Editorial

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Brain Injury and prehospital care: Reachable goals in India

Harjinder S Bhatoe VSM
Department of Neurosurgery, Command Hospital (SC) & Armed Forces Medical College
Pune 411040. Maharashtra

The impact of traumatic brain injury (TBI) on patients, families, and society is tremendous, and management to lessen its effect starts at the injury site and in the pre-hospital stage. The pre-hospital assessment and treatment by first responders are the critical steps in initiating safe and appropriate care for the patient. Moderate to severe traumatic brain injury usually requires active prehospital management by first responders in the field. Pre-hospital care of TBI clearly influences the morbidity and mortality outcomes in patients with TBI. Recent clinical and laboratory evidence suggests that secondary brain injuries can be lessened with appropriate and expeditious interventions performed in the pre-hospital arena. Tackling hypotension, ensuring airway and breathing, and expeditious, efficient and comfortable transport to a neurosurgical care center are the pillars of pre-hospital care of the head-injured.

Pre-hospital care provision in a patient with head injury is racing the biological clock, to salvage a rapidly perishable entity, the human brain function. Experimental evidence suggests that several early cellular and molecular events take place immediately following TBI even in the absence of radiographic or anatomic findings. Opening of sodium channels and leukocyte histamine release lead to cellular swelling, and membrane depolarization triggers calcium influx causing release of excitatory neurotransmitters such as glutamate[1-9]. Excitatory overstimulation leads to increased cellular metabolism, which becomes toxic when the cell's demand for oxygen outstrips the ability of the body to provide that cell with enough oxygen. Reduced or inadequate cerebral perfusion after trauma leads to the formation of lethal amounts of cell metabolites and free radicals that lead to cell death. In addition, endothelial injury by mechanical or chemical forces causes focal hemorrhage and edema, further compromising cerebral perfusion and the blood-brain barrier. Halting these biochemical cascades and potentially reversing the effects are of utmost importance in salvaging functional brain before permanent damage sets in, both in directly injured brain and in "at risk" brain. Recognition that secondary brain insults (SBI) are primary determinants of outcome in the patient with severe TBI has heightened emphasis on prehospital management and resuscitation in an attempt to minimize the detrimental effect of SBI on the brain. SBI often requires critical care monitoring and aggressive surgical and medical management. Systemic SBI is the result of hypotension, hypoxia, anemia, hyperthermia, hypercapnia, hypocapnia, electrolyte imbalances, hyperglycemia, hypoglycemia, acid-base abnormalities, and systemic inflammatory disorders.

Hypotension and hypoxia are two factors that have an immediate impact in the outcome in severe head injury, and these two factors can be addressed in the pre-hospital management of head injury. Even a single episode of hypotension doubles the mortality when compared with those groups without observed hypotensive episodes, and patients with combination of hypoxia and hypotension have worse outcome. Even a single episode of systolic blood pressure < 90 mm Hg, and hypoxia have powerful negative effects on outcome. The concept, though universally recognized, is not followed in letter and spirit across the country due to scant resources. The recognition of these conditions can be fairly simple and treatment uncomplicated, including fluid boluses with isotonic or hypertonic saline and field intubation. Hypotension is rarely seen in a patient with TBI alone in adults, except in terminal brainstem injury although children can suffer exanguination and hypotension from scalp wound. The usual accompaniments that predispose to hypotension are thoracic, abdominopelvic, vascular or long bone injury. Analysis of data from Traumatic Coma Data Bank (TCDB) demonstrated a statistically significant effect of

Address for Correspondence:
Col Harjinder S Bhatoe VSM
Department of Neurosurgery
Command Hospital (SC) & Armed Forces Medical College
Pune 411040. Maharashtra
E-mails: hsbhatoe@gmail.com, harjinderbhatoe@yahoo.co.in

pre-hospital hypotension and Glasgow Outcome Score (GOS)[10]. The effect of early hypotensive episodes differs in the setting of neurologic injury compared with that of trauma patients with hypotension of similar magnitude without neurologic involvement. Even a single episode of hypotension doubles the mortality when compared with those groups without observed hypotensive episodes, and patients with combination of hypoxia and hypotension have worse outcome. Manley et al in a prospective cohort study concluded that 24% of patients with head injury suffered hypotensive episodes, and these patients were three times more likely to die compared to those patients without hypotension, and multiple hypotensive episodes are additive for fatal outcome[2]. In a prospective analysis of 315 patients with severe head injury, Fearnside et al observed the unfavorable outcome even with a single episode of hypotension[11]. When hypotensive patients were further stratified into groups with less than 60, between 60 and 80, and more than 80, it was observed that lower SBP correlated with poor outcome[12]. In the pediatric age group, hypotension (blood pressure readings less than fifth percentile for age) exceeding five minutes was associated with poor outcome[13]. Pigula et al reported four-fold increase in mortality in 58 patients younger than 17 years experiencing a single episode of hypotension following severe head trauma[13].

Unfortunately, no prospective randomized trial evaluating the benefit of preventing or correcting prehospital hypotension in improving outcomes exists. Also, hypotension defined as SBP < 90 mm Hg is an arbitrary limit and may not actually represent the optimal minimal blood pressure target in head injury patients. Future studies will focus on delineating optimal resuscitation protocols and clarification of minimal blood pressure limits.

FLUID RESUSCITATION

Prevention and treatment of hypotensive shock are key components in the pre-hospital management of head trauma. Although only 5% of patients present in severe hemorrhagic shock[14], its detrimental effect in the head-injured patient is significant in whom cerebral autoregulation is often impaired. Loss of autoregulation places the brain at increased ischemic risk from fluctuations in systemic blood pressure. Hypotension in the setting of trauma is usually related to decreased preload (hemorrhagic shock)[10]. Crystalloid fluid is commonly used in the field to augment cardiac preload and maintain cardiac output in the setting of falling blood pressure and head injury. General recommendations include rapid infusion of 2 L of isotonic fluid, usually normal saline or Ringer's lactate, as initial resuscitation. Concern, however, exists that additional fluid can worsen cerebral edema and raise ICP. Choice of intravenous fluids and their quantity to be administered have been topics of projects related to trauma resuscitation. Certainly, there is no merit in the advocacy of "run the patient dry." There are no studies specifically addressing the issue of pre-hospital use of fluids and its effect on volume status. In the critical care setting, Scalea et al19 have shown that ICP is unrelated to central venous pressure, pulmonary capillary wedge pressure, and amount of fluid or blood given[15]. The only significant relationship found was between ICP and serum lactate. Rising lactate, being an indication for inadequate resuscitation, correlated with rising ICP. Clinical studies have advocated the use of hypertonic saline over standard isotonic crystalloid. Although there is no single randomized, prospective study demonstrating a clear advantage of hypertonic saline over standard crystalloid, several reports have compelling evidence that supports the use of hypertonic saline in TBI. Wade et al examined 233 head-injured patients who received either hypertonic saline–dextran or normal saline as fluid resuscitation in the pre-hospital phase or emergency room. Patients receiving hypertonic saline–dextran were twice as likely to survive as those receiving normal saline (p = .048)[16]. A prospective, randomized, double-blind multicenter study of 144 patients with severe brain injury showed that giving 250 mL of hypertonic saline over normal saline as initial resuscitation improved survival rates significantly in patients with GCS < 8, although overall survival for the entire group studied was unchanged[17]. Additional studies from Vassar and her colleagues have corroborated the improvement in blood pressure and survival when hypertonic solutions have been used early in resuscitation compared with Ringer's lactate and normal saline[18]. Of note, the addition of dextran into hypertonic solutions did not add any further benefit[19]. In patients without TBI, there is concern that automatic fluid resuscitation without surgical hemostasis can lead to secondary blood loss by dilution of thrombotic and coagulation factors and decrease in oxygen carrying capacity by hemodilution. One randomized series of patients with penetrating thoracic trauma and hypotension showed that patients with resuscitation

delayed until after surgical hemostasis had lower mortality[20]. Also, animal models of uncontrolled hemorrhagic shock and TBI demonstrated higher ICP and lower cerebral oxygen delivery in animals with early resuscitation compared with those in which resuscitation was delayed until hemostasis was achieved[21]. lso, the use of early pressor support to maintain blood pressure improved cerebral perfusion pressure in swine but resulted in a poorer outcome than in animals resuscitated with fluid alone[22]. Thus, pre-hospital resuscitation should be performed to maximize correction of hypotension in the TBI patient without potentiating secondary blood loss and dilutional coagulopathy. Unfortunately, there is a paucity of clinical and scientific evidence specifically addressing these issues.

OXYGENATION AND AIRWAY MAINTENANCE

Hypoxia in TBI patients is a fairly common occurrence in the pre-hospital setting. One study showed that 27% of 131 patients with severe TBI were hypoxic on admission to the hospital[23]. A prospective observational study in Italy found that 55% of TBI patients were hypoxic (arterial oxygen saturation [SaO2] < 90%) at the accident scene[12]. Both of these studies correlated poor outcome with desaturations in oxygenation. It is thus evident that hypoxemia and airway-ventilation management in the pre-hospital setting have significant effects on the outcome of TBI patients, and hypoxemia in TBI patients is clearly harmful in both adult and pediatric patients. The TCDB study by Chesnut et al[10] examined the outcome of 717 patients who had suffered severe head injury (GCS = 8) and explored the impact on outcome of hypotension and hypoxia, defined as a partial pressure of arterial oxygen (PaO2) equal to 60 mm Hg or apnea or cyanosis in the field. Hypoxia, particularly in combination with an episode of hypotension, was associated with increased morbidity and mortality. Mortality was 28.0% in patients with hypoxia and 57.2% in patients with both hypoxia and hypotension versus 26.9% when neither condition was present. These findings were statistically independent of age and the presence of multiple trauma. While hyperventilation is a simple way to decrease ICP by inducing vasoconstriction of cerebral vasculature in response to a reduction in arterial partial pressure of CO2 (PaCO2), unfortunately, in our scenario, monitoring of tissue oxygenation cannot be assured and chances of hyperventilation-induced cerebral ischemia are high. Hyperventilation should be reserved for emergency scenarios during which clear signs of clinical cerebral herniation are present. There has been conflicting evidence in terms of the harms and benefits of pre-hospital endotracheal intubation in patients with TBI[24-27]. One retrospective case control study examined 1092 patients with severe head injury (GCS 9 and a head or neck Abbreviated Injury Score [AIS] > 4) and assessed outcome comparing those who underwent prehospital intubation with those who did not[24]. Paramedics were not permitted to use pharmacologic agents to aid in intubation and attempted intubation only if the patient was apneic, unconscious with ineffective ventilation, and had no gag reflex. This study showed that patients who underwent pre-hospital intubation had a mortality of 26% whereas patients that were not intubated had a mortality of 36.2%, suggesting that pre-hospital intubation is indeed beneficial. A retrospective review of patients' data performed in Los Angeles, however, showed that patients with severe head trauma (GCS < 9 or AIS > 4) who underwent pre-hospital intubation had a 74% increased risk for mortality compared with patients who were not intubated[27]. Prospective studies on this issue seem to support the evidence against pre-hospital intubation. A retrospective study in which subjects were matched to historical controls showed that patients who underwent rapid sequence intubation had increased mortality and poorer outcome compared with matched controls[26]. In another prospective study, Bochicchio and colleagues reported that patients with severe TBI intubated in the field suffered increased morbidity (measured by ventilator days, hospital days, intensive care unit days, and incidence of nosocomial pneumonia) and nearly double the mortality (23% vs. 12.4%) compared with patients intubated upon arrival in the hospital[25]. It is not clear why these patients had worse outcomes, but prospective randomized studies must be performed to confirm these results. Current collected evidence shows that even a single episode of hypoxemia leads to increased morbidity and mortality in patients with severe TBI. Routine hyperventilation should be avoided to minimize the risk of decreased cerebral blood flow and cerebral ischemia. EMS personnel should be vigilant in maintaining adequate oxygenation and ventilation in the TBI patient to avoid episodes of hypoxemia, but randomized controlled trials must be performed to clarify the role of endotracheal intubation in the pre-hospital arena.

HOSPITAL TRANSPORT

Patients with TBI are likely to worsen rapidly from the twin adverse factors in the pre-hospital phase, namely hypotension and hypoxia. The first responders have to recognize and assess TBI, and make the important decision about the choice of hospital destination. These are the important decisions affecting patients' outcome. Organized pre-hospital transport is still non-existent in most parts of India, and the laypersons and relatives etc who bring the patient to medical care have little awareness or knowledge about the importance of immobilization, position, prevention of aspiration, etc. Several fundamental processes and tasks are required from EMS to manage and direct patients with TBI properly. First, EMS call-takers and dispatchers must be able to determine the presence of TBI in the trauma patient and, if present, dispatch the highest level of care and EMS provider to the scene. Second, EMS personnel must be highly skilled in their ability to evaluate appropriately the severity of injury. This includes ascertaining the exact mechanism of trauma, determining a field GCS score, assessing the patient's vital signs, and performing a pupillary examination in order to establish the extent of brain injury. Third, EMS personnel must be skilled enough to provide appropriate interventions such as fluid resuscitation and airway management to correct any SBI that could lead to increased morbidity and mortality. Fourth, the response team must be conscientious when choosing a hospital to which the patient will be transported. Hospitals receiving patients with severe TBI should have the appropriate resources, including the capability for immediate computed tomography, to diagnose intracranial pathology. Transcranial hand-held scanners are now available for onsite determination of the presence of intracranial hematoma with reasonable certainty. These can be part of the kit of the first responders. A neurosurgeon should be available at the destination hospital if drainage of an intracranial hematoma is necessary. There are special considerations for both urban and rural trauma victims. EMS personnel in urban centers must contend with a larger call volume and traffic that may impede rapid hospital transport. EMS in rural settings must deal with extended transport times, as a hospital with the appropriate resources may not be located nearby. In this setting, EMS should have access to more rapid forms of transport (e.g., helicopter). Unfortunately, there are no randomized, controlled trials evaluating methods of hospital transport and differences in EMS protocols in the outcome of severe TBI. Analysis of outcomes in several cities, before and after implementation of coordinated regional trauma systems in San Diego, Oregon, and Quebec, suggests that trauma systems confer better outcomes in all trauma settings[28-30]. Also, centers with extensive experience in treating large volumes of trauma patients per year were shown to have 30% less chance of death than lower volume centers[31]. The availability of specialty care is an obvious necessity for comprehensive treatment of complicated multitrauma victims.

CONCLUSION

Prehospital care of the head injured begins at the site with safe extrication, attention to airway and insertion of intravenous line followed by rapid and reliable transport with provision of en-route assessment and rsuscitation[32]. Morbidity and mortality outcomes clearly relate to prehospital factors. Among the most powerful pre-hospital predictors of poor outcome following TBI are hypotensive and hypoxic episodes. These conditions propagate SBI, which in turn causes permanent neurologic deficit. Interventions, including fluid resuscitation and intubation, to correct or prevent these episodes seem to have a beneficial effect, but definitive investigations are still needed. Clearly, first responders treating a TBI patient should avoid hypotension and hypoxia, refrain from hyperventilation, and expedite rapid and safe transport to a trauma center. Accurate and timely recording of vital signs and clinical examination are also high-priority measures that will help in the hospital phase of treatment by the receiving trauma team. Trauma, and the issue of how to care for a trauma victim, is a problem for communities worldwide. Continued vigilance in providing both improved pre-hospital and hospital care for these victims to prevent long-lasting morbidity is of utmost importance.

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