. Crystalloids primarily fill the interstitial space; consequently, edema is an expected outcome of resuscitation according to the ATLS guidelines. To expand plasma volume, the classical axiom is that three times more volume of crystalloid is required than the volume of blood that was lost. Today, this ratio is questioned, and should probably be 5:1 because of the decreased colloid osmotic pressure secondary to decreased serum protein concentration from hemorrhage, capillary leaks, and crystalloid replacement. Tissue edema might become an important consideration, especially when dealing with head injury patients. Colloid fluids have many attractions and advantages over crystalloid resuscitation. They are more efficient than crystalloids in expanding plasma volume and achieve similar resuscitation endpoints faster and with much smaller volumes.

Regardless of the evidence that colloids also cause significant brain and lung edema, advocates of colloid use argue that by using smaller volumes and increasing the colloid-osmotic pressure, colloids reduce tissue edema compared with crystalloids. Despite all favourable characteristics metaanalyses suggest that the use of colloids may be associated with increased mortality when used in trauma resuscitation. Crystalloid-based fluid strategies are favored in trauma-resuscitation protocols, although the evidence supporting these strategies in cases of brain injury is limited. Colloid infusion after a focal cryogenic injury in animals did not increase cerebral oxygen delivery or reduce either cerebral edema formation or intracranial pressure when compared with lactated Ringer's solution. This is probably because cerebral capillaries have extremely tight intercellular junctions and are impermeable to most ions. It is the osmolality, rather than the plasma oncotic pressure, that is the major determinant of water movement between the vascular and the extravascular compartments of those areas where the blood-brain barrier is intact. In patients with leaky capillaries, albumin has been demonstrated to increase interstitial fluid volume.

There is some evidence suggesting that hypertonic saline may be a useful resuscitation fluid in TBI. Hypertonic saline solution has a number of beneficial effects in head-injured patients, including the extraction of water from the intracellular space, a decrease in the ICP , expansion of intravascular volume and increase in cardiac contractility. In recent years, small volume resuscitation by means of hypertonic

saline infusion has gained attention, not only because of its beneficial effects on the restoration of hemodynamic variables and microcirculatory improvements but also because of its effects on different cell populations involved in the complex inflammatory/immune cascade. Vassar and colleagues have conducted a series of studies investigating the use of hypertonic saline in trauma patients. Hypertonic saline did not increase the rate of bleeding. Logistic regression of a trial of hypertonic saline (7.5%) vs Ringer's lactate in heterogeneous trauma patients, including TBI, found improved survival with hypertonic saline. They conducted a multicenter trial analyzing 194 patients 74% of which had severe brain injury. Although there was no overall effect on the survival, the patients in the hypertonic saline group with a GCS of < 8 had better survival. HTS is also used for resuscitation in combination with hypertonic colloids (usually dextran 70) to increase duration of effect. In 1997, Wade and colleagues carried out a meta-analysis including nine trials and 1,889 patients, demonstrating that hypertonic saline dextran (HSD) was superior to HS and normal saline for shock resuscitation. However, the combinations are more expensive and in a randomized comparative 4-group trial, highest survival rates were achieved with HTS alone (HTS alone, 60%; HTS with dextran 70, 56%; Ringer's lactate solution alone, 49%). A recent randomized controlled trial of 229 patients with TBI with a GCS<9 and hypotension were randomly assigned to receive a rapid intravenous infusion of either 250 mL of 7.5% saline or Ringer's lactate solution in addition to conventional intravenous fluid and resuscitation protocols. In this study, patients with hypotension and severe TBI who received prehospital resuscitation with HTS had almost identical neurological function measured by the extended Glasgow Outcome Score (GOSE) 6 months after injury compared to those received conventional fluids. The brain trauma foundation has advocated that for fluid resuscitation for patients with hypotension should be done with isotonic fluids and hypertonic resuscitation is a treatment option for TBI patients with GCS < 8. The primary concerns with the use of HTS are the potential for hypernatremia, osmotic demyelination syndrome (ODS), pulmonary edema, acute renal insufficiency, rebound brain edema, hematologic abnormalities including increased hemorrhage, coagulopathy and red cell lysis.

Role of hyperventilation

Hyperventilation has long been a standard component of the management of TBI patients perceived to be at risk for increased ICP. Skippen and colleagues using xenonenhanced CT and CBF studies, demonstrated a 2.5-fold increase in the number of regions of brain ischemia in children with TBI who were hyperventilated. ***Routine hyperventilation of brain trauma patients is no longer recommended***. The brain trauma foundation recommends that the patients should be maintained with normal breathing rates and a PCO_2 of 35 to 40mmHg >

Management of the patients in the ICU

Oxygenation, ventilation and cerebral perfusion

The management of the patient with traumatic brain injury in the ICU involves the stabilization of the patients who had been resuscitated from the emergency department and who had been operated . The goals of management of TBI patients such as to maintain oxygenation and cerebral perfusion should be aggressively continued with the help of intensive monitoring. Protocol based management in the neurosurgical ICU has been associated with better outcome in traumatic brain injury patients.

Monitoring in ICU
Invasive arterial pressure and central venous pressure lines are essential for the maintenance of the CPP and normovolemia, for administration of drugs, hyperosmolar therapy etc. The Brain trauma foundation recommends that intracranial pressure (ICP) should be monitored in all salvageable patients with a severe traumatic brain injury GCS score of 3–8 after resuscitation and an abnormal CT scan (that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns). ICP data can be used to predict outcome and worsening intracranial pathology, calculate and manage CPP, allow therapeutic CSF drainage with ventricular ICP monitoring and restrict potentially deleterious ICP reduction therapies. Intraventricular

catheters are preferred as these allow for continuous measurement of ICP, most accurate,can be recalibrated *in situ* and can allow drainage of CSF to control raised ICP.

However, monitoring ICP gives only limited information regarding other factors known to be important to the pathophysiology of TBI, such as cerebral blood flow and metabolism. There have been considerable attempts over a long period of time to develop additional monitoring devices to provide information regarding cerebral blood flow and metabolism and there has been some developments. Methods to continuously monitor measures of adequate cerebral perfusion have been developed. There are devices to monitor measure CBF directly (thermal diffusion probes, trans-cranial Doppler), measure adequate delivery of oxygen (jugular venous saturation monitors, brain tissue oxygen monitors, near-infrared spectroscopy) and assess the metabolic state of the brain (cerebral microdialysis). Of all these devices the Brain trauma foundation advocates a Level III recommendation for use of jugular venous saturation and brain tissue oxygen monitoring, in addition to standard intracranial pressure monitors,in the management of patients with severe TBI based upon available evidence.

Current evidence suggests that episodes of desaturation (SjO2 _ 50–55%) are associated with worse outcomes, and high extraction (AJVO2) are associated with good outcome. Low values of PbrO2 (_10–15 mm Hg) and the extent of their duration (greater than 30 min) are associated with high rates of mortality.

Management of ICP
Current data support 20–25 mm Hg as an upper threshold above which treatment to lower ICP should generally be initiated. The control of arterial oxygen tension ($PaO_2 > 60mmHg$), arterial carbon dioxide tension ($PaCO_2 – 35\text{-}40mmg\ Hg$) is mandatory and will affect cerebral haemodynamics and ICP. Mechanical ventilation is crucial in all head-injured patients with high ICP. PEEP may increase intrathoracic pressure and potentially increase ICP by impeding venous drainage or reducing CPP. *However, when PEEP is necessary to maintain oxygenation it is not absolutely contraindicated as the clinical relevance of these small changes in ICP at modest pressures may not be significant and not affect the CPP*. Flexion, extension or torsion of neck should be avoided as the ICP may increase due to cerebral venous outflow obstruction. In prone or supine-positioned patients a reverse Trendelenburg position of 10-15º reduces ICP without affecting CPP. The most effective method of lowering ICP is the removal of space-occupying lesions and this must be considered at every stage of patient management. Drainage of CSF when an intraventricular catheter is already in place is a simple and effective measure to reduce ICP. Even withdrawal of a few mls of CSF will tremendously relieve the increased ICP.

Hyperosmolar therapy

Hyperosmolar therapy has been traditionally used to reduce the ICP and improve the CPP. Mannitol has been dominant among the hyperosmolar agents over the past few decades. Mannitol acts rapidly probably due to a plasma-expanding effect and improved blood rheology due to a reduction in haematocrit and also due to an osmotic effect by drawing water across an intact blood brain barrier. Mannitol is effective for control of raised intracranial pressure (ICP) at doses of 0.25 gm/kg to 1 g/kg body weight. It has been used both on a short term and long term basis in the management of ICP.Its beneficial effects on ICP, CPP, CBF and brain metabolism, and its short-term beneficial effect on neurological outcome are widely accepted as a result of many mechanistic studies performed in humans and in animal models. *There is, however, a lack of evidence to recommend repeated, regular administration of mannitol over several days.*
Current guidelines restrict mannitol use prior to ICP monitoring to patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes. The prolonged administration of mannitol may lead to intravascular dehydration, hypotension, and prerenal azotemia. As mannitol is entirely excreted in the urine there is a risk of acute tubular necrosis, particularly if serum osmolarity exceeds 320 mOsmol/l. Therefore, plasma osmolarity has to be monitored during therapy with hyperosmotic agents. As an alternative therapeutic avenue, hypertonic saline solutions have gained renewed interest and, recently, more common application in neurocritically ill patients. After effective and apparently safe use in a few patients not responding to mannitol, their employment has even been supported with some enthusiasm. There are only a small number of comparative trials evaluating mannitol against hypertonic saline in the treatment of intracranial hypertension. The factors that stimulate interest in HTS

are the belief that it preserves or even improves the hemodynamics and has the theoretical advantage that it has a higher osmotic reflection coefficient (1.0 compared with 0.9 mannitol) across an intact blood–brain barrier. Current evidence is not strong enough to make recommendations on the use, concentration and method of administration of hypertonic saline for the treatment of traumatic intracranial hypertension.

Sedation

All mechanically ventilated patients with traumatic brain injury should receive sedation and analgesia to facilitate mechanical ventilation and to avoid ventilator dyssynchrony. Pain and agitation in these patients can increase the ICP and blood pressure and therefore should be avoided. A variety of pharmacological agents are used for sedation and analgesia in the head injury patients each with their own advantages and disadvantages. Opioids provide analgesia, depression of airway reflexes and minimal hemodynamic effects. Morphine and fentanyl are widely used but tachyphylaxis and withdrawal symptoms are common problems. There exists some uncertainty about the effect of synthetic narcotics on the ICP and cerebral hemodynamics. Among the benzodiazepines midazolam is effective as a sedative, short acting and the effect can be reversed by flumazenil but accumulation of 1- hydroxy midazolam is a problem. Propofol is a widely used neurosedative agent in patients with an acute neurologic insult, as it is easily titratable and rapidly reversible once discontinued allowing for periodical neurological evaluation. ***But high dose of propofol infusions have been associated with propofol infusion syndrome*** (hyperkalemia, hepatomegaly, lipemia, metabolic acidosis, myocardial failure, rhabdomyolysis, and renal failure) in both children and adults. Thus extreme caution must be taken when using doses greater than 5 mg/kg/h or when usage of any dose exceeds 48 h in critically ill patients. Propofol when used in hypothermic patients can accumulate and cause hyperlipidemia.

Nutrition and fluid-electrolyte balance:

Current data and guidelines suggest that head injury patients should be fed to attain full caloric replacement by end of first week. Jejunal feeding through feeding jejunostomy placed surgically or fluoroscopic guided has advantages of better acceptability compared to gastric feeding and avoidance of intravascular catheters as in parenteral nutrition. No clearly superior method of feeding has been demonstrated either in terms of nitrogen retention, complications, or outcome. Both animal and human studies have associated hyperglycemia with adverse neurological outcome in brain injury. ***Hence tight control of glycemia should be a routine part of management of traumatic brain injury.*** Dyselectrolytemias are common in patients with traumatic brain injury due to complex etiologies like cerebral salt wasting syndrome, SIADH syndrome and central diabetes insipidus syndrome. The diagnosis and management of these conditions are different with varying fluid management regimes. Both hyponatremia and hypomagnesemia lower the seizure threshold. Hence, electrolytes should be frequently monitored in these patients.

Seizure prophylaxis

Post TBI have high incidence of post traumatic seizures which cannot be prevented by prophylaxis during the acute management. ***So routine prophylaxis with phenytoin or valproate is not recommended for preventing late posttraumatic seizures.*** Anticonvulsants may be indicated to decrease the incidence of early PTS (within 7 days of injury), which are not usually associated with worse outcomes.

DVT prophylaxis

The risk of deep venous thrombosis may be high in head injury patients due to long periods of immobility and associated polytrauma. Graduated compression stockings or intermittent pneumatic compression (IPC) stockings are recommended as there can be increased risk for expansion of intracranial hemorrhage with chemothromboprophylaxis. ***Most authors agree that low molecular weight heparin or low dose unfractionated heparin can be used after 72 hours of injury.***

Conclusion

Management of patients with traumatic brain injury right from admission in the emergency department to intensive care management involves a multidisciplinary approach with cooperation. Avoidance of secondary brain injury is the main goal of management of these patients. With the better understanding of pathophysiology of brain injury, improved resuscitation methods and diagnostic modalities, timely surgical intervention and protocolized management of these patients in the intensive care unit, an improvement in the outcome of these patients can be definitely seen. While operating for a non neurological surgery in a head injured patient the most important principle will be to avoid all the causes that will result in secondary brain injury.