



The management of severe traumatic brain injury in the initial postinjury hours – current evidence and controversies

Iftakher Hossain^{a,b,*}, Elham Rostami^{c,d,*} and Niklas Marklund^e

Purpose of review

To provide an overview of recent studies discussing novel strategies, controversies, and challenges in the management of severe traumatic brain injury (sTBI) in the initial postinjury hours.

Recent findings

Prehospital management of sTBI should adhere to Advanced Trauma Life Support (ATLS) principles. Maintaining oxygen saturation and blood pressure within target ranges on-scene by anesthetist, emergency physician or trained paramedics has resulted in improved outcomes. Emergency department (ED) management prioritizes airway control, stable blood pressure, spinal immobilization, and correction of impaired coagulation. Noninvasive techniques such as optic nerve sheath diameter measurement, pupillometry, and transcranial Doppler may aid in detecting intracranial hypertension. Osmotherapy and hyperventilation are effective as temporary measures to reduce intracranial pressure (ICP). Emergent computed tomography (CT) findings guide surgical interventions such as decompressive craniectomy, or evacuation of mass lesions. There are no neuroprotective drugs with proven clinical benefit, and steroids and hypothermia cannot be recommended due to adverse effects in randomized controlled trials.

Summary

Advancement of the prehospital and ED care that include stabilization of physiological parameters, rapid correction of impaired coagulation, noninvasive techniques to identify raised ICP, emergent surgical evacuation of mass lesions and/or decompressive craniectomy, and temporary measures to counteract increased ICP play pivotal roles in the initial management of sTBI. Individualized approaches considering the underlying pathology are crucial for accurate outcome prediction.

Keywords

coagulopathy, emergency department, intracranial pressure, prehospital management, surgical treatment, traumatic brain injury

INTRODUCTION

Traumatic brain injury (TBI) affects approximately 70 million people globally [1]. With ageing populations, the epidemiology of TBI is changing worldwide, especially in high-income countries [2^{••}]. Thus, more patients with TBI have comorbid medical conditions making their management more challenging. The survivors of severe TBI (sTBI) are often left with debilitating deficits in motor, sensory, and cognitive functions with marked impact on their quality of life [3]. After the introduction of international treatment guidelines [3–5], the acute mortality decreased significantly. Disappointingly, however, during the last two decades TBI outcomes or acute mortality have not improved further [6].

The initial impact may result in immediate death to neuronal and nonneuronal cells and may

^aNeurocenter, Department of Neurosurgery, Turku University Hospital, Turku, Finland, ^bDepartment of Clinical Neurosciences, Neurosurgery Unit, University of Cambridge, Addenbrooke's Hospital, Cambridge, UK, ^cDepartment of Medical Sciences, Section of Neurosurgery, Uppsala University, Uppsala, ^dDepartment of Neuroscience, Karolinska institute, Stockholm and ^eDepartment of Clinical Sciences Lund, Neurosurgery, Lund University, Department of Neurosurgery, Skåne University Hospital, Lund, Sweden

Correspondence to Niklas Marklund, MD, PhD, Department of Clinical Sciences Lund, Neurosurgery, Lund University, Department of Neurosurgery, Skåne University Hospital, Lund, Sweden.

Tel: +46 72 595 0261; e-mail: niklas.marklund@med.lu.se

*These authors contributed equally to this work.

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KEY POINTS

- The primary goal of prehospital management of severe traumatic brain injury (sTBI) is to minimize the secondary insults.
- Prehospital and emergency department (ED) managements of sTBI should adhere to Advanced Trauma Life Support (ATLS) principles and to prioritize airway control, stable blood pressure, spinal immobilization, and correction of impaired coagulation, respectively.
- Noninvasive techniques could help to identify raised intracranial pressure (ICP) and osmotherapy as well as hyperventilation could be applied as temporary measures to reduce ICP.
- Surgical evacuation of mass lesions and/or decompressive craniectomy are the key measures to counteract increased ICP in the acute management of sTBI.
- Treatment of sTBI patients with very low Glasgow Coma Scale (GCS) and poor pupillary reactivity remains a matter of debate, not least in the elderly population.

injure blood vessel causing extra- or intraparenchymal hemorrhages. This primary injury is exacerbated by early pathophysiological changes in the brain that include a complex cascade of secondary injury mechanisms. Since the injured brain is highly vulnerable to systemic insults such as hypoxia, hypotension, and coagulopathy, a primary goal of prehospital management of sTBI is to minimize these secondary insults. Upon arrival to the emergency department (ED), stabilization of vital parameters must be continued, and many potential treatment opportunities be considered simultaneously. Further refinement of the acute management of sTBI focusing on the early identification and minimization of the secondary insults is needed to reduce the acute mortality.

The aim of this narrative review is to provide a summary of recent studies reflecting the novel strategies, controversies, and challenges of the acute management in the critical initial postinjury hours of sTBI.

SEARCH CRITERIA

A literature search was performed in PubMed, Scopus, Google Scholar and ISI Web of Knowledge for articles in English with the words “traumatic brain injury” together with one or a combination of the words “prehospital management”, “emergency department management”, “secondary injury”,

“coagulopathy” and “intracranial pressure”. The search included recent guidelines, meta-analyses, randomized controlled trials (RCTs) and systematic reviews. The search was restricted mainly to literature published in English during the last 18 months.

PRE-HOSPITAL MANAGEMENT

The initial management of patients with TBI should be performed according to the Advanced Trauma Life Support (ATLS) principles of managing airway, breathing, and circulation, followed by a rapid neurologic exam [7]. All patients with a Glasgow Coma Scale (GCS) of ≤ 8 should liberally be intubated [8], although the use of a strict threshold GCS to dictate intubation remains controversial [9]. Major systemic, extracranial injuries often mandate intubation in the prehospital setting [2^{***}]. Time to hospital should be minimized although in unstable patients with hypotension and/or hypoxia, stabilization and rapid sequence intubation (RSI) at the scene of the accident may be preferable. Plausibly, the chances of survival can be influenced by the expertise and experiences of, for example, paramedics or emergency doctors [10]. While the presence of an on-scene physician experienced in airway, fluid, and blood pressure management, for example, decreased mortality rates, the benefits of physician-staffed prehospital management on sTBI outcomes have not been firmly established [11–13].

After prehospital stabilization, the sTBI patients must promptly be transferred to definitive care while maintaining adequate oxygen saturation (SpO_2 ; treatment goal $>90\%$). Presumably even more important, the systolic blood pressure must be maintained at ≥ 100 – 110 mmHg at all times. An isolated TBI does not result in hypotension, and if present other differential causes must be considered. Permissive hypotension, a treatment option during the initial resuscitation of polytrauma patients with significant bleeding [14], aims at minimizing further blood loss and tissue damage by reducing the pressure exerted on injured blood vessels. However, this strategy may be detrimental for sTBI patients, as hypotension with systolic blood pressure <90 mmHg significantly impairs the outcome [15].

The incidence of dual diagnosis of brain and spinal cord injuries (SCIs) can range from 25% to more than 60% in sTBI, particularly in cases involving motor vehicle collisions or falls, emphasizing the significance of spinal motion restriction (SMR) in prehospital care [16]. Although the use of spinal immobilization by a rigid neck collar is still common, there is little evidence showing its benefit [17,18]. In fact, cervical fixation with a stiff collar is suboptimal and it could, for example, occlude

venous return and thus increase intracranial pressure (ICP). Cushion blocks on either side of head or vacuum mattress are preferable prehospital options. There should be a suspicion of an associated SCI in all sTBI patients [4]. Assessment of the GCS score, pupillary reactivity, and focal neurological deficits is performed at the scene of the accident, and preferably on arrival at the hospital (the postresuscitation GCS). Patients presenting with low GCS scores, including extensor posturing and/or dilated pupil (s), have a high likelihood of increased ICP and/or cerebral herniation, requiring temporary ICP-reducing measures. Hyperosmolar solutions such as mannitol or hypertonic saline (HTS) [2[■]], and mild-to-moderate hyperventilation to, but not less than, a p_aCO_2 of 4.0–4.5 kPa due to a risk of cerebral vasoconstriction exacerbating ischemic injuries, are ICP-lowering options [19].

Based on the findings of the largest randomized trial in TBI to date, CRASH-3 [20], and subsequent studies [21,22], early administration of tranexamic acid (TXA) within 3 h postinjury is safe and recommended for moderate head injuries, as well as mild injuries with bleeding observed on acute CT scans [23]. Although recent systematic reviews found no significant impact on mortality or disability, early TXA administration is still considered in sTBI patients due to its favorable adverse side effect profile and potential benefits. This recommendation takes into account the challenges of accurately classifying TBI in the prehospital setting, as well as its use in polytrauma [22,24].

EMERGENCY DEPARTMENT MANAGEMENT

Upon arrival to the emergency department (ED) the ATLS is reassessed with a focus on airway management, maintaining mean arterial pressure (MAP) at 80 mmHg or higher, immobilization of the spine (time on spine board should be minimized, and must be <2 h), and securing venous access as well as placement of an arterial line and an indwelling urinary catheter (see Table 1 for thresholds of target goals for key physiological parameters). The airway is secured according to local institutional protocols, although endotracheal intubation is mandatory in any sTBI patient. Induction agents such as propofol should be cautiously used, possibly only in conjunction with induction inotropes, in view of the risk of systemic hypotension resulting in an impaired cerebral blood flow (CBF) [19]. Ketamine is a commonly used option, although early studies using ketamine in sTBI patients implied that it could increase ICP and decrease cerebral perfusion pressure (CPP) [25[■]]. To date, an ICP-reducing effect

Table 1. Thresholds for avoiding secondary insults

ICP < 20–22 mmHg
CPP \geq 60 mmHg ^a
Systolic BT >100 mmHg
pO_2 >10 kPa
O ₂ saturation >95%
pCO_2 4.5–5.5 kPa
Temperature < 38°C
β -Glucose 5–10 mmol/l
S-Na 140–150

ICP, intracranial pressure.

^aThe recommended target cerebral perfusion pressure (CPP) for favorable outcomes is between 60 and 70 mmHg. The optimal minimum threshold, whether it is 60 or 70 mmHg, depends on the patient's autoregulatory status [15,79].

of ketamine is suggested and in hypotensive sTBI patients ketamine may be preferred due to less risk of hypotension compared with other induction agents [26]. Barbiturates can cause hypotension and are not recommended for intubating sTBI patients [27[■]].

While intraparenchymal or intraventricular ICP monitoring is gold standard, those techniques require surgical insertion. In the ED, noninvasive techniques for the estimation of raised ICP such as optic nerve sheath diameter (ONSD) measurement, pupillometry and transcranial Doppler (TCD) are emerging. These tools may be useful in the detection of intracranial hypertension and may be applied as triage tools in the ED [28].

Intracranial pressure lowering therapies

When signs of increased ICP and/or cerebral herniation are present, temporary ICP-reducing measures must be considered (Table 2). Any coagulation disorder must promptly be corrected (*vide infra*). Obviously, head-and whole-body imaging by multislice CT scanning, also evaluating the entire spine, is acquired immediately following stabilization. Depending on the CT findings, prompt surgical evacuation of mass lesions or a primary decompressive craniectomy (DC) may be needed (e.g., see Fig. 1) [29[■]]. A CT-angiogram should be performed in suspicion of traumatic cerebrovascular injuries such as skull base fractures involving the carotid canal [15]. In sTBI patients, ICP and CPP monitoring is also needed - either following initial surgical treatment, or as a primary measure [2[■]]. Extensive surgeries on other organ system should be avoided, or minimized to the extent possible, in the emergent phase [30].

Table 2. ICP lowering options

Always consider – is there a mass lesion in need of evacuation?

1. Head in neutral position, avoid neck extension – optimize jugular vein effluent
2. Elevated head of bed (HOB) at 15–30°, treat fever
3. Optimize pain relief and sedation
4. Normal or negative fluid balance
5. Osmotherapy (3% NaCl or mannitol; 0.25–0.5 g/kg of 20% solution over 10–15 min)
6. Optimize S-Na 145–150 mmol/l
7. High suspicion of seizures, consider continuous EEG monitoring
8. Increase analgesia and sedation
9. Increase S-Na to 150–155 mmol/l, consider moderate hyperventilation $p\text{CO}_2$ 4.0–4.5 kPa
10. Consider adding ketamine
11. Barbiturates
12. Mild hypothermia 34–35°C
13. Decompressive hemicraniectomy

ICP, intracranial pressure.

Since CPP is a surrogate marker for cerebral blood flow (CBF), it should be maintained by optimizing MAP using fluids, inotropes, and vasopressors, and/or by decreasing ICP. To decrease ICP, bedside manoeuvres such as sedation, cerebrospinal fluid (CSF) drainage, and, in refractory cases, barbiturate coma or DC can be used (Table 2) [15,31,32]. Of note, while several potentially neuroprotective agents that have been investigated in clinical trials, all failed to show benefit for sTBI [33,34].

Hyperosmolar therapy by 3% to 30% of HTS or mannitol for ICP lowering has been extensively studied. While reduced ICP can be achieved with hyperosmolar therapy, there is no firm evidence about the most effective agent, its administration, or timing. Whether HTS or mannitol is more effective for reducing ICP has been debated for over 20 years and despite several systematic reviews, including two Cochrane reviews and five meta-analyses, the optimal choice remains unsettled [35]. Lately, the COBI RCT showed that in moderate to severe TBI patients, continuous infusion of 20% HTS compared with standard care did not improve neurological outcome at 6 months postinjury [36]. An RCT in the UK entitled “Sugar or Salt” comparing the use of bolus HTS and mannitol for the management of elevated ICP in the neurointensive care unit (NICU) setting is currently underway [37]. However, there are no RCTs targeting the prehospital and ED settings. At present, osmotherapy should only be used as an interim measure for emergent ICP-



FIGURE 1. CT Image of a patient with severe traumatic brain injury. A young patient with life-threatening TBI presenting with unilaterally dilated pupils on the right side, and a GCS score of 5. The CT image shows diffuse brain swelling in addition to brain contusions (marked with *), effaced basal cisterns (indicated with a black arrow) and a pseudosubarachnoid hemorrhage sign (indicated with a white arrow) typically seen in patients with markedly increased intracranial pressure. This patient received emergent intubation, hyperosmolar therapy, mild hyperventilation and was subjected to an acute decompressive craniectomy. After prolonged neurocritical care therapy, the patient had an excellent outcome at 6 months postinjury. GCS, Glasgow Coma Scale; TBI, traumatic brain injury.

lowering, by bolus administration and not as a continuous therapy [38].

RED BLOOD CELL TRANSFUSION AND ERYTHROPOIETIN

The optimal hemoglobin (Hb) level triggering transfusion for sTBI patients is controversial. A recent RCT showed that sTBI patients transfused “liberally” (at Hb 9 g/dl) had less posttraumatic vasospasm, lower hospital mortality and better neurological status at 6 months postinjury compared to patients transfused “restrictively” (at Hb 7 g/dl) [39]. While current recommendations argue against a liberal transfusion strategy at Hb 10 g/dL, a recent meta-analysis found no difference in mortality between restrictive (Hb <7 g/dl) and liberal (Hb <10 g/dl)

thresholds ($P=0.79$). At present, best available evidence suggest a Hb threshold for transfusion between 7 and 9 g/dl [40^{***}].

Erythropoietin (EPO) is a potential neuroprotectant with effects on many organ systems and has thoroughly been investigated in clinical and experimental TBI. A recent meta-analysis of 10 RCTs of 2402 TBI patients suggested a reduced mortality with EPO [41^{***}]. However, EPO has a poor blood-brain barrier penetration and rather mixed efficacy in available RCTs. At present, EPO cannot be recommended in the routine management of sTBI patients [42].

CORRECTION OF IMPAIRED COAGULATION

Impaired coagulation is a crucial yet potentially treatable secondary insult in sTBI [43]. The proportion of sTBI patients on drugs targeting coagulation is increasing due to their increasing age. TBI *per se* also induces a coagulopathy, and 2/3 of patients in the ED have abnormal coagulation with risk factors being GCS ≤ 8 , base excess (BE) ≤ -6 , hypothermia and hypotension. Moreover, low fibrinogen levels should be corrected.

Management of low platelet count and platelet dysfunction

A platelet count $\leq 100 \times 10^9/l$ is associated with increased mortality. Moreover, platelet dysfunction is common regardless of the use of platelet inhibitors. However, while platelet transfusion attenuates hematoma expansion there is no clear benefit on outcome [44,45], and the potential harms and risks of platelet transfusion of must be considered. Platelet transfusion, desmopressin, and tranexamic acid remain options for sTBI patients requiring surgery.

Anticoagulants (warfarin/direct oral anticoagulant)

In most studies, mortality and hemorrhage expansion are worse with warfarin when compared to direct oral anticoagulants (DOACs). In sTBI patients on warfarin, rapid reversal using Prothrombin complex concentrate (PCC) with the addition of Vitamin K1 (Vit K) is mandatory. Note that Vit K in itself is too slow, and that fresh-frozen plasma may not be well tolerated in elderly patients due to the volume load.

Due to the short half-lives of DOACs [46], up to 80% of the therapeutic effects will be gone with normal renal function, if the last dose taken at least 24 h prior to urgent surgery [47]. The antidote Idarucizumab should be administered to sTBI patients on Dabigatran. The antidote andexanet alpha, a modified recombinant inactive human factor Xa (FXa) was designed to reverse FXa inhibitors available for patients on apixaban or rivaroxaban [48]. To date, PCC administration is recommended for sTBI patients since the indications of andexanet alpha in sTBI have not been defined [49]. It should be noted, however, that it was recently found superior to standard care in FXa inhibitor-treated patients with spontaneous intracerebral hemorrhage, and its role in sTBI patients may be revisited in the near future (Fig. 2) [46,50].

STEROIDS

There is level 1 evidence that dexamethasone is ineffective in improving outcomes and is associated with significantly increased complication rates and mortality, and should thus be avoided in the acute management of sTBI [15].

SEIZURE MANAGEMENT

There is no evidence that the prophylactic use of any antiepileptic drug prevents late post traumatic

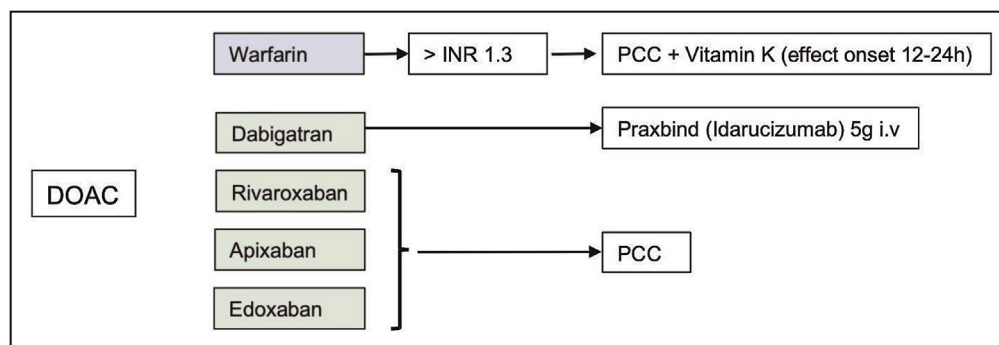


FIGURE 2. Reversal strategy for TBI patients on anticoagulant therapy. Recommended reversal strategy of oral anticoagulants using multifactor prothrombin complex concentrates (PCC) and antidotes in patients with severe TBI. DOAC, direct oral anticoagulant; INR, international normalized ratio; TBI, traumatic brain injury.

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seizures (PTS) or improve outcome. Phenytoin is recommended in the recent Brain Trauma Foundation guideline since it is effective in decreasing early PTS, although early PTS is not correlated to worse outcome [11]. Levetiracetam is an alternative with a lower incidence of adverse effects [38^{*}]. The ongoing MAST trial aims to define best practice in the use of antiepileptic drugs following TBI [51].

TEMPERATURE MANAGEMENT

Temperature management in TBI addresses three main issues: the use of therapeutic hypothermia as a neuroprotectant, use of hypothermia to reduce ICP, and the avoidance of hyperthermia [19]. When summarizing several RCTs on hypothermia for sTBI, including the POLAR [52] and EURO THERM [53] studies, ICP is reduced. However, the outcome is not improved, in fact in the EURO THERM study the mortality was higher and the functional outcomes were worse. Current practice, based on limited evidence, focuses on reducing significant elevations in temperature while active hypothermia should only be evaluated in the setting of RCTs [15].

INITIAL NEUROCRITICAL CARE MONITORING

One of the cornerstones of acute sTBI management is the monitoring of ICP, which has been shown to reduce in-hospital and 2-week postinjury mortality [15]. Advanced neuromonitoring including, for example, brain tissue oxygen (PbtO₂) and microdialysis can be used in ICP reducing treatment algorithms [32,54]. A comprehensive discussion on NICU multimodality neuromonitoring is beyond the scope of this review and has been extensively covered in previous literature [55^{*},56,57^{**},58^{*},59].

SURGICAL PERSPECTIVES

Immediate removal of mass lesions and/or decompressive craniectomy (DC) is often lifesaving, and may be the most crucial treatment in the early management of sTBI patients. Mass lesions consists of epidural (EDH), acute subdural hematoma (ASDH) and cortical contusions (CCx). At time of initial surgery, ICP monitoring may be initiated. Both intraparenchymal devices and external ventricular drain (EVD) can be used for ICP monitoring, however, the EVD offers the benefit of draining CSF. In patients with effaced basal cisterns and/or marked brain swelling presenting in a poor neurological state, a primary DC is considered. A large frontotemporoparietal DC (15 cm in diameter) is recommended, as it reduces mortality and improves neurological outcomes [60].

Surgical evacuation is the primary treatment for EDHs. They are often associated with skull fractures, occur in young individuals and should be surgically evacuated regardless of GCS if exceeding 30 cm³, or if they present with coma and/or anisocoria. Non-operative management may be considered for smaller hematomas in alert patients monitored by serial CT scans and close neurological surveillance.

Surgical intervention is generally recommended for ASDHs with a thickness ≥ 10 mm or a midline shift ≥ 5 mm. However, smaller ASDHs may still require surgery if certain conditions are met: a drop of two or more points in the GCS, presence of asymmetric or fixed and dilated pupils, or ICP exceeding 20 mmHg. A recent CENTER-TBI study found no evidence that an aggressive surgical approach in ASDH yielded better functional outcomes. However, treatment for patients with ASDH varied across different centers and the group that underwent acute surgery had a lower initial GCS score, larger volumes of ASDHs, and more often large contusions. The observational CENTER-TBI project has provided a unique increase in TBI knowledge. However, it was not designed to firmly evaluate treatment efficacy and changed management routines cannot be based solely on this ASDH study [61^{**}].

The Randomised Evaluation of Surgery with Craniectomy for patients Undergoing Evacuation of Acute SubDural Haematoma (RESCUE-ASDH) trial comparing DC versus craniotomy showed that patients with traumatic ASDH who underwent craniotomy or primary DC (as a primary operation), disability and quality-of-life outcomes were similar with the two approaches with no added benefit of performing DC [29^{**}].

Cerebral contusions (Ccx) are common in sTBI and usually coexist with traumatic subarachnoid hemorrhage (tSAH) and ASDH. Many progress – “blossom” - within the first 24 h [62^{*}]. In localized Ccx, immediate craniotomy and hematoma removal is indicated in sTBI patients showing progressive neurological deterioration and significant mass effect on CT, with significant midline deviation and compressed basal cisterns. As an alternative, a primary DC can be performed, particularly in Ccx accompanied by diffuse swelling [63]. It should be noted that DC significantly increases the risk of contusion expansion [64].

Penetrating head injury are mainly caused by gunshot and stab wounds. Gunshot wounds (GSWs) have higher mortality than stab wounds and have a higher incidence of intracerebral hemorrhage (ICH), and raised ICP [65,66]. Stab wounds and GSWs are also commonly associated with vascular injuries [67] that need to be evaluated by digital subtraction angiography (DSA), which is superior to

CT-angiography [68]. Coagulopathy combined with cerebrovascular injury forms a distinct endophenotype [69]. Patients with penetrating head injuries are prone to significant brain swelling where primary DC is a treatment option. Debridement of necrotic brain tissue, and wound revisions, are surgical options where excessive removal of foreign material should be avoided. There is a significant risk of posttraumatic epilepsy and infectious complications in sTBI patients with penetrating injuries and prophylactic anticonvulsants and antibiotics are recommended [69,70].

Recently, cisternostomy has emerged as a potential surgical approach for treating sTBI. This neurosurgical method includes, for example, extradural anterior and posterior clinoidectomy along with basal cisternostomy with opening of membrane Lilliequist and lamina terminalis [71]. A recent, criticized, RCT compared DC with cisternostomy and found similar outcomes [72²²]. Importantly, this technique requires the expertise of highly skilled and trained neurosurgeons, surpassing the level typically available during on-call shifts handled by trainees [72²²,73,74²⁴].

Finally, hinge craniotomy could offer an intermediate intervention between treatment-refractive medical therapy and traditional DC, however, no RCT currently exists for this surgical technique [75].

PROGNOSTIC FACTORS

Outcome is difficult to predict since sTBI is a markedly heterogeneous disease. A limitation of classification based on GCS is that the underlying pathology is not considered. However, severity of TBI is one the most robust negative predictive factors followed by advanced age, absence of pupillary reaction, and initial hypotension [76]. The timing of GCS assessment is crucial since rapid alterations are common in the initial postinjury hours. Patients with low GCS persisting from the scene with bilaterally fixed dilated pupils (BFDP) have a dismal chance of survival [77]. The mortality in sTBI patients with a GCS of 3 and BFDP in the field is 90–95%, with merely 2–3% achieving a good functional recovery [78]. Patients with GCS ≤ 8 after resuscitation also face high mortality rates although many survivors reach a favorable recovery. Currently, there are no national guidelines recommending prognostic models for individual patient care decisions in sTBI and existing prognostic models, such as International Mission on Prognosis and Analysis of Clinical Trials (IMPACT), are primarily designed for research purposes and not widely implemented in clinical practice. Each patient should be approached individually, considering all

the above-mentioned factors in addition to the underlying TBI pathology. In sTBI patients with a low GCS caused by an EDH, evacuation is more likely to result in a favorable outcome than if the cause is, for example, multifocal contusion and/or diffuse injuries. Furthermore, additional factors such as comorbidities and additional systemic injuries should be considered in the decision-making for advanced neuromonitoring and further treatment. These factors play a significant role in determining the appropriate approach to treatment and the potential outcomes for sTBI patients. Additionally, while the sTBI population is aging, the biological age is also shifting and patients ≥ 65 years old today may be healthier than in the recent past. Many older adults with TBI respond positively to aggressive management and rehabilitation, arguing that chronological age and TBI severity alone are insufficient prognostic markers. Making decision of palliative care or end-of-life decisions for this population is ethically complex and there are no specific guidelines for managing TBI in the geriatric population. Recent developments of frailty indexes may aid in prognostication in the elderly population.

Overall, balancing the goal of maximizing functional independence with the patient's wishes and quality of life becomes critical in decision-making processes. Additionally, the local or regional availability of rehabilitation services can significantly impact the functional outcome of sTBI patients.

There are many controversies surrounding prognostication and end-of-life decisions in sTBI. Legal and cultural variations may influence the decision-making process, including the acceptance of palliative care and withdrawal of life-sustaining treatments. Understanding and respecting these variations is crucial when making decisions for sTBI patients.

CONCLUSION AND FUTURE DIRECTION

The acute managements in the critical initial postinjury hours of sTBI is focused on preventing any possible secondary insults. Advancement of the prehospital and the ED care include rapid correction of impaired coagulation, noninvasive techniques to identify raised ICP in addition to the neurological exam, and rapid management of presumed or established increases in ICP and/or cerebral swelling. Upcoming clinical trials and large observational studies might provide stronger evidence for the use of osmotherapy, antiepileptic medications, and temperature management. Awaiting the emergence of neuroprotective therapies, multimodal monitoring, and treatment of secondary injury factors during the initial postinjury hours remain the

best treatment of patients with sTBI. A comprehensive approach tailored to each individual is recommended for the decision-making process of older patients with sTBI.

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Conflicts of interest

There are no conflicts of interest.

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- of outstanding interest

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