



Review article

Emergency department management of traumatic brain injuries: A resource tiered review



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ABSTRACT

Introduction: Traumatic brain injury is a leading cause of death and disability globally with an estimated African incidence of approximately 8 million cases annually. A person suffering from a TBI is often aged 20–30, contributing to sustained disability and large negative economic impacts of TBI. Effective emergency care has the potential to decrease morbidity from this multisystem trauma.

Objectives: Identify and summarize key recommendations for emergency care of patients with traumatic brain injuries using a resource tiered framework.

Methods: A literature review was conducted on clinical care of brain-injured patients in resource-limited settings, with a focus on the first 48 h of injury. Using the AfJEM resource tiered review and PRISMA guidelines, articles were identified and used to describe best practice care and management of the brain-injured patient in resource-limited settings.

Key recommendations: Optimal management of the brain-injured patient begins with early and appropriate triage. A complete history and physical can identify high-risk patients who present with mild or moderate TBI. Clinical decision rules can aid in the identification of low-risk patients who require no neuroimaging or only a brief period of observation. The management of the severely brain-injured patient requires a systematic approach focused on the avoidance of secondary injury, including hypotension, hypoxia, and hypoglycaemia. Most interventions to prevent secondary injury can be implemented at all facility levels. Urgent neuroimaging is recommended for patients with severe TBI followed by consultation with a neurosurgeon and transfer to an intensive care unit. The high incidence and poor outcomes of traumatic brain injury in Africa make this subject an important focus for future research and intervention to further guide optimal clinical care.

African relevance

- Traumatic brain injury is the leading cause of disability in for age < 40 y, with catastrophic social and economic costs.
- Traumatic brain injury is a significant and growing cause of death and disability in Africa, in part due to increasing incidence of road traffic injuries.
- A systematic approach and prevention of secondary injury is key to TBI management and achievable in all resource settings.

Introduction

Traumatic brain injury (TBI) is a growing international public

health problem. In injured persons, TBI results in the most death and disability globally [1]. The true scope of the problem is not known; 2016 Global Burden of Disease data estimate just over 27 million cases of TBI annually, or a rate of 369 per 100,000 persons [2,3]. Deriving the incidence of TBI from road traffic incidents results in the much higher global estimate of 55.9 million cases annually [1]. The African burden of TBI is estimated to be approximately 8 million cases per year (801 per 100,000 persons) when derived from estimates of road traffic incidents [1].

Approximately 90% of global deaths from trauma occur in low- and middle-income countries (LMIC), and TBI is a contributing factor in one-third to half of these deaths [4,5]. Persons injured in road traffic incidents in the African region are more than twice as likely to suffer

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from a TBI (55%) compared to those seen in the high-income countries (HIC) of North America (25%) [1]. Additionally, patients with severe TBI are twice as likely to die in LMICs compared to HICs [6]. Dewan et al estimate 708,000 patients with TBI in the African region require a neurosurgical intervention annually [7].

TBI is the leading cause of disability in injured persons younger than 40, leading to significant social and economic impact due to high costs for treatment, rehabilitation, long-term care, and lost societal contributions [4]. The estimated economic impact will be 1.1 trillion US Dollars in gross domestic product losses in LMICs between 2015 and 2030 [8].

The true epidemiology and burden of African TBI is likely underestimated. A few retrospective observational studies provide site specific estimates. One study at a large tertiary hospital in Malawi found 18% of admitted trauma patients had a TBI with a 30% mortality rate for all TBI [9]. A retrospective analysis at a tertiary hospital in Tanzania reported 56% of TBIs were due to road traffic incidents with 17.8% of TBI patients classified as severe and a 30.7% mortality rate [10]. A retrospective review of over ten-thousand injured patients presenting to tertiary hospitals in Western Cape, South Africa identified TBI in 24% of injured patients; 27% of these TBIs were moderate to severe and required admission [11]. A retrospective study in Burkina Faso evaluated data from 183 TBI patients. Out of 110 computed tomography (CT) scans performed, traumatic injuries were present on 74%. Surgery was indicated for 15% of patients who had a CT scan and medical treatment was changed in an additional 18% [12]. The majority of TBI patients are young males (average age range 28.8–38.8 years), with the majority of TBI originating from road traffic incidents, interpersonal violence, and falls [1,4,9,10].

Classification of TBI

TBI is any injury that disrupts normal brain function and can manifest as any combination of cognitive, behavioural, motor, and sensory symptoms [13]. TBI encompasses a spectrum of disease, subdivided into mild, moderate and severe according to the Glasgow Coma Scale (GCS) [13]. Patients are classified as having mild TBI if GCS 13–15, moderate if GCS 9–12, and severe if GCS < 9 [13]. Globally, approximately 80% of TBI injuries are classified as mild, 11% as moderate, and 8% as severe [1]. The term ‘moderate’ itself is a misnomer. Mortality for patients with moderate TBI is reported at a rate of up to 15% [14,15]. Though the overall mortality is 15% for moderate TBI patients, 75% of those deaths occur in patients with an initial GCS of 9–10 [16]. There are limitations when using GCS to classify TBI including difficulty of use, inconsistent application by providers and confounding patient factors such as intoxication, drugs and polytrauma injuries [14]. Additionally, GCS is symptom based and has poor correlation with specific intracranial pathology [14,17]. Other methods of classifying TBI include other coma grades, abbreviated injury severity scale and description of injury based on anatomic location [17]. However, the majority of the literature uses the GCS scale to classify TBI despite its limitations. For consistency, the GCS categories of mild, moderate and severe TBI are used to describe the assessment and management of TBI in this review.

Methods

The objective of this paper was to provide a review of the evidence regarding management of traumatic brain injuries with an emphasis placed on the resource limited setting. A systematic review was conducted in accordance with the PRISMA guidelines [18], and the authors were guided by recommendations for resource-tiered review [19].

The initial search was performed using the PubMed online database through May 2018. The search query was designed with intent of retrieving research describing epidemiology, diagnostics and management of patients with traumatic brain injury. Full search terms can be

found in Appendix B. No restrictions were placed in regard to data, or study design for the initial search. All citations were initially exported and managed in EndNote X9.3, then transferred to a uniquely designed data collection tool in Microsoft Excel for Mac (Microsoft Corporation, Redmond, WA, USA).

The results of the initial search were then screened by title and abstract by two reviewers for final inclusion according to pre-determined inclusion and exclusion criteria. Any differences were resolved by a third reviewer. Inclusion criteria: included content on clinical care provided within the first 48 h of injury, and available in English as full text. Those that were grossly unrelated to the subject of TBI were excluded. Additionally, articles relating to chronic sequelae of TBI were excluded. Full inclusion and exclusion criteria can be found in Appendix B.

One hundred and fifteen studies were included, evaluated and qualitatively summarized to develop reviews and recommendations regarding TBI patients with an emphasis on resource limited settings (Fig. 1 in Appendix B). Additional articles were identified through references of included articles and PubMed searches for select clarification of clinical management when insufficient detail was found in included articles.

Discussion

Assessment of traumatic brain injury

History and physical

Risk stratification of patients starts with an understanding of the evidence-based mechanisms of injury, symptoms and physical exam findings associated with significant head injury. These are summarized in Table 1 by the positive likelihood ratio that the described mechanism of injury is associated with a head injury requiring prompt intervention which includes observation, admission to the hospital or ICU and/or neurosurgical intervention [20]. The injury mechanisms most commonly associated with severe head injury, even when the patient presents with symptoms of mild TBI, include pedestrian struck by a vehicle, an occupant ejected from a motor vehicle, or a fall from elevation of more than 1 m or 5 stairs [20]. Bike collisions, absence of bike helmets, and chronic alcoholism are associated with increased risk of severe intracranial injury even with mild initial presentation [21].

The presence of vomiting, particularly greater than two episodes, has higher likelihood of being associated with severe head injury [20]. Post-traumatic seizure is also positively associated with severity [21] (Table 1). Despite concern from patients and their families, recent

Table 1
High-risk history and physical exam features in adults [20].

History and physical exam features	Likelihood ratio of TBI
History - risk factors	
Pedestrian struck by automobile	LR range, 3.0–4.3
Dangerous mechanism ^b	LR 2.1; 95% CI, 1.5–2.9
Age ≥ 65	LR, 2.3; 95% CI, 1.8–3.1
Age > 60	LR, 2.2; 95% CI, 1.6–3.2
History - symptoms	
2 or more episodes of vomiting	LR, 3.6; 95% CI, 3.1–4.1
Post traumatic seizures	LR, 2.5; 95% CI, 1.3–4.3
Physical exam	
Any skull fracture	LR, 16; 95% CI, 3.1–59
Basal skull fracture ^a	LR, 6; 95% CI, 3.9–8.0
Depressed GCS: GCS = 13, GCS < 14 2 h from injury or decline in GCS	LR range, 3.4–16
Focal neurological deficit	LR 1.9–7.0

^a Defined as postauricular ecchymosis (the Battle sign), hemotympanum, cerebrospinal fluid otorrhea, or peri-orbital ecchymosis.

^b A pedestrian struck by a vehicle, an occupant ejected from a motor vehicle, or a fall from elevation of more than 1 m or 5 stairs.

articles suggest that brief loss of consciousness or post-injury headache in adults does not predict head injury severity, concussion, clinical course, or long-term cognitive impairment [21,22,23,24].

For those with evidence of mild to moderate TBI, physical exam findings can be helpful in predicting severe injuries. Focal neurologic deficit is associated with severe TBI [21,25]. Signs of a skull fracture (open, depressed, or basilar) such as hemotympanum, cerebrospinal fluid otorrhea, peri-orbital ecchymosis, or isolated basilar skull fracture identified by postauricular ecchymosis (the Battle sign) are also associated with severe TBI [20] (Table 1). Severe injuries in intoxicated patients presenting unconscious can be easily missed or attributed to intoxication. In minor TBI, an initial GCS of 13, worsening of GCS, and GCS < 14 2 h post-injury were all associated with higher likelihood of severe intracranial injury [22,25,26]. A high index of suspicion and frequent neurological assessments will help identify any worsening condition and concerning signs and symptoms.

Children, especially those under the age of 2, can suffer from TBI with a mild mechanism of injury. Additionally, children are often difficult to assess, and their significant intracranial injuries are often asymptomatic [27,28]. Signs that are known to be associated with severe intracranial injury in children include loss of consciousness [29,30], vomiting (especially when multiple episodes or combined with other indicators of severity) [31], post traumatic seizure [32], and skull fracture [28,33]. Up to two-thirds of children with severe head injuries can have other substantial injuries they will be unable to report [34].

Imaging

Not all patients with head trauma will require neuroimaging, many can be safely discharged after a thorough history and physical exam and a brief period of observation. Using many of the high-risk features discussed above, the Canadian Head CT Rule [21] and the New Orleans Criteria [35] are two validated decision rules that can assist the clinician in identifying low-risk adult patients with minor head injury who do not require neuroimaging. Patients with minor head injury (GCS ≥ 13), otherwise well-appearing on exam, and the complete absence of any criteria from the Canadian Head CT Rule and New Orleans Criteria, see Table 2, have a very low risk of severe intracranial injury (LR 0.04 and 0.08, respectively) and do not require neuroimaging [20]. Notably, the Canadian CT Rule has better diagnostic accuracy than the New Orleans Criteria [20]. It is also important to note these decision rules were validated in high-income acute care settings, and their generalizability to clinical decision-making in LMICs may be limited.

The Paediatric Emergency Care Applied Research Network (PECARN) criteria can similarly be used with paediatric patients (< 18 years of age) with minor head trauma to identify patients who do not require neuroimaging. The PECARN criteria are different for patients under the age of 2 years and those between the ages of 2 and 18 years (Table 3) [36]. A recent validation study comparing PECARN with two other paediatric head injury decision rules found superior sensitivity with PECARN, which was nearly 100% sensitive for

Table 2
Adult decision rules for neuroimaging in minor head trauma.

Canadian head CT rule [22]	New Orleans criteria [35]
<ul style="list-style-type: none"> • > 65 years old • Dangerous mechanism^a • Vomiting > 1 episode • Amnesia longer than 30 min • GCS < 15 at 2 h • Suspected open, depressed or basilar skull fracture <p>Consider CT head in patients who have 1 or more of above criteria.</p>	<ul style="list-style-type: none"> • > 60 years old • Intoxication • Headache • Any vomiting • Seizure • Amnesia • Visible trauma above the clavicle <p>Consider CT head in patients who have 1 or more of above criteria.</p>

^a Pedestrian struck by motor vehicle, occupant ejected from motor vehicle, or fall from > 1 m or > 5 stairs.

Table 3
PECARN rules for traumatic brain injury in children [36].

Patients < 2 years old	Patients 2–18 years old
<ul style="list-style-type: none"> • GCS ≤ 14 or other altered mental status^a • Severe mechanism of injury^b • Loss of consciousness > 5 s • Temporal, parietal or occipital haematoma (excluding frontal haematoma) • Palpable skull fracture • Acting abnormally per parent 	<ul style="list-style-type: none"> • GCS ≤ 14 or other altered mental status^a • Severe mechanism of injury^b • Any loss of consciousness • History of emesis • Signs of basilar skull fracture^c • Severe headache

NB: PECARN, Paediatric Emergency Care Applied Research Network.

^a Altered mental status: GCS ≤ 14, agitation, sleepiness, slow response to verbal communication, or repetitive questioning.

^b Severe mechanism: motor vehicle crash with patient ejected, death of another passenger, or rollover; pedestrian or bicyclist without a helmet struck by vehicle; falls > 0.9 m if < 2 years old, or > 1.5 m if ages ≥ 2 years old; or head struck by a high-impact object

^c Signs of basilar skull fracture: hemotympanum, retro-auricular bruising (Battle sign), periorbital bruising (raccoon eyes), cerebrospinal fluid otorrhea or rhinorrhoea

clinically significant TBIs (95% CI 84–100%) [37].

For patients with head trauma who do not meet the criteria listed above, the emergency provider should consider prompt neuroimaging with non-contrast CT. CT is recommended as the imaging of choice and can identify a subdural haematoma, epidural haematoma, subarachnoid haemorrhage, skull fractures, pneumocephalus, mass effect, and signs of cerebral oedema [38]. CT is less sensitive in detecting non-haemorrhagic lesions such as contusions, diffuse axonal injury, early cerebral oedema, and subtle injuries next to bony structures [38]. More than 24 h after injury, the sensitivity of CT imaging falls and can fail to detect radiologically small injuries [38]. However, initial urgent imaging with CT is appropriate in all patients with severe TBI [38]. CT may also show subtle signs of diffuse axonal injury, especially if presentation is delayed from the initial injury, but non-contrast magnetic resonance imaging (MRI) is the test of choice for the diagnosis of diffuse axonal injury and subacute injuries [38].

Plain radiographs of the skull have not been shown to be superior to a careful physical exam looking for focal neurologic exam or depressed skull fracture in increasing or decreasing the suspicion for TBI and thus should not be routinely used [38]. In fact, the poor sensitivity and specificity of skull films may provide a false sense of reassurance if negative [39]. Studies have shown routine skull films after trauma to be non-contributory to the evaluation, management and outcome of acute traumatic brain injury [40].

For patients with mild and moderate TBI, a common practice is to defer immediate imaging in favour of a period of observation in the emergency unit with appropriate protective interventions. Subsequent imaging is obtained if the patient’s clinical condition worsens or does not improve. For patients who do not meet criteria to avoid imaging and appear to have mild to moderate TBI, there are currently no guidelines to determine which adult patients require imaging and which are safe to observe and to delay or avoid neuroimaging [14]. This is particularly challenging when patients present acutely intoxicated. The authors recommend that emergency providers adhere to any locally adopted guidelines for neuroimaging and observation. If no guidelines exist to the authors recommend providers have a low threshold to obtain neuroimaging in patients with head trauma and altered mental status.

In resource-limited settings, the decision to pursue neuroimaging often requires transferring the patient to another facility with the appropriate imaging modality. While awaiting transfer, patients with moderate to severe TBI should be stabilised and managed as described below and any other injuries evaluated and treated. As head trauma

Table 4
Summary of emergency management of severe and moderate TBI – resource tiered recommendations.

	Key management steps
Airway	<ul style="list-style-type: none"> ● Keep airway patent; use nasopharyngeal airway (if no facial trauma) or oropharyngeal airway (if no gag reflex) when needed ● If GCS \leq 8, intubation for airway protection^a
Breathing	<ul style="list-style-type: none"> ● Maintain normoxia <ul style="list-style-type: none"> ○ Avoid hypoxia: Provide supplemental oxygen to keep oxygen saturation $>$ 90% and PaO₂ $>$ 60 mm Hg^b ○ Avoid hyperoxia, PaO₂ $<$ 300 mm Hg^b, and do not provide 100% O₂ via NRB for prolonged periods of time. ● Do not hyperventilate if assisting ventilation; <ul style="list-style-type: none"> ○ Goal PaCO₂ 35–40 mm Hg^b or EtCO₂ 35–40^a ● If signs of herniation, may temporarily increase minute ventilation until definitive care achieved <ul style="list-style-type: none"> ○ Goal PaCO₂ 30–35 mm Hg^b or EtCO₂ 30–35 mm Hg for as minimal time as possible.^a ● Avoid aspiration and place NGT if no facial trauma. ● Obtain serial ABG^b with goal PaO₂ $>$ 97 mm Hg and PaCO₂ 35–40 mm Hg
Circulation	<ul style="list-style-type: none"> ● Maintain SBP $>$ 110, MAP 80–90 mm Hg <ul style="list-style-type: none"> ○ Give isotonic fluids (NS or RL) ○ Use vasopressors (epinephrine or norepinephrine) if MAP $<$ 80 with fluids
Disability	<ul style="list-style-type: none"> ● Check blood glucose and give dextrose for hypoglycaemia. ● Elevate the head of the bed to greater than 30°. ● Loosen cervical collar if applied to decrease venous pressure. ● If seizure, bleeding, oedema or midline shift on CT^b, administer antiepileptic. <ul style="list-style-type: none"> ○ If GCS \leq 8 and reversible causes addressed, may be appropriate to administer an empiric antiepileptic.
Environment	<ul style="list-style-type: none"> ● Early and aggressive pain control and sedation to avoid ICP spikes with close monitoring of airway if patient is not intubated. ● Avoid hyperthermia and give paracetamol if needed. <ul style="list-style-type: none"> ○ Avoid use of passive cooling techniques such as wet sheets ● Avoid hypothermia, do not leave patient exposed for long periods of time.

^a Moderate resources: Usually available at a well-stocked district or small regional hospital.

^b Full resources: Usually available at a well-stocked national or larger regional hospital.

often occurs in the setting of traumatic injuries to the cervical spine and torso, decisions for neuroimaging are often made in conjunction with CT imaging for other injuries.

Applications for ultrasound in limited resource settings have been gaining attention lately, making ultrasound for TBI worth commentary. Sonographic measurement of the optic nerve sheath diameter showed initial promise in detecting elevated intracranial pressure (ICP) [41]. In the setting of trauma, elevated ICP is likely to suggest haematoma or significant oedema. However, Oberfoell et al. found significant inter-rater variability in optic nerve sheath diameter measurements, even among emergency physicians with fellowship training in ultrasound [42]. At this time, there are no formal recommendations for ultrasound as the sole imaging modality for diagnostic purposes in head trauma.

Laboratory testing

Laboratory testing of blood or cerebrospinal fluid for biomarkers in order to diagnose and risk stratify TBI patients is an area of intense research [43] but at this time is not standard practice and no active recommendations exist for the use of this diagnostic modality.

Acute management considerations

Severe TBI

The cornerstone of management of severe TBI is the avoidance of secondary injury. Primary causes of secondary brain injury include hypotension, hypoxia, hypercarbia, and worsening ICP. These secondary injuries can result from myriad causes. The majority of recommendations to avoid secondary injury do not require advanced resources. A regimented and organised approach, as with all trauma patients, is indicated for patients with severe TBI and summarized in Table 4.

Airway. Airway protection is of paramount importance in patients with severe TBI who, by definition, have a depressed level of consciousness. Patients with severe TBI should be intubated for airway protection and to ensure adequate oxygenation and ventilation [44]. Supraglottic airway devices should be considered for airway management only in settings where endotracheal intubation is not feasible and after the failure of basic airway manoeuvres such as a jaw thrust. It should be noted that supraglottic airway devices have been shown to raise ICP,

thereby reducing cerebral blood flow in animal models, potentially compromising cerebral perfusion pressure [45]. To prevent significant rises in ICP, the patient should be properly sedated and paralysed with standard dosing rapid sequence intubation agents. Though limited data suggests superiority of rocuronium over succinylcholine in severe TBI patients due to increased mortality with succinylcholine, the authors feel it is appropriate to allow local availability to guide therapeutic decisions [46]. A haemodynamically neutral induction agent such as ketamine or etomidate is preferred in order to prevent transient drops in cerebral perfusion pressure. There is debate as to whether ketamine is a safe option in patients with TBI, as its sympathomimetic properties are hypothesised to cause a surge in ICP. However, recent studies including a large retrospective review show no difference in mortality or other patient-centred outcomes when comparing etomidate and ketamine for TBI patients. Given its widespread availability in resource-limited settings, it should be considered a suitable option in performing rapid sequence intubation [47].

Cervical spine immobilization should be maintained, as 4–8% of brain-injured patients have been shown to have concomitant cervical spine injury [48]. It is important to note that this study was done in the United States, where seatbelts are commonly worn. In LMIC settings with decreased seat belt use [49] and increased motorcycle use a higher rate of cervical spine injury is very plausible [3].

Breathing. Hypoxia is a critical cause of secondary brain injury and can lead to cerebral oedema. If blood gas monitoring or intubation are not available, oxygenation monitored by pulse oximetry should be maintained greater than 90% with supplemental oxygen provided if necessary [44]. If there is access to serial blood gas measurements, PaO₂ should be maintained above 60 mm Hg [50]. Additionally, prolonged hyperoxia is associated with increased mortality and should be avoided [51,52]. Though no recommendations specify upper limits of safe oxygenation, a reasonable strategy is to provide the smallest amount of supplemental oxygen necessary to avoid hypoxia.

Ventilation and CO₂ management are equally important in patients with TBI, as hypercarbia can result in cerebral vasodilation with subsequent development of cerebral oedema, and therefore a decrease in cerebral perfusion. PaCO₂, measured by blood gas or by end-tidal CO₂ detector, is ideally kept between 35–40 mm Hg. If there is concern for

acute worsening of ICP or development of herniation syndrome, the patient can be briefly hyperventilated to decrease the PaCO₂ to achieve subsequent cerebral vasoconstriction with goal PaCO₂ 30–35 [44,50,53]. If no blood gas or end tidal CO₂ are available, relative hyperventilation can be a temporizing measure for approximately 15–30 min [50,53]. However, long-term cerebral vasoconstriction from hyperventilation will eventually lead to cerebral ischaemia, increased ICP, and worsened neurological outcomes [50,53].

Circulation. Management of normal haemodynamics in the severely brain-injured patient is also of paramount importance, as hypotension is a major cause of secondary brain injury. Cerebral perfusion pressure (CPP) is the key marker for adequate blood flow to the brain and is defined by the equation $CPP = \text{mean arterial pressure (MAP)} - \text{ICP}$ [44]. Maintenance of a normal MAP with goal of 80–90 mm Hg (or systolic blood pressure (SBP) > 110 mm Hg) is thus critical in maintaining adequate CPP [44,50]. Even transient hypotension can contribute to worsened outcomes, as a single episode of hypotension (SBP < 90 mm Hg) can increase mortality by 150% [54]. Continuous blood pressure monitoring with invasive blood pressure monitoring, such as an arterial line, should be utilised when available. Isotonic fluids and as needed vasopressors should be first-line in achieving haemodynamic goals, with blood products used as needed for trauma resuscitative purposes. Hypotonic or dextrose-containing fluids can worsen cerebral oedema and should be avoided [55]. Up to one-third of patients with severe TBI develop a coagulopathy, which can markedly worsen neurologic and trauma outcomes [56], making identification of occult injury even more important.

Disability. Post-traumatic seizures increase both cerebral metabolism and ICP, leading to poor outcomes [44]. Patients with seizure, or CT finding of oedema, intracranial haemorrhage or midline shift should be given a bolus dose of an available anticonvulsant medication such as phenytoin (15 mg/kg), followed by maintenance dosing [57]. An empiric antiepileptic using treatment dosing can be considered when GCS < 8 [57]. Hypoglycaemia is also associated with poor outcomes, so standard practice should include checking and correcting the glucose level as part of the primary survey for a patient with altered mental status.

The degree and duration of elevated ICP in the severe TBI patient has been shown to correlate closely with worse neurologic outcomes [58,59]. There are several non-invasive strategies that can aid in the management of ICP. All severely brain-injured patients should be positioned with the head of bed raised to 30° to facilitate venous return from the brain. Loosening the cervical collar and endotracheal tube ties, as much as safety will allow, can decrease external pressure on cervical venous outflow tracts [44].

Early and aggressive pain control and sedation should be prioritised to prevent ICP spikes. Specific medication choices will be driven by local availability and be given as needed bolus doses or continuous infusions. However, anxiolytics or pain medications that have less effect on haemodynamics (including fentanyl and long-acting benzodiazepines such as diazepam) may reduce iatrogenic harm from cerebral hypoperfusion [14,44]. Regardless of specific medication choice, continuous haemodynamic monitoring is essential for all sedated patients. Duration of action should also be considered as over-sedation can make serial neurological exams more difficult to follow. One study noted that bolus dosing of thiopental and propofol can effectively manage agitation, particularly during painful or uncomfortable procedures [44].

For the patients with severe brain injuries who have progressive neurological decompensation despite adequate implementation of the above measures, hyperosmotic therapy is indicated. Mannitol (0.5–1.5 g/kg/dose) and 3% hypertonic saline (150–250 mL/dose) both effectively pull excess fluid from brain parenchyma and reduce ICP [50,55]. Administration of mannitol results in an osmotic diuresis with resultant decrease in blood pressure and therefore CPP reduction. For patients in whom hypotension is a concern and hypertonic saline is unavailable,

sodium bicarbonate is a viable alternative and has been shown to effectively reduce ICP for up to 6 h [60]. These therapies should be given as temporizing measures in the setting of concern for acute brain herniation with a goal of stabilising the patient, obtaining neuroimaging, and admitting or transferring to an intensive care setting [50].

Definitive care of the severe TBI patient involves serial neurological monitoring in an intensive care setting. Invasive ICP monitoring and cerebrospinal fluid diversion can be utilised for patients who decompensate or whose conditions are refractory to conservative therapy [50]. The decision to pursue decompressive craniectomy [50] is nuanced and best determined by experienced neurosurgical consultants and beyond the scope of this review.

Environment. Hyperpyrexia drives cerebral metabolism and core temperature should be maintained below 37 °C with paracetamol or cooling devices. At the same time, hypothermia in TBI is not therapeutic and is potentially deleterious, especially for the coagulopathic polytrauma patient [50]. Unmonitored cooling with wet sheets and fans or exposed patients should be avoided.

Disposition. Because of the need for close haemodynamic monitoring, frequent neurological checks, and possible operative management, severe TBI patients should be transferred to the nearest available neurosurgical intensive care unit (ICU) as soon as the patient is stabilised, or when local resources have been exhausted. If a neurosurgical ICU is unavailable, then a facility with ICU capabilities is preferred. Additionally, the presence of a surgeon capable of performing emergent decompressive craniectomy is ideal in the case that the patient's condition deteriorates and requires neurosurgical intervention.

If transport is delayed or definitive care is unavailable, ongoing temporizing measures should be attempted, including repeated dosing of hyperosmolar agents and/or hyperventilation with a goal of transient ICP reduction. The avoidance of secondary brain injury is top priority while the patient remains in the emergency unit and during transport.

Moderate TBI

Unlike mild and severe traumatic brain injuries, the body of evidence for the management of patients with moderate traumatic brain injury is sparse. Overall clinical management should mirror recommendations for severe TBI. Often, discussions regarding the management of these patients is grouped with discussions of severe brain injuries [61]. Initial studies classified brain-injured patients with GCS 9–12 as moderately brain-injured, and this is still a commonly used definition [62]. On further review, the definition was changed to include brain-injured patients with a GCS 9–13 because patients with a GCS 13 were shown to have a similar instance of traumatic lesions on CT imaging of the brain as patients with a GCS 9–12, this definition will be utilised for this section [63].

Godoy et al. proposed the use of a tiered model that would prompt more aggressive management of patients with moderate TBI but poor expected clinical course [14]. This model applies well to the resource-limited setting as it emphasises aggressive early care for the more severely injured patients while potentially avoiding excessive diagnostics or transfers in the more stable portion of this cohort. “Potentially severe,” patients include: 1) patients with GCS 9–10 but negative neurological imaging, and 2) patients with GCS 11–13 but positive neurological imaging [14]. While these patients do not necessarily require aggressive airway management and are less likely to require neurosurgical intervention, they will require close neurological monitoring, blood pressure monitoring, and serial blood gases, and therefore be transferred to a facility with ICU capabilities.

For moderate TBI patients with GCS 11–13 and negative CT scan, hospitalization for serial neurological examination is required. While routine repeat CT imaging has not been found to be beneficial [64], repeat CT imaging is recommended at 12 h if the patient has not

returned to a GCS 14-15 because 32% of this cohort will show radiographic deterioration by this point [63]. ICU capabilities are not necessary for the care of this patient population unless the patient shows evidence of clinical deterioration.

Patients with moderate TBI are often alert enough to adequately protect their airway, so intubation is not mandatory unless otherwise clinically indicated. Upon recognition of the presence of moderate TBI, resuscitative efforts should be focused on avoidance of secondary brain injury risks, including hypotension and hypoxemia. As with severe TBI, management goals are SBP > 110, or MAP 80–90 mm Hg [44,50] and avoidance of intermittent hypotensive episodes, and pulse oximetry > 90%. Additional efforts should be made to avoid elevations in ICP including elevation of the head of bed to 30° and loosening of the cervical collar to allow for venous drainage.

The heterogeneity in clinical severity and eventual prognosis of patients with moderate TBI should prompt a varied approach to both treatment and disposition. At a minimum, all patients with moderate TBI warrant admission to a hospital for serial neurological monitoring. However, early recognition of high-risk patients with the greatest likelihood for decompensation is critical.

Mild TBI

Despite the high prevalence of mild TBI globally, there is little known about optimal treatment nor is there consensus regarding how to manage these patients [65]. Additionally, existing studies and resultant guidelines for management and treatment predominantly have been conducted in high-resource settings in North America and Europe, potentially limiting their application globally.

Emergency centre care for the mild TBI patient includes symptom control with anti-emetics and analgesics as needed. Management of mild TBI patients with positive neuroimaging should be discussed with neurosurgical consultants and include admission or observation for worsening symptoms and/or change in neurological exam.

Disposition. In general, the disposition of mild TBI patients presenting to the emergency centre depends on two factors: assessment of safety (risk of deterioration); and education regarding post-concussive symptoms and care. The Centres for Disease Control/American College of Emergency Physicians joint practice guideline on mild TBI supports the safe discharge of patients with mild TBI from the emergency department who have negative CT of the head. However, this recommendation excludes patients who are on anticoagulation therapy, patients with an underlying bleeding disorder, and patients who have had a previous neurosurgical procedure [66,67].

The decision to discharge a mild TBI patient from the ED must include education on symptoms and instructions on home care, particularly the expected course of post-concussive syndrome and return precautions. Post-concussive symptoms are generally divided into three categories [68,69]: somatic, cognitive and affective (Table 5).

Symptoms generally improve over time and are rarely still present 6 months post-injury. However, some patients go on to develop post-concussive syndrome, which is defined as concussive symptoms that persist beyond the expected 7 to 10 day recovery period from mild TBI [70,71]. Patients presenting with prolonged amnesia, dizziness, headache, anxiety, noise sensitivity, or trouble with verbal recall during the initial emergency centre evaluation may be at higher risk for developing post-concussive syndrome [72].

The CRASH trial, a 2009 study of 8927 TBI patients from 46 countries of varying resource levels, revealed that mild TBI patients

from LMICs were half as likely to experience disability from both mild and moderate TBI than those from HICs. The reasons for this are not clear. However, it may be due to differences in the definition of disability between countries, as well as socioeconomic and environmental differences in how societies define and react to disability [6].

Neither hospitalization nor referral to specialty care and multi-disciplinary treatment reduces the duration or severity of symptoms. However, there is evidence to suggest that education regarding symptoms, i.e. “what to expect” may reduce long-term post-concussive complaints [73,74]. Outcomes were also improved for patients who were provided recovery expectations and symptom management strategies including a plan to gradually resume normal activities [75]. Signs and symptoms that suggest complication following mild TBI should be clearly written in the patient’s native language and also verbally communicated to both the patient and their support persons.

Information about post-concussion symptoms and care should be provided in a similarly straightforward fashion. Symptom management should include rest, as well as limited physical and cognitive exertion [76]. In general, mild TBI patients should be advised to avoid returning to exercise or strenuous work until concussion symptoms have resolved. Return to exercise and strenuous work should be gradual, and stepwise with regards to physical demand and risk for contact or reinjury. Progression should be halted and the process started over if symptoms recur [77].

Prehospital and interfacility transport care

Half of the patients who die from TBI do so within the first 2 h after injury, making prehospital assessment and interventions critical [78]. The building blocks of good prehospital care of TBI patients are not resource-intensive and can be implemented by providers with basic levels of training. Prehospital providers must be trained to recognise when a head injury is significant or not, and to make transport destination decisions accordingly. The principle aims of prehospital management of TBI patients are to objectively monitor the patient for decompensation and to prevent further brain injury by preventing hypoxia, hypoglycaemia, and hypotension [79]. Secondly, providers should repeatedly assess adequacy of airway, breathing, and circulation, as well as disability and environmental exposure status.

Training prehospital providers in use of the GCS and pupillary assessment while providing prompts to record reassessments of these parameters over time can identify patients who are decompensating versus those who are stable. In turn, this can aid in triage decisions and direct higher-level care for patients once they are hospitalised [80]. Continuous measurement of oxygen saturation and intervention to prevent hypoxia (i.e. SaO₂ < 90%) is recommended. If a patient with a severe TBI requires assisted ventilation, hyperventilation should be avoided. Proper short-term hyperventilation for management of cerebral herniation requires a higher level of care than is available in resource-limited prehospital settings [81]. Frequent measurement of blood pressure and treatment of hypotension with isotonic fluids is recommended to prevent further brain damage from poor cerebral perfusion.

Limitations

This resource tiered review employed classic PRISMA methodology to extract the identified articles. However, there were a small number of articles that directly addressed the care of those with TBI in LMICs.

Table 5
Post concussive symptoms.

Somatic	Headache, dizziness, nausea, photophobia, phonophobia, tinnitus, blurred vision, light headedness, anosmia, fatigue
Cognitive	Difficulty with memory and concentration, word finding
Affective	Mood lability, irritability, sleep disturbances, anxiety, depression, personality changes

Searching additional databases may have improved retrieval of identified research. In addition, publication bias and selective reporting within studies could have suggested heightened results from higher resourced interventions. Wherever possible, extrapolation of proven interventions from high income countries that are translatable to resource limited environments were emphasized.

This review covered a broad clinical topic with a large number of clinical management recommendations. It was not feasible to provide strength of the recommendation information in the body of the text for each management recommendation. The authors evaluation of the strength of the evidence can be found in [Appendix C](#). It should be noted that for TBI management there is significant heterogeneity in the supporting evidence and a paucity of randomized control trial data to support recommendations.

Conclusion

TBI is a major global public health problem, with a disproportionately high incidence and mortality in LMICs. A high index of suspicion for TBI and an understanding of the risk factors, signs, and symptoms that are most likely to require surgical management or lead to long-term neurocognitive sequelae are the foundation of TBI care in the emergency unit. The care of these patients begins with rapid and appropriate triage, prompt recognition of TBI, and immediate stabilisation of the severely brain-injured or otherwise critically ill trauma patient. For the mildly brain-injured patient, clinical decision tools exist to aid in determining which patients require neuroimaging and which can be simply observed or discharged. Appropriate instructions regarding expectant management of long-term symptoms is key at time of patient discharge.

Patients with moderate and severe brain injury are best served by a regimented approach. The approach begins with recognition of high risk mechanisms of injury and signs and symptoms concerning for a significant head injury. This is followed by a systematic assessment and management of life threats with aggressive measures to control the airway, respiratory physiology, and haemodynamics- all with the primary goal of maintaining adequate cerebral perfusion. A thorough head-to-toe inspection of the patient for significant injuries is completed after life threats are managed. Moderate and severe brain-injured patients typically require neuroimaging and possible transfer to tertiary centres for closer neurological monitoring.

An evidenced-based and standardised approach for the care of these patients would not only be beneficial on a case-by-case basis but would also likely drive better outcomes at a systems level. Further investigation regarding resources and limitations for diagnostics and treatment of TBI patients in an Africa-specific context are needed.

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Authors' contributions

Authors contributed as follow to the conception or design of the work; the acquisition, analysis, or interpretation of data for the work; and drafting the work or revising it critically for important intellectual content: JD contributed 20%; GC 14%, JW, DR and TWB contributed 12% each; and NL, NM and EJC contributed 10% each. All authors approved the version to be published and agreed to be accountable for all aspects of the work.

Declaration of competing interest

The authors have no conflict of interest to declare.

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