Approaches to Pediatric Traumatic Brain Injury

Diagnosis and Management

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KEYWORDS

• Pediatric traumatic brain injury • Glasgow coma scale • Intracranial pressure
• Moderate traumatic brain injury • Severe traumatic brain injury

KEY POINTS

- Moderate/severe pediatric TBI requires a structured, multidisciplinary approach that includes immediate stabilization, intracranial pressure management, and precise fluid/electrolyte monitoring to reduce secondary injuries.
- Imaging, primarily through CT scans, is vital for assessing moderate to severe TBI, while biomarkers show potential for future outcome prediction but are not yet clinically standardized.
- Effective sedation, seizure prevention, and temperature control are essential to managing TBI in pediatric patients, with careful drug selection to minimize side effects.
- Rehabilitation and long-term support are critical to optimize functional recovery and provide children with the best quality of life postinjury.

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of morbidity and mortality in children. In the United States, approximately 500,000 children under the age of 15 experience TBI each year, the majority of which are mild TBI or concussion with no long-term sequelae.[1] Males account for about 60% to 70% of cases across all age groups, with falls being the leading cause of TBI under age 9 and motor vehicle accidents, sports-related injuries, and assaults predominately affecting children greater than 10 years of age.[1] It is estimated that 145,000 children in the US live with long-lasting disabilities

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Abbreviations	
ADH	antidiuretic hormone
AED	antiepileptic
CPP	cerebral perfusion pressure
CSF	cerebrospinal fluid
CSW	cerebral salt wasting
CT	computed tomography
CUS	cranial ultrasound
EMS	emergency medical services
EPIC4Kids	Excellence in Prehospital Injury Care for Children
EVD	extraventricular drains
fMRI	fast acquisition MRI
GCS	Glasgow coma scale
ICP	intracranial pressure
IPM	intraparenchymal monitors
MAP	mean arterial pressure
PECARN	Pediatric Emergency Care Applied Research Network
SIADH	secretion of antidiuretic hormone
SpO2	oxygen saturation
TBI	traumatic brain injury

from TBI.[2] This article will focus on the moderate to severe TBI population with a brief discussion of the evaluation of minor head injuries.

MEDICAL EVALUATION

Physical examination focuses on assessing the patient's ability to protect their airway, predicted by their level of consciousness, along with evaluating for signs of increased intracranial pressure (ICP) and identifying any additional injuries to guide appropriate treatment. To determine the level of consciousness, the Glasgow Coma Scale (GCS) is used to assess eye, verbal, and motor responses.[3] A GCS score below 8 typically indicates severe TBI, 9 to 12 suggests moderate TBI, and 13 to 15 indicates mild TBI.[3] Monitoring changes in GCS over time provides valuable insights for clinicians.

Evaluating for elevated ICP includes assessing for Cushing's triad, which consists of 3 key signs: hypertension, bradycardia, and irregular respirations. These signs result from increased ICP compressing the brainstem, leading to a compensatory response to maintain cerebral perfusion.[4] Other indicators of elevated ICP include pupil reactivity changes, papilledema, bulging fontanelle (in infants), and neurological signs such as cranial nerve palsies, abnormal posturing, or flaccidity of extremities.[4] Additionally, identifying other injuries and maintaining cervical spine precautions until clearance is essential for optimal treatment within a comprehensive trauma evaluation.[5]

CLINICAL DECISION RULES

The Pediatric Emergency Care Applied Research Network (PECARN) head trauma study was a large, prospective multi-center cohort study focused on minimizing unnecessary head computed tomography (CT) scans in children with minor head injuries.[6] Gambacorta and others performed a retrospective study testing the validity of the PECARN rules and when applied to over 3500 patient presentations, the rules would have reduced CT scans by around 30%. This study also demonstrated a sensitivity of about 98% in patients less than 2 year old and 97% in patients greater than 2 years old for the PECARN rules correctly identifying patients who should undergo head CT to identify significant TBI. There was a high rate of false positives with a

specificity of under 50%. The variables that were most consistent in correlating with CT scan abnormalities were repeated episodes of vomiting, severe mechanisms, and trauma to parieto-occipital areas.[7] PECARN study does not account for underlying disorders such as coagulopathies that may predispose children to a more significant injury.[8] Overall, the PECARN head trauma study was successful in reducing unnecessary imaging and interventions but did allow for a large amount of subjectivity to correlate with clinical judgment.[9] This article focuses on patients with moderate to severe TBI, who would typically fall out of the PECARN criteria and require imaging in the emergency department (ED) to facilitate management and diagnosis.

IMAGING TECHNIQUES

CT and MRI are essential imaging tools in the evaluation of TBIs. CT is widely regarded as the standard first-line imaging modality due to its speed, availability, and high effectiveness in detecting critical injuries such as skull fractures, hemorrhages, and mass effects. Despite these benefits, CT exposes patients to radiation, which has been associated with an increased risk of cancer. It is estimated that radiation exposure from healthcare-related imaging could lead to approximately 170 additional fatal cancer cases each year among children under 15.[10] Still, in the context of severe TBI, the risk-benefit ratio favors imaging in the ED. It should not be deferred out of concern for radiation exposure in this high-risk patient population. While MRI is not typically used for the initial assessment due to limited availability, longer scan times, and the potential need for sedation in younger children, it plays an important role in further evaluation, especially in cases where CT results are inconclusive or when nonhemorrhagic injuries are suspected.[10]

A comparative study demonstrated that fast acquisition MRI (fMRI) is comparable to CT in identifying extra-axial hemorrhage, with enhanced sensitivity for detecting intraparenchymal and diffuse axonal injuries with a slightly reduced ability to detect skull fractures. This finding suggests that fMRI could be a safer alternative to CT for minimizing radiation exposure in pediatric patients.[11] Fast-acquisition or rapid MRI is gaining interest as a potential acute assessment tool due to its shorter scan times than conventional MRI. However, CT remains the preferred modality for the initial evaluation of moderate to severe TBI cases due to availability as well as the frequent need for additional imaging of traumatic injuries that require CT use.[12,13] Ongoing studies aim to evaluate the predictive value of early MRI in determining outcomes in children with moderate to severe TBI. Ferrazzano and colleagues, in a study across 27 US centers, found that about 60% of centers performed MRI within the first week following injury, often including sequences to assess for diffuse axonal injury and ischemia, though less frequently using perfusion or spectroscopy imaging.[14]

For children under 1 year of age, cranial ultrasound (CUS) offers a noninvasive imaging option through the anterior fontanelle, allowing for the evaluation of intracranial hemorrhages, ventricular size, and brain parenchyma changes. Peter and colleagues found that CUS had a sensitivity of 93% and a specificity of 98% in detecting intracranial hemorrhage and skull fractures. Missed diagnoses were limited to skull fractures without clinical decompensation. CUS provides advantages such as radiation-free imaging, bedside availability, and utility for serial monitoring, though it typically requires review by a trained radiologist. As a result, CUS is generally used alongside more definitive modalities like CT or MRI but not as the initial imaging in the ED setting.[15]

CLINICAL BIOMARKERS

Given the radiation exposure with CT imaging and the need for better prognostic tests, there is interest in developing usable biomarkers for TBI. Research into clinical

biomarkers shows significant promise in enhancing early prognostication and has potential to guide treatment approaches for pediatric TBI. Recent studies have highlighted various markers of inflammation, damage, degeneration, and changes in metabolism with potential clinical applications, although none are currently routine clinical use-[16] Plasma biomarkers, including GFAP and UCH-L1, have been associated with poorer outcomes, while S100B, despite being less specific in cases involving extracranial injuries, remains correlated with injury severity.[17] Another study that examined a biomarker panel of GFAP, UCH-L1, S100B, and NSE, demonstrated high sensitivity and specificity for identifying TBI in children within the critical early hours following injury.[18] Specific microRNAs were notably elevated or decreased in cases of severe TBI, correlating with injury severity and poorer outcomes, specifically attention difficulty.[19] Additionally, elevated tau protein levels in cerebrospinal fluid (CSF) and serum have been associated with diffuse axonal injuries and poor functional outcomes.[20] Although clinical biomarkers have not yet been widely adopted in pediatric protocols, many have potential importance in advancing tailored approaches for managing pediatric TBI.[21] However, these are only investigational and not standard to obtain in the ED setting.

INJURY CLASSIFICATIONS

Moderate and severe pediatric TBI is a clinical diagnosis. It can be associated with a variety of imaging findings, including hemorrhage, diffuse axonal injury, acute-on-chronic injuries, and penetrating trauma. TBI usually involves both a primary injury, which is the direct impact on brain tissue, and a secondary injury that emerges as a consequence of neural tissue remodeling and is responsible for many long-term effects.[1] The primary focus of clinical management discussed in the following section is on preventing secondary injuries and facilitating brain recovery from injury.

Hemorrhages associated with pediatric TBI can be further categorized into subarachnoid, epidural, subdural, and intraparenchymal types. In cases of nonaccidental trauma, there may be acute-on-chronic injuries, often located in the subdural region. Subarachnoid hemorrhage involves bleeding between the brain and its protective meninges and is commonly associated with symptoms like severe headache, neck stiffness, photophobia, and hydrocephalus.[22] Epidural hemorrhage, involving bleeding between the skull and the dura mater, is frequently associated with skull fractures and may require rapid surgical intervention due to rapid expansion of the hematoma.[22] Subdural hemorrhage, characterized by bleeding between the dura mater and the brain, results from ruptured bridging veins and typically presents with more gradual symptom onset. Lastly, intraparenchymal hemorrhage refers to bleeding within the brain tissue itself, which may result from penetrating or blunt trauma, arteriovenous malformations, or coagulopathies. The severity and symptoms of intraparenchymal hemorrhage vary depending on the hemorrhage's size and location within the brain[22] (**Figs. 1–3**).

PREHOSPITAL MANAGEMENT

Prehospital management strategies for pediatric TBI focus on preventing secondary injury and ensuring rapid transport to specialized trauma centers with pediatric expertise. The EPIC4Kids (Excellence in Prehospital Injury Care for Children) study demonstrated that implementing a standardized prehospital algorithm significantly improved survival rates across all TBI severity levels. By emphasizing evidence-based interventions to optimize oxygenation and ventilation, and preventing hypotension, the study reduced variability in emergency medical services (EMS) care and improved both

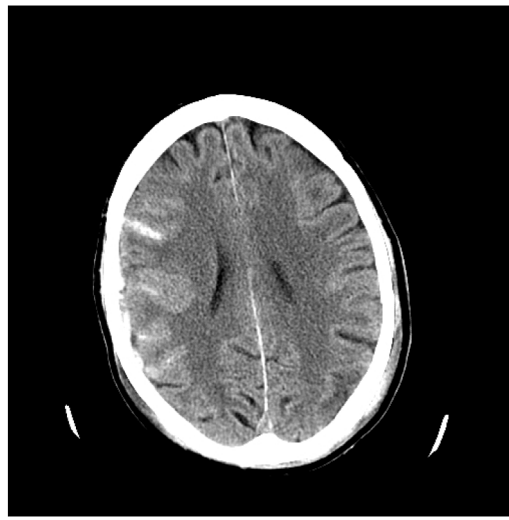

Fig. 1. Subarachnoid hemorrhage.[21] (*Image courtesy of* Dr. Robert Vezzetti.)

short-term survival and neurological outcomes.[23] Key strategies include maintaining oxygen saturation (SpO2) above 94% and normocapnia ($Paco_2$ 35–45 mm Hg) while avoiding the risks of hypoxia or prolonged hyperoxia. Aggressive fluid resuscitation with isotonic crystalloids, such as normal saline or lactated Ringer's solution, is recommended in patients with volume deficits to prevent hypotension and maintain mean arterial pressure (MAP), critical for adequate cerebral perfusion. Administration

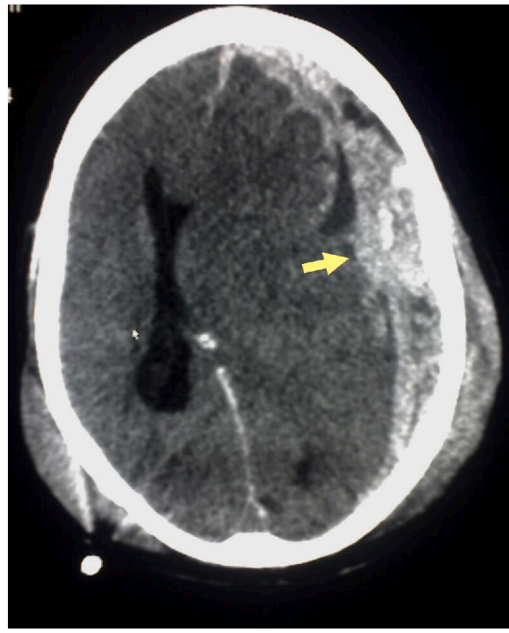

Fig. 2. Subdural hematoma (*arrow*) with midline shift.[21] (Trauma subdural arrow by Glitzy queen00 - Wikimedia Commons. (n.d.). https://commons.wikimedia.org/wiki/File:Trauma_subdural_arrow.jpg.)

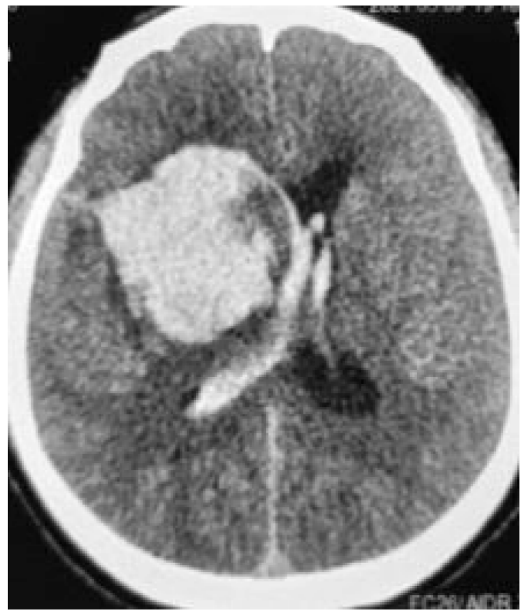

Fig. 3. Intraparenchymal hemorrhage.[21] (Contributed by Dr. Sunil Munakomi, MD.)

of 20 mL/kg boluses is typical practice while also cognizant of any factors that may provoke fluid overload leading to cerebral edema. Ensuring early, precise fluid resuscitation is crucial for stabilizing hemodynamics and minimizing secondary brain injury.

Proper patient positioning can also help reduce ICP and optimize intracranial venous drainage. Recommendations include maintaining a midline head position, ensuring cervical collars or tracheal ties are not overly tight, and elevating the head of the bed to 30 to 45°, provided no contraindications exist.[24,25] Together, these measures form the foundation of effective prehospital TBI care, as highlighted by the EPIC4Kids study and other guidelines to improve outcomes for pediatric patients.

ANALGESIA

Effective analgesia requires balancing benefits and risks, often achieved through a multimodal strategy. Acetaminophen is a common first-line option due to its tolerability, nonsedating profile, and lack of impact on platelet function, though its analgesic potency is limited, particularly in acute pain. Nonsteroidal antiinflammatory drugs (NSAIDs), such as ibuprofen, offer similar analgesic and antiinflammatory effects, but come with higher risks, including gastrointestinal irritation, bleeding, and renal impairment. These side effects stem from their cyclooxygenase enzyme inhibition, which reduces platelet function and increases bleeding risk.[26]

For more severe pain, opioids and benzodiazepines are effective, but present challenges, including sedation that interferes with neurological assessment and respiratory depression that can raise ICP due to hypercapnia. Long-term use also risks tolerance, dependence, and withdrawal.[27] In cases such as severe pediatric TBI, the most effective pain management combines acetaminophen with low-dose

opioids, enhancing analgesia while minimizing the side effects associated with individual medications.[26]

INTUBATION AND SEDATION

When intubating a child with TBI, careful management is critical to prevent secondary injury and control ICP. Rapid sequence intubation with preoxygenation is generally recommended to minimize aspiration and hypoxia.[28] The selection of sedatives varies significantly, with minimal evidence to support the preferential use of specific agents but most often considerations made based on individual patient injury patterns, associated injuries and comorbidities regarding the below agents.[29–32] Most of these agents may also be considered to reduce metabolic demand and ICP but can hinder neurological assessments due to their prolonged effects.[30] More recent studies show that ketamine does not increase ICP as once previously thought.[33] These strategies highlight the delicate balance required for effective sedation and neuromuscular management while minimizing risks in pediatric TBI cases (**Table 1**).

BLOOD PRESSURE MANAGEMENT

Careful regulation of blood pressure is crucial due to the potential impact on cerebral perfusion and secondary brain injury. Cerebral perfusion pressure (CPP) is the difference between MAP and ICP. Normal values for adults are between 60 to 70 mm Hg, and for children, 40 to 60 mm Hg. In severe traumatic brain injuries, in order to maintain higher CPP, MAP goals tend to be on the higher side, around 80 to 100 mm Hg, while avoiding excess pressures and the risk of cerebral edema. The use of arterial lines to measure MAP and extraventricular drains (EVD) or intraparenchymal bolts (ICP bolts) can be used in management. However, this is often challenging in the ED and is typically in conjunction with neurosurgical and critical care specialists.[34]

Blood pressure management in severe TBI involves the use of appropriate fluid resuscitation, blood transfusions, and vasopressors. Fluid management aims to maintain euvolemia, with specific choices tailored to sodium management, which will be further discussed in the context of overall treatment strategies. Blood transfusion targets in TBI management typically range between 7 to 9 g/dL, though these goals may be adjusted based on patient comorbidities or ongoing hemorrhage. Coagulopathy correction may involve platelets, fresh frozen plasma, or vitamin K, especially in cases where intracranial hemorrhage could exacerbate intracranial hypertension.[34]

While no clear evidence supports the superiority of any specific vasopressor for increasing blood pressure in severe TBI, clinical choices are often dictated by the patient's cardiac function and risk for tachydysrhythmias. Norepinephrine, epinephrine, or phenylephrine are commonly used as first-line agents, with phenylephrine potentially offering a mortality benefit, as suggested by a retrospective study of 24,718 patients.[35] Dopamine is less commonly utilized due to its renal effects at certain doses and the potential for complicating management, particularly when combined with mannitol or hypertonic saline. Overall, clinicians are advised to use the most familiar agent in their setting to achieve MAP goals critical for maintaining cerebral perfusion.

HYPERTONIC SALINE AND MANNITOL

Hypertonic saline and mannitol are 2 medications that are osmotic agents and, in theory, reduce ICP, thus improving cerebral blood flow and pressure. In the past 10 years, there has been an ongoing shift to the preferential use of hypertonic saline over mannitol to decrease ICP in severe traumatic brain injuries, as studies have shown

Table 1
Common sedation and neuromuscular blockade considerations[27,28]

Agent	Class	Benefits	Contraindications
Propofol	General anesthetic	• Easily titratable • Antiepileptic properties	• Significant hypotension • Long-term use is limited due to the risk of propofol infusion syndrome
Ketamine	NMDA antagonist	• Analgesia • Antiepileptic properties • No direct effect on CPP as previously published[30]	• Significant hypertension
Dexmedetomidine	Alpha-2 agonist	• Minimal effect on blood pressure	• Severe bradycardia • Needs adjunctive agent for adequate sedation
Midazolam	GABA agonist	• Antiepileptic	• Respiratory and cardiac depression • Tissue accumulation can delay accurate neurologic examination • Less effective than propofol for intracranial hypertension
Fentanyl	Opioid	• Analgesia • Less hemodynamic effects • Rapid onset	• Respiratory depression • May need an adjunctive agent
Barbiturates	GABA agonist	• Strong antiepileptic effect • Long-acting	• Respiratory and cardiac depression • Inaccurate neurologic examination for hours to days after stopped
Succinylcholine	Depolarizing NMBA	• Rapid paralytic effect for intubation	• Hyperkalemia • Muscular dystrophies • Crush injuries • Burns
Vecuronium Rocuronium	Nondepolarizing NMBA	• Paralysis for sedation and ventilation	• Neuromuscular disease • Hypotension • Longer-acting so can interfere with accurate neurologic examination

From Payen JF, Schilte C, Behouche A. Sedation, pain, and delirium in patients with traumatic brain injury. In: Brogi E, Coccolini F, Ley EJ, editors. Traumatic brain injury. Hot topics in acute care surgery and trauma. Springer; 2024. https://doi.org/10.1007/978-3-031-50117-3_14; and Liu SY, Kelly-Hedrick M, Temkin N, et al. Association of early dexmedetomidine utilization with clinical and functional outcomes following moderate-severe traumatic brain injury: a transforming clinical research and knowledge in traumatic brain injury study. Crit Care Med 2024;52(4):607–17. https://doi.org/10.1097/CCM.0000000000006106.

greater effectiveness in the rate and duration of ICP reduction with a reduced ratio of side effects. A recent meta-analysis recommended an optimal concentration of 3% for hypertonic saline administered as a bolus dose between 1.4 to 2.5 mL/kg. However, this study included mostly adult patients. In a survey of pediatric emergency department usage of hypertonic saline, the most common dosing was 3 to 5 mL/kg at an infusion rate of less than 15 minutes and encountered few side effects.[36] No initial maximum dose was ascertained, but most manufactured solutions are packaged in 500 mL aliquots, thus serving as a reasonable and cost-effective approach. It is important to note hypertonic saline can also be delivered with stronger concentrations, including 5%, 7.45%, 7.5%, 10%, 15%, and 23.4%, but there poses an increased risk of administration risks, including thrombophlebitis and extravasation related injury.[37]

Hypertonic saline side effects are seldom reported and include hypernatremia, pulmonary edema, coagulopathy, and hyperchloremic metabolic acidosis. Though very few cases are reported, acknowledgment of the risk of central pontine demyelination with rapid hyponatremia correction should always be considered, with a decreased risk associated with serum sodium correction of less than 6 to 8 mEq/day.[36] Reported side effects of mannitol use include acute renal failure, pulmonary edema, and hypotension secondary to its diuretic effect. For both therapies, especially those receiving multiple doses, patients should be closely monitored for severe electrolyte and volume derangements.[38] Hypertonic saline administration has less diuretic effect than mannitol due to increased serum sodium, leading to increased antidiuretic hormone (ADH) release.[37] In theory, hypertonic saline and mannitol use should be avoided in cases of epidural hematomas, as the decrease in ICP may lead to expansion of the hematoma.

SODIUM MANAGEMENT

Careful monitoring and adjustment of serum sodium levels are essential to TBI management in the critical care setting. While not always feasible in the ED during initial management, understanding these principles is key to facilitating further management. Increased sodium levels lead to increased serum osmolarity and higher levels create a gradient in water movement. This increases water movement from the brain parenchyma, reducing cerebral edema and ICP.[35] Electrolyte imbalances, especially hyponatremia, can be frequently seen in TBI. Two mechanisms, cerebral salt wasting (CSW) and the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), are proposed to explain hyponatremia in TBI.[38] CSW involves hyponatremia with elevated urine sodium and hypovolemia, likely due to increased brain natriuretic peptide or hypothalamic damage disrupting the sympathetic system.[38] In contrast, SIADH results from pituitary damage causing inappropriate ADH release, leading to a euvolemic or hypervolemic hyponatremic state.[39] The initial choice of using normal saline as a resuscitative fluid is typically to be preferred due to the effect of sodium on osmotic gradients. Still, it is prudent to have a balanced approach in severely hypovolemic patients requiring large infusions.[38] General target serum sodium levels range from 145 to 155 mEq/L.[40]

SEIZURE PROPHYLAXIS

The risk of seizures is reported to be between 2% to 16%, therefore the use of antiepileptics (AEDs) is supported to prevent early posttraumatic seizures in severe TBI patients.[41,42] In the inpatient setting, especially for those with severe TBI or pharmacologically sedated, it is important to consider subclinical status epilepticus, as these patients are at higher risk, up to 30%.[41–43] There has been a shift to

levetiracetam from prior commonly used AEDs due to its favorable effectiveness and low-side effect profile. Evidence suggests prophylactic AED use is beneficial in the first 7 days after injury, but long-term indications are generally not recommended in the absence of seizures due to a lack of significant benefits.[44,45] Higher dosing levels, on average of 25mg/kg/day, were shown to significantly lead to optimal serum drug levels and reduce the risk of seizures by 68% compared to lower dose regimens.[45] Some studies report concern that prolonged use of AEDs may inhibit functional recovery in some patients, though other studies describe a worsened outcome in patients who have posttraumatic seizures.[44]

TEMPERATURE MANAGEMENT

Early posttraumatic fever can occur in 40% to 70% of TBI patients. The pathophysiology is likely attributed to a disrupted hypothalamic set point. Adult critical care studies emphasize the importance of preventing fever, as fever prevention may lessen cerebral damage.[46] Similar to adult studies, fevers in children with TBIs are associated with worse outcomes.[47] Targeted temperature management to prevent fevers is the favored approach, as therapeutic hypothermia has not been demonstrated to improve neurological outcomes in several multicenter randomized controlled studies.[48–51] Although many treatment methods have been suggested to control posttraumatic fever, none have been identified as superior. Routine pharmacological methods include acetaminophen, aspirin, and nonsteroidal antiinflammatory medications.[51] There is some evidence supporting the use of propranolol to control the autonomic dysfunction sometimes seen in severe TBI.[52] External cooling methods can involve using rotary fans and surface cooling devices.[51] Ongoing research continues in the field of temperature management after TBI, but at this time, the best evidence suggests normothermia should be maintained.

NEUROSURGICAL MANAGEMENT

Sudden severe headaches, vomiting, and changed levels of consciousness can be the first indicator of increased ICP. However, when a patient's examination is limited due to the severity of their injury or secondary to pharmacological interventions, advanced ICP monitoring is often indicated in TBI.[53] There are both noninvasive and invasive methods for measuring ICP. Noninvasive measurements, including pupillometry, optic nerve sheath diameter, transcranial doppler, and tympanic membrane displacement, are advantageous due to the lack of risk of intracranial infection; however, they lack significant accuracy and, therefore, are not reliable in the clinical setting. Invasive monitors are the gold standard for ICP measurement.[54] Intraparenchymal monitors (IPM) are placed intraparenchymal or in the subarachnoid, subdural, or epidural spaces and are sometimes referred to as a *bolt*. This allows for the continuous transduction of ICP waveforms; however, although it has a reduced risk of infection it cannot drain CSF to decrease ICP. This contrasts with an EVD, which is larger and can be used for diagnostic ICP measurements and therapeutic CSF drainage. The choice between a decompressive craniotomy/craniectomy and EVD depends on the severity of the injury, the injury pattern, and the need for rapid ICP decrease. A decompressive craniectomy involves the removal of a section of the skull and opening the underlying dura mater to relieve ICP, and this can be done unilaterally or bifrontally, depending on the pathology location. The decision for a craniotomy, craniectomy, EVD, and IPM depends on a patient's clinical status, need for therapeutic drainage, and proceduralist preferences.[26,53]

Table 2
Key interventions across various categories for the management of pediatric traumatic brain injury

Category	Key Interventions
Prehospital Care	• Maintain *SpO_2>94%* to ensure adequate oxygenation • Prevent hypotension through fluid resuscitation with *isotonic crystalloids* (eg, normal saline, Lactated Ringer's, Plasma-Lyte) • Ensure rapid transport to specialized trauma centers with pediatric expertise • Position the patient's head *midline* and elevate the head of the bed *30°–45°* to optimize venous drainage and reduce ICP (if no contraindications)
Initial Stabilization	• *Airway Management*: Secure airway promptly to prevent hypoxia • *Blood Pressure*: Maintain CPP. Avoid hypotension to reduce secondary brain injury
Intracranial Pressure (ICP) Management	• *ICP Monitoring*: Watch for elevated ICP signs (eg, Cushing's triad) • *Hypertonic Saline (3%)*: Preferred for ICP reduction due to longer-lasting effects and fewer side effects compared to mannitol • *Mannitol*: Use as a second-line option when hypertonic saline is unavailable • Elevate head to *30°–45°* if possible to optimize venous drainage
Imaging	• *CT scans*: First-line for moderate to severe TBI • *MRI*: For detailed imaging, especially for diffuse axonal injuries • *Cranial ultrasound*: For infants (<1 y)
Fluid and Electrolyte Management	• Use *isotonic crystalloids* (eg, *normal saline, Lactated Ringer's, or Plasma-Lyte*) for resuscitation • *Volume goal*: Administer boluses of *20 mL/kg* to achieve euvolemia and maintain adequate MAP • Monitor serum sodium levels (target: *145–155 mEq/L*) • Avoid hypotension to prevent secondary brain injury
Sedation and Analgesia	• *Sedation*: Use *ketamine*, *propofol*, or *dexmedetomidine* based on ICP and hemodynamics • *Analgesia*: Use a multimodal approach with *acetaminophen, NSAIDs*, and *low-dose opioids*
Seizure Prevention	• Administer *levetiracetam* for first 7 d postinjury to prevent early posttraumatic seizures • Use higher dosing (eg, *25 mg/kg/day*) for optimal effectiveness • Prophylactic AED use beyond 7 d is *not recommended* unless seizures are present • Consider *continuous EEG monitoring* for detecting subclinical seizures in severe cases

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Table 2
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Category	Key Interventions
Temperature Control	• Maintain *normothermia* • Use *external cooling methods* (eg, rotary fans, surface cooling devices) • *Internal cooling methods*: Consider for severe dysregulation (eg, intravascular cooling catheters)
Surgical Interventions	• *ICP monitoring*: Use *EVD* or *IPM when appropriate* • *Decompressive craniectomy*: For severe ICP elevation or hemorrhage.
Rehabilitation	• Initiate rehabilitation within *3 d post-injury.* • Include *physical, cognitive*, and *psychosocial therapies* to support recovery.

REHABILITATION AND PROSPECTIVE THERAPIES

About 50% to 60% of children who survive severe TBIs experience lifelong disabilities affecting cognitive, physical, behavioral, and emotional functioning, quality of life, or learning ability. The Pediatric Glasgow Outcome Scale is a tool that has been modified to better reflect the developmental stages of children and is used to assess the long-term outcomes of children who have suffered TBI. Children are scored at different time points post-TBI, typically postdischarge and at subsequent follow-up visits to categorize the level of disability to help tailor rehab efforts to maximize the quality of life and functional recovery.[55]

Continued research is ongoing for the treatment of traumatic brain injury after the immediate acute phase. There is no structured, comprehensive rehabilitation program standard for the treatment of pediatric TBI, though evidence does suggest that delay in therapy has been correlated with worsened functional outcomes. Evidence has shown that initiating rehabilitation within 3 days of injury is not harmful.[55] Medically, there have been attempts to trial innovative therapies to combat TBI symptoms. A meta-analysis of hyperbaric oxygen therapy was inconclusive regarding the effects of increased oxygen levels on the treatment and rehabilitation of TBI, with the initial hypothesis that increased oxygen levels would reduce intracranial swelling and pressure.[56] A 2005 multicenter placebo randomized control study with 10,008 TBI patients showed an increased risk of death within 2 weeks and concluded that steroids should not be routinely administered.[57] Additionally, some clinical trials are investigating the neuroprotective effects of erythropoietin, statins, and stem cells but have failed to show any consistent improvement in outcomes.[58] Overall, it is recommended for institutions to implement protocols encouraging early rehabilitation and transition to the outpatient phase when patients can do so clinically.

SUMMARY

Managing moderate to severe TBI in children requires a multidisciplinary approach to address the complex and diverse needs of these patients. Early and intensive stabilization in the acute phase, including prompt airway management, ICP monitoring and intervention, and meticulous fluid and electrolyte balance, is crucial to preventing secondary brain injury. After the acute phase, care shifts toward a combination of

cognitive and physical rehabilitation along with tailored pharmacologic treatments to control pain, seizures, and other complications. Rehabilitation is individualized—incorporating physical, occupational, and behavioral therapies to support the child's recovery and reintegration into their community, striving for the best possible quality of life. Ultimately, the primary goal is to optimize neurological recovery, enhance functional independence, and provide the child and family with the resources to navigate the lifelong challenges of such a significant injury (**Table 2**).

CLINICS CARE POINTS

- Early airway and blood pressure management: Secure the airway promptly and maintain cerebral perfusion pressure, avoiding hypotension to reduce the risk of secondary brain injury.
- Use of GCS: Regularly assess the GCS score to monitor neurological status; a score below 8 often indicates severe TBI and the need for aggressive intervention.
- CT imaging criteria and surgical consideration: Utilize CT for moderate to severe TBI assessment, with clinical decision rules like PECARN to limit unnecessary imaging in minor cases. CT findings can guide surgical decisions, as decompressive craniectomy or other surgical interventions may be necessary for hemorrhage or elevated ICP.
- Controlled fluid resuscitation and sedation management: Use isotonic crystalloids for volume support, aiming for euvolemia to avoid worsening cerebral edema. When sedation is necessary, choose agents like ketamine or dexmedetomidine that allow for reliable neurological monitoring without increasing ICP.
- Temperature and seizure prophylaxis: Maintain normothermia and consider antiepileptic drugs (eg, levetiracetam) in severe cases to prevent posttraumatic seizures, as seizures are associated with poorer outcomes.

DECLARATION OF AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the authors used Chat GPT and Scholar GPT to create tables and identify research articles useful for this publication. After using this tool/service, the authors reviewed and edited the content as needed and took full responsibility for the content of the publication.

DISCLOSURES

The authors have no relevant financial disclosures.

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