REVIEW ARTICLE

Pre-hospital and initial management of head injury patients: An update

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ABSTRACT

Background: Most of the bad outcomes in patients with severe traumatic brain injury (TBI) are related to the presence of a high incidence of pre-hospital secondary brain insults. Therefore, knowledge of these variables and timely management of the disease at the pre-hospital period can significantly improve the outcome and decrease the mortality. The Brain Trauma Foundation guideline on "Prehospital Management" published in 2008 could provide the standardized protocols for the management of patients with TBI; however, this guideline has included the relevant papers up to 2006. **Methods:** A PubMed search for relevant clinical trials and reviews (from 1 January 2007 to 31 March 2013), which specifically discussed about the topic, was conducted. **Results:** Based on the evidence, majority of the management strategies comprise of rapid correction of hypoxemia and hypotension, the two most important predictors for mortality. However, there is still a need to define the goals for the management of hypotension and inclusion of newer difficult airway carts as well as proper monitoring devices for ensuring better intubation and ventilatory management. Isotonic saline should be used as the first choice for fluid resuscitation. The pre-hospital hypothermia has more adverse effects; therefore, this should be avoided. **Conclusion:** Most of the management trials published after 2007 have focused mainly on the treatment as well as the prevention strategies for secondary brain injury. The results of these trials would be certainly adopted by new standardized guidelines and therefore may have a substantial impact on the pre-hospital management in patients with TBI.

Key words: *Evidence, pre-hospital management, secondary brain injury, traumatic brain injury*

INTRODUCTION

Traumatic brain injury (TBI) is a major burden (approximately 10 million annually victims) on the health care system throughout the world.[1] Moreover, of the patients who survived from the primary insult, some may still have long-term disability. Most of these outcomes are related to the presence of a high incidence of pre-hospital secondary brain insults.[2,3] Therefore, knowledge of these variables and timely management of the disease at the pre-hospital period can significantly improve the outcome and decrease the mortality.[2,3]

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Evidence-based clinical practice "Prehospital Management" by the Brain Trauma Foundation (BTF) guidelines have been modified and revised in 2008.[4] These guidelines could provide the standardized protocols for the management of patients with TBI; however, this guideline has included the relevant papers up to 2006. Thereafter, the results of various trials have been published and the major focus of these is mainly to minimize the secondary brain injury following the primary insults. However, the results and conclusions of these trials are yet to be adopted by standard guidelines.

In this article, we have summarized the various aspects of pre-hospital as well as initial management at emergency department in patients with TBI, including the emerging evidence mainly after the BTF 2008.[4]

METHODS

This is a narrative review based on a PubMed search on the terms including "Traumatic brain injury," "Head

injury," "Head trauma," "prehospital," "Resuscitation" and "Management." This review has involved the relevant clinical trials and reviews (from 1 January 2007 to 31 March 2013) that specifically discussed about the topic. The studies with a minimum of 50 patients are included. The papers in any language have been included; however, pediatric and pregnant patients are not included in this review.

Etiopathogenesis

The most common cause for inciting head injury is motor vehicle accident (MVA).[1-3] The other causes of TBI are falls from heights, physical assaults, accidents at home, offices or while playing sports, gunshot and blast injuries. Head injury can be either closed (blunt) or open (penetrating) injury and is further classified into primary injury and secondary injury. This classification is rather more useful while considering the therapeutic and preventive strategies [Table 1].

Brain tissue destroyed by the primary impact is very difficult to be salvaged. Secondary brain injury, the sequel of primary insult, is potentially treatable and most of the therapies are targeted to prevent these sequelae. This secondary injury is primarily amplified by various cofactors, including hypoxemia, hypotension, hypercarbia, hypoglycemia, hyperglycemia, hypothermia/hyperthermia, seizures, etc., [Table 1]. The morbidity, mortality and long-term disability are substantially dependent on the management of secondary brain insult. Therefore, functional outcome may be improved by good intensive medical care and by prompt surgical interventions.[2,3,5]

Evaluation

Physiological parameters including Glasgow Coma Scale (GCS) score, systolic blood pressure and respiratory rate are useful predictors for the outcome and can be utilized in the pre-hospital triage of patients with TBI [Table 2].[5] The GCS is a quick, reproducible scoring system that has worldwide acceptance and is used to define the severity of TBI, with severe TBI defined as GCS of ≤ 8. It is based on eye opening, verbal response and the best motor response. An early, non-sedated GCS has been shown to predict prognosis. The lowest total score indicates likely fatal damage, especially if both pupils fail to respond to light and oculovestibular responses are absent. Higher initial scores tend to predict better recovery.[5] The data from the 2007 National Trauma Data Bank (NTDB) National Sample Program showed that after controlling for age, gender, race, Injury Severity Score (ISS) and length

Table 1: Classification of brain injury and factors affecting these in patients with TBI

Type of traumatic brain injury	
Primary injury (direct mechanical impact)	Preventive management
Diffuse brain injury	
Brain concussion (loss of consciousness lasting <6 h)	
Diffuse axonal injury (loss of consciousness lasting >6 h)	
Focal brain injury	
Brain contusion (below or opposite the region of impact)	
Epidural hematoma (laceration of the middle meningeal artery)	
Subdural hematoma (tearing of the bridging veins)	
Intracerebral hematoma	
Secondary injury	Target of TBI management
(Minutes, hours or days of the initial injury)	
Aggravating factors	
Hypoxemia, hypercapnia	
Hypotension, low cardiac output	
Intracranial hypertension	
Biochemical and metabolic derangements	
Hyperthermia	
Seizures	
Hypo- or hyperglycemia	

TBI – Traumatic brain injury

Table 2: Evidence on the effect of pre-hospital evaluation/transport methods/personals/ duration on outcome

Author/year	Intervention	Outcome
Bulger *et al.* (2012) 2049	Mode of transport (Air vs. other)	No difference Air transport-longer Pre-hospital time, more pre-hospital intubation and larger fluid received
Franschman *et al.* (2012) 497	Pre-hospital run time (P-HEMS vs. EMS based)	Similar run time in both groups Less favorable outcome-EMS
Berlot *et al.* (2009) 194	Mode of transport (Air vs. ground)	Mortality (21% vs. 25%) Survival (54% vs. 44%) Disabilities (25% vs. 31%)
Franschman *et al.* (2011) 339	Effect of PH risk factors on outcome of CT-confirmed severe TBI	Pupillary reflex (odds -5.8) GCS (odds-4.9) Hypotension (odds-3.5) PH intubation-not related to outcome
Caterino *et al.* (2012) 52,412	SMS score vs. GCS score Comparison of sensitivities For different parameters	Mortality (72.2% vs. 76.4%) TBI (40.8% vs. 45.4%) Intervention (52.9% vs. 60%) Intubation (72.7% vs. 75.5%)

P-HEMS – Physician-based helicopter emergency medical services; EMS – Paramedics-based emergency medical services; SMS – Simplified motor score (three points); GCS – Glasgow coma scale (15 points)

of stay in the hospital, patients who had a GCS score ≤13 were 17-times more likely to die than TBI patients who had a higher GCS score.[5] The other scoring system, the simplified motor score (SMS), a three-point measure of TBI severity, is also found to perform equally well as GCS and is validated in many trials. In a retrospective review of 52,412 patients, sensitivity, specificity and area under the receiver – operating characteristic curves were similar between the SMS and GCS for all outcomes.[6] Sensitivity for mortality was 72.2% for SMS and 74.6% for GCS. Sensitivity for TBI was 40.8% for SMS and 45.4% for GCS. Sensitivity for neurosurgical intervention was 52.9% for SMS and 60.0% for GCS.[6] However, the severity and prognosis are predicted more accurately by also considering the computed tomography (CT) scan findings and other clinical factors including a disturbed pupil reflex and arterial hypotension.[5,7,8] Other investigations like X-ray and ultrasound are also helpful to rule out other major injuries.

An early non-contrast brain CT is required to guide subsequent therapy. In fact, CT scan facility located in the trauma or emergency room has been found to reduce the time to acquire CT images and improves overall mortality.[8] Once airway, breathing or ventilation and blood pressure is maintained, one should concentrate on the management of intracranial hypertension and evaluate the other associated injuries.

Transportation

The outcome in patients with TBI is also found to be affected by the transport methods, duration and physician or paramedics lead team [Table 2].[9,10] In some systems, air medical crews may provide a higher level of care but may require longer transport times. Based on the analysis of data (2049 patients) from two randomized trials of pre-hospital hypertonic resuscitation, patients transported by air (in comparison with ground transport) had higher rates of pre-hospital intubation (81% vs. 36%), received more intravenous fluids (mean 1.3 L vs. 0.8 L) and had longer pre-hospital times (mean 76.1 min vs. 43.5 min).[9] However, there was no significant impact of mode of transport on survival or 6-month neurologic outcome.[9] Similarly, in a retrospective review of 194 patients, air-based emergency medical services were associated with a lower rate of mortality (21% vs. 25%) as well as neurological disabilities (25% vs. 31%).[11] In addition, a retrospective study of 497 patients with severe TBI showed that in comparison with paramedics, physician-lead helicopter emergency services were found to have a more favorable outcome and mortality scores and there were also similar pre-hospital run times between the two groups.[10] Therefore, there is a need to integrate the most effective transport system through which patients of TBI can be shifted to a specialized trauma center within the minimum possible time. The time is a crucial factor for the occurrence as well as the prevention of secondary brain injury; therefore, the transport vehicle should have all possible drugs/equipment and monitoring devices. Accompanying emergency medical personnels should also have a thorough knowledge of important risk factors and guideline-based management strategies to ensure substantial prevention of secondary brain injury.[9-11]

Initial management

The guidelines of Advanced Trauma Life Support (ATLS) provide a standardized protocol for approaching the severely injured patient, with special attention to airway, breathing and circulation.

Hypoxemia

The first most important consideration in TBI is the maintenance of a clear and unobstructed airway [Tables 3, 4]. Hypoxemia is the most important factors related to worse outcome and therefore needs urgent attention.[12] In a multicenter prospective study of 150 patients, the mortality was found to be 20% in patients without secondary insults; however, it increased to 37% during hypoxic episodes.[12] Patients with severe TBI are to be intubated by rapid sequence induction by skilled personnel. The cervical spine must be protected and should always be assumed to be injured until definitively cleared while securing the airway. A randomized controlled trial showed that rapid sequence intubation (RSI) performed by paramedics was associated with an increase in the rate of favorable neurologic outcome at 6 months compared with intubation in the hospital; this highlights the importance of immediate airway control in cases of severe TBI.[13] Similarly, an observational study of 334 patients with pre-hospital hypothermia (PH) intubations and GCS score ≤8 and proven TBI (on CT scan) revealed a higher rate of overall intubations and was associated with no increase in mortality over the emergency department intubation.[14] In addition, the use of RSI with appropriate use of anesthetic agents is of paramount significance to prevent aspiration-related adverse events and simultaneously prevent sympathetic surge incited by airway manipulations; however, avoidance of hypotension should be ensured. The role of lidocaine in cases of RSI is also controversial; however, a recent retrospective cohort of 101 patients showed that lidocaine can be used without producing adverse hemodynamic changes.[15]

The technique and equipment are also equally important to manage the pre-hospital intubations, especially by the paramedics. In this regard, the role of video-laryngoscopes and other difficult intubation kits may certainly give better chances for airway management and thus would prevent the secondary brain injuries related to hypoxemia.[16]

Table 3: Evidence on pre-hospital airway management/monitoring

Author/year	Intervention	Outcome
Bernard *et al.* (2010) 312	Pre-hospital intubation by Paramedics versus in-hospital Intubation by physicians	More favorable outcome in paramedic (51% vs. 39%)
Vandromme *et al.* (2011) 334	Intubation vs. no (PH and ED) GCS <8 and CT scan-proven TBI	GCS <8- and proven TBI-overall more intubation, no increased risk of mortality in PH
Davis *et al.* (2011) 1555	PH-intubation attempts on outcome In severe TBI	Higher mortality in attempted Intubations (odds-2.9) Sites with higher intubation-overall lower mortality in all victims (odds-1.4)
Davis *et al.* (2011) 98	Incidence of pulse oximetry failure during RSI in severe TBI (Loss of signals and latency)	Loss of signals-79% latency-55%
Davis *et al.* (2010) 11,000	ETI and outcome In severe TBI using trauma and injury severity score	PH intubation-improve outcome in more critical TBI air medical-better outcome hypo-or hyperventilation-worse outcome
Davis *et al.* (2008) 87	Rate of SpO_2 decline and threshold for BMV	Inflection point SpO_2 <93% below and above (SpO_2 <93%) rate of desaturation to hypoxemia (100% vs. 6%)
Lin *et al.* (2012) 101	Lidocaine in RSI (Hemodynamic changes)	Not related to hypotension

PH – Pre-hospital; ED – Emergency department; TBI – Traumatic brain injury; RSI – Rapid sequence intubation; GCS – Glasgow Coma Scale; CT – Computed tomography; BMV – Bag mask ventilation

Table 4: Evidence on pre-hospital ventilation and fluid resuscitation

Author/year	Intervention	Outcome
Dumont *et al.* (2010) 77	Effect of PH hyperventilation on In-hospital mortality in severe TBI	Normocarbia-12% hypocarbia-77% hypercarbia-61%
Caulfield *et al.* (2009) 100	PH Hypocapnia and outcome guideline based vs. non-adherence	Guideline-oriented mortality 29% non-adherence group mortality 45%
Warner *et al.* (2008) 851	ED ventilation on outcome target range of $PaCO_2$ vs. outside target	Mortality (21.2% vs. 33.7%)
Bulger *et al.* (2010) 1282	7.5% saline/6% dextran-70% or 7.5% saline vs. 0.9% saline	No difference at 6 months outcome
Rhind *et al.* (2010) 65	7.5% hypertonic saline 6% dextran-70% vs. 0.9% NS	HSD suppresses more inflammatory and coagulative markers

PH – Pre-hospital; ED – Emergency department; TBI – Traumatic brain injury; HSD – Hypertonic saline dextrose

In addition, a high incidence of oxygen desaturation during PH intubations warrants standard monitoring such as pulse oximetry. A study on 87 patients with TBI undergoing RSI by pre-hospital providers revealed that the rate of SpO_2 decline increases as the SpO_2 decreases, with an inflection point occurring around 93%.[17] Intubation attempts below this value are almost always associated with subsequent desaturation.[17] However, a high incidence of pulse oximetry failure was also observed with the use of a digital pulse oximetry probe during pre-hospital RSI.[18] In a retrospective study of 124 patients with severe TBI, 79% of the patients had pulse oximetry failure at the time of RSI. In addition, a latent period appears to exist in the majority of patients (55%) undergoing desaturation.[18] Therefore, caution should be paid to all differential causes of oxygen desaturation, including pulse oximetry related, while attempting RSI.

Hypercarbia/hypocarbia

The intubated patients should be ventilated so as to maintain normocarbia ($PaCO_2$ 35 and 40 mmHg). Monitoring of oxygen saturation and capnography is recommended in severely head-injured patients so as to avoid unrecognized hypoxemia or changes in ventilation [Tables 3, 4].[4] This is best assessed by end tidal capnography, and generally corresponds to $EtCO_2$ 30-35 mmHg.[4] In analysis of data of 11,000 patients, both hypo- and hypercapnia were associated with worse outcomes in intubated patients.[19] In another retrospective review of 77 patients, both hypocarbia ($PaCO_2$- <35 mmHg) and hypercarbia ($PaCO_2$- >45 mmHg) were associated with a substantial increase in in-hospital mortality (77% and 61%, respectively).[20] A study on guideline-based management in pre-hospital ventilation ($PaCO_2$ >29 mmHg) in 100 patients (without herniation signs) showed that the in-hospital mortality was 29% among those in whom guideline levels were achieved and 46% (16 of 35) in those in whom guideline levels were not achieved pre-hospital. Similarly, a retrospective review of 851 patients on targeted ventilation ($PaCO_2$ 30-39 mmHg) at the emergency department also found decreased mortality (21.2% vs. 33.7%) in comparison with ventilation achieved outside the target range.[21] Therefore, pre-hospital normoventilation should be ensured. However, in cases with signs of potential brain herniation, transient hyperventilation may be an option. There is strong evidence that capnography can

affect the outcome. Therefore, continuous capnography is recommended in all ventilated TBI patients.[21,22] As $ETCO_2$ values may be confounded by low tissue perfusion, long-term ventilation should be guided by the frequent sampling of blood gases.[23] The frequency of blood gas sampling is yet to be quantified.

Oxygenation and ventilation can become difficult if there are other major injuries, particularly chest injuries, aspiration of gastric contents or acute pulmonary edema. Appropriate positive end expiratory pressure (PEEP) should be used. There is no evidence that 10 mmHg PEEP impairs cerebral blood flow or elevates intracranial pressure (ICP) and, indeed, ICP may improve with improved oxygenation.[24]

Hypotension/hypertension

Hypotension is a major secondary brain insult and studies have demonstrated that a single episode of hypotension dramatically worsens the outcome.[5] Hypotension should be treated with fluid resuscitation to euvolaemia and, if necessary, vasopressors are used to maintain optimum blood pressure. According to BTF, prior to the insertion of an ICP monitoring, a MAP ≥ 80 mmHg is recommended. The rationale for a MAP ≥ 80 mmHg is to maintain cerebral perfusion pressure (CPP) ≥ 60 mmHg for a treatment threshold of ICP > 20 mmHg.[25] Following the insertion of an ICP monitoring, the management of MAP will be directed by the ICP/CPP values.

However, recent studies have challenged the lower limit to define hypotension in patients with isolated head injury.[26,27] In a study of 15,733 patients with moderate to severe head injury, the value of systolic blood pressure of 110 mmHg or less is computed as the best fit model for hypotension when mortality was taken as the outcome measure.[26] In another study, maintaining a systolic blood pressure of approximately 120 mmHg has been found to be more efficacious to minimize the secondary brain injury.[27] Therefore, further large clinical trials are needed to support these findings. It is recommended that patients with severe TBI have arterial and central venous lines placed as soon as practical.

Hypertension may be a physiological response produced due to sympathetic surge, and may signify a compromised cerebral perfusion. Hypertension should not be treated unless a cause has been excluded or treated and the systolic blood pressure is >180-200 mmHg or the MAP is >110-120 mmHg.[28] Lowering an increased blood pressure, as a compensatory mechanism to maintain an adequate CPP, exacerbates cerebral ischemia. For the treatment of hypertension to achieve CPP targets, an infusion of short-acting beta blocker is very useful, which maintains blood pressure. These agents do not cause cerebral vasodilatation when compared with nitrates and calcium channel blockers and, therefore, do not increase the cerebral blood volume and ICP.[28]

Fluid resuscitation

Further to this, the prompt resuscitation with fluids is of paramount importance to preserve optimum CPP [Table 4]. However, the choice of fluids is still a matter of great conflict. Some studies have suggested that hypertonic fluids might suppress the biomarkers of TBI and are correlated with better outcome.[29] Many studies have investigated the effect of various fluids on pre-hospital resuscitation. No studies have reported better survival and functional outcomes over the use of isotonic crystalloids.[30] Further to this, a multicenter randomized control trial (RCT) of 1087 patients (15 years or older with blunt trauma who did not meet the criteria for hypovolemic shock) with severe TBI (GCS 8 or less) showed that even the initial resuscitation with hypertonic saline or hypertonic saline/dextran, compared with normal saline, were found to be comparable in terms of 6-month neurologic outcome or survival.[31] However, this study was terminated by the data and safety monitoring board after randomization of 1331 patients. On the other hand, post hoc analysis (460 patients) of a multicenter RCT study titled "Saline Albumin Fluid Resuscitation Trial (SAFE)" highlighted that the use of albumin was associated with the higher mortality data in patients with severe head injury.[32] Among patients with severe brain injury (GCS 3-8), 41.8% patients died in the albumin group as compared with 22.2% patients in the saline group; however, among patients with GCS scores of 9–12, death occurred in eight of 50 patients in the albumin group (16.0%) and eight of 37 patients in the saline group (21.6%).[32] Based on these findings, colloids should not be used as the first choice of fluid, especially in patients with severe TBI, and isotonic crystalloids seem to be a safer choice and are cost-effective as well. The role of colloids in mild to moderate severe head injury is at present inconclusive and therefore needs well-designed RCTs.

PH

Hypothermia has some protective effects in TBI by reducing ICP, cerebral metabolic demands, decreasing disruption of the blood–brain barrier and inhibiting the inflammatory cascade. Induced hypothermia is now an accepted measure to improve outcome following anoxic brain injury associated with cardiac arrest, but its benefits in TBI are inconclusive at present.[33] In a systemic review (12 RCTs), data support the use of early prophylactic mild-to-moderate hypothermia in patients with severe TBI (GCS score ≤8) to decrease mortality and improve the rates of good neurologic

recovery.[33] However, this review advocates for early use (emergency department) of therapeutic hypothermia and also recommends a long-term or goal-directed cooling protocol, in which cooling was continued for at least 72 h and/or until stable normalization of ICP for at least 24 h was achieved. On the other hand, in an epidemiological study (level III evidence) of 21,023 patients, 44.6% patients presented with PH.[34] Moreover, hypothermic patients in comparison with normothermic patients required 26% more fluid resuscitation and 17% more total blood products in the emergency department. PH was found to be independently associated with increased adverse events, including need for a transfusion, mortality, incidence of adult respiratory distress syndrome and pneumonia.[34] Similarly, a retrospective review of 1281 patients highlighted that presence of hypothermia (<35°C) on Intensive Care Unit (ICU) admission was found to be an independent factor to increase the mortality. In addition, penetrating injury, Injury Severity Score ≥25 and exploratory laparotomy before admission were found to be independent risk factors for the development of hypothermia on ICU admission.[35] The impact of preventative measures used to avoid the development of hypothermia and the effectiveness of measures for restoring normothermia warrant further investigation.

CONCLUSION

Severe TBI patients impose very high mortality and morbidity and, strikingly, a substantial portion of these bad outcomes can be prevented by prompt evaluation and pre-hospital as well as early emergency management of such cases [Table 5]. Based on the evidence, majority of the management strategies comprise of rapid correction of hypoxemia and hypotension, the two most important predictors for mortality. However, there is still a need to define the goals for the management of hypotension and inclusion of newer difficult airway carts as well as proper monitoring devices for ensuring better intubation and ventilatory management. Isotonic saline should be used as the first choice for fluid resuscitation. The PH has more adverse effects and should therefore be avoided.

Most of the management trials published after 2007 have focused mainly on the treatment as well as the prevention strategies for secondary brain injury. The results of these trials would certainly be adopted by new standardized guidelines and, therefore, may have a substantial impact on the pre-hospital management in patients with TBI.

Table 5: Keys issues in the pre-hospital management of TBI patients

Issue	Conclusion
Transport (air vs. ground)	Similar transportation time for initiating ICU better overall outcome with air lift
Personals (physician vs. paramedics)	Paramedics can be utilized with similar results slightly better outcome with physician-lead transport
Intubation	RSI intubation at PH improves outcome maintenance of SpO_2 >93%, use of BMV at this point inclusion of difficult airway kits (video laryngoscopes and fiberoptic)
Monitoring	Pulse oximetry-failure rate considerations capnometry to verify ETT placement long-term ventilation (blood gas guided) blood pressure measurement
Ventilation	Normoventilation ($ETCO_2$-30-35) PH hyperventilation-bad outcome (avoid) transient hyperventilation in cases of brain herniation
Hypotension	Re-defining BP goals Systolic 90 versus 110 or 120
Fluids	Isotonic crystalloid-first choice albumin-high mortality data in severe TBI hypertonic saline/dextran-no benefit over isotonic saline vasopressor to maintain optimal CPP No preference on vasopressors
PH hypothermia	Recent data-more adverse effects proven effect in patients with cardiac arrest rewarming goals should be defined

TBI – Traumatic brain injury; ICU – Intensive care unit; CPP – Cerebral perfusion pressure

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