

Management of Paediatric Traumatic Brain Injury: A Comprehensive Review of Current Evidence and Emerging Strategies

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ABSTRACT

Paediatric Traumatic Brain Injury (TBI) is a significant health concern, contributing to morbidity and mortality. Epidemiology shows variations influenced by demographics, age and injury severity. Status Epilepticus (SE) is a common complication of seizure activity, negatively affecting clinical outcomes. Secondary TBI increases neuronal damage, underscoring the necessity of effectively managing Intracranial Pressure (ICP) and Cerebral Perfusion Pressure (CPP). Assessing strategies such as direct ICP measurement and brain tissue oxygen monitoring is pivotal for informing interventions. Pharmacological agents, including osmotic therapies and antiepileptic medications, are crucial for managing TBI symptoms and preventing complications. Temperature monitoring is essential for reducing metabolic demand. Individuals surviving severe TBI (sTBI) face functional disabilities, cognitive impairments and mental health issues, impacting their quality of life. Despite diagnostic advancements, the pathophysiology and optimal management of paediatric TBI remain controversial. Nevertheless, global perspectives exist to enhance knowledge and optimise clinical management guidelines to improve outcomes. The present study aimed to provide a comprehensive review of the epidemiology, pathophysiology and management strategies for paediatric TBI, emphasising current approaches and emerging modalities.

Keywords: Cerebral perfusion, Intracranial pressure, Seizure complications, Status epilepticus

INTRODUCTION

Paediatric Traumatic Brain Injury (TBI) is a significant paediatric health concern due to its established association with the development of seizures and epilepsy. TBI results from external forces applied to the brain, causing multiple injury processes, including impacts from falls, vehicle accidents and athletic injuries, as well as penetrating injuries such as gunshot wounds and episodes of forceful shaking. These injuries can lead to brain contusions, intracerebral haemorrhage and various types of haematomas, each linked with prognosis for brain function and recovery [1]. Moreover, TBI severity can lead to long-term neurological, cognitive and psychosocial concerns beyond the primary injury [2].

Long-term sequelae include the development of Post-Traumatic seizures (PTS) and Post-Traumatic Epilepsy (PTE). Approximately 5-21% of children experience PTS and about 32-40% are at risk for recurrence, exceeding the general epilepsy prevalence of 0.7% [3]. This highlights the necessity of identifying risk factors for PTE, including age, injury severity, intracranial haemorrhage and early seizure activity after injury [1].

In relation to investigating PTE, it is vital to assess Status Epilepticus (SE), a neurological emergency defined by prolonged seizure activity, resulting in significant morbidity and mortality among paediatric populations. SE outcomes are influenced by several factors, including seizure duration and prompt treatment, underscoring the complexities of managing such cases [4]. Current treatment approaches for paediatric TBI and SE do not yield a universally accepted consensus; thus, a comprehensive understanding and literature review of TBI and its complications, including PTE and SE, are warranted [5-7].

The present study aimed to fill the knowledge gap by providing a thorough review of the epidemiology, pathophysiology and management of TBI and SE in the paediatric population, thereby contributing to clinical practice.

Epidemiology: The epidemiology of paediatric TBI reveals significant differences attributable to demographic variations, diagnostic criteria and data sources, contributing to substantial disability and mortality, particularly among children aged 0-4 years and adolescents aged 15-19 years [1]. In 2024, an estimated 837,000 children and adolescents aged 0-19 years experienced prolonged cognitive, physical, or behavioural disabilities as a consequence of TBI [8].

Approximately 500,000 children aged 0-14 years in the United States are treated in emergency departments for TBI each year, corresponding to an estimated 511,257 TBI-related visits, hospitalisations and fatalities annually [9]. A systematic review reported the yearly incidence of TBI at 691 per 100,000 among individuals aged 0-24 years based on emergency data [10]. The global incidence of paediatric TBI ranges from 47 to 280 per 100,000 children, with the United States reporting higher hospital stay rates than other countries [11].

Age and gender are key factors affecting TBI rates, with children aged 0-4 years exhibiting higher emergency visits at 1,591 per 100,000 [12]. Boys have higher TBI rates, with those aged 0-9 years being 1.4 times more likely and older boys 2.2 times more likely than girls [10]. Nevertheless, mild TBI (mTBI) has been reported more frequently among female athletes in high school and college [13].

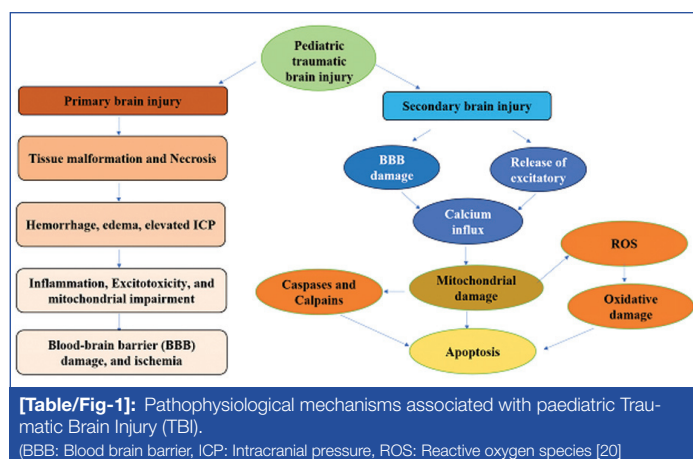
Nearly 80% of paediatric TBIs are classified as mTBI, with incidences of 692 per 100,000 among children aged 0-15 years [14,15], with global incidence ranging from 12 per 100,000 in Sweden to 486 per 100,000 in Australia [11]. Moreover, the Global Burden of Disease Study indicated that India exhibits an incidence of TBI that increased from 499 per 100,000 in 1990 to 554 per 100,000 in 2019 [16]. However, global estimates show a 5.5% decrease alongside stable prevalence rates, necessitating preventive measures to reduce the impact of TBI on paediatric populations [17]. This rise in incidence rates in India may reflect rapid urbanisation, increased motor-vehicle use and inadequate road-safety measures, whereas global declines are probably due to improved prevention strategies and better trauma-care systems in many countries.

DISCUSSION

Pathophysiology

Traumatic Brain Injury (TBI) progresses through two phases: primary injury and secondary brain injury. The initial stage occurs at the moment of impact, causing disruption of cellular membranes and alterations in ion gradients of potassium, sodium and calcium. Elevated intracellular calcium activates calpain, a proteolytic enzyme that degrades cytoskeletal structures. Calcium also activates N-methyl-D-aspartate (NMDA) receptors, leading to neuronal depolarisation, mitochondrial calcium overload and increased production of Reactive Oxygen Species (ROS), contributing to apoptosis. These changes impair oxidative metabolism, resulting in lactate accumulation, acidosis and cerebral oedema [18].

The secondary injury phase includes blood-brain barrier disruption, neuroinflammation, oxidative stress, excitotoxicity, cellular death and mitochondrial dysfunction, contributing to increased neuronal damage [19]. TBI triggers the release of excitatory neurotransmitters, including glutamate and aspartate, which activate NMDA and α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic Acid (AMPA) receptors, elevating intracellular calcium and triggering downstream pathways including calcineurin, calpain and caspases, leading to cell death [19]. Mitochondrial dysfunction plays a crucial role in secondary TBI, as mitochondria regulate calcium homeostasis through transport processes, including the Mitochondrial Calcium Uniporter (MCU) and uncoupling proteins [19]. This alters membrane potential, increases ROS and reactive nitrogen species and induces opening of the mitochondrial Permeability Transition Pore (mPTP). The pore releases apoptotic factors, including cytochrome c and apoptosis-inducing factor, promoting apoptosis via both caspase-dependent and caspase-independent pathways [18]. Pathophysiological mechanisms associated with paediatric TBI are shown in [Table/Fig-1] [20].



Diagnosis and Neuroimaging

Early monitoring is vital for evaluating the severity of brain injury and detecting patients in need of immediate surgical intervention. Computed Tomography (CT) is a widely used imaging modality due to its rapid availability and reliable outcomes [21]. Magnetic Resonance Imaging (MRI) has higher sensitivity than CT for detecting intracranial abnormalities including diffuse axonal injury; however, evidence regarding MRI's role in TBI management is limited [21]. Neuroimaging is commonly used to diagnose paediatric TBI and SE and to detect underlying causes. CT is utilised in critical scenarios for identifying haemorrhage and oedema, while MRI offers improved sensitivity for detecting signal changes on T2-weighted and Fluid-Attenuated Inversion Recovery (FLAIR) sequences associated with SE [22]. Moreover, Beauchamp MH et al., reported an increase in Cerebrospinal Fluid (CSF) volume and a decrease in total gray matter volume, suggesting that analysing volumetric alterations post-mTBI via T2- or FLAIR-weighted sequences could aid in detecting mild

injuries contributing to long-term non structural effects in specific locations, including the hippocampus [23].

Brain Oxygen Monitoring and Autoregulation

According to the Glasgow Coma Scale (GCS), paediatric TBI is classified as follows: scores of 13 or more indicate mild TBI (mTBI); scores 9-12 indicate moderate TBI (moTBI); and scores of 8 or less indicate severe TBI (sTBI). A preliminary neurological assessment is necessary to evaluate injury severity and resultant consequences. Elements such as level of consciousness, presence of neurological deficits and motor responses play important roles in GCS assessment; motor scores less than 3 are strong predictors of poor outcomes [24]. Pupillary response assessments are essential for brainstem function, particularly in sedated patients or those transitioning from mTBI to moTBI, as bilaterally fixed dilated pupils are associated with higher mortality [24]. Despite the limitations imposed by sedation and altered consciousness, continued cognitive examination is required to identify new deficits [25].

Significant metabolic demands of the brain require stable Cerebral Blood Flow (CBF), maintained by Cerebral Autoregulation (CA) to ensure continuous cerebral perfusion despite changes in systemic blood pressure. After TBI, neurocognitive abilities are often compromised, thereby increasing the risk of secondary brain injuries [25]. Recent clinical guidelines suggest age-specific CPP targets; however, they may not reflect the cerebrovascular autoregulation status of individual patients, potentially causing cerebral ischaemia or oedema [26]. Furthermore, these guidelines do not account for developmental changes in CA from infancy to adulthood, underscoring the need for development- and age-specific CPP targets.

Because direct measurement of CBF in clinical settings is challenging, surrogate indicators such as ICP, CPP, regional oximetry and transcranial Doppler are used [25]. In the paediatric population, continuous monitoring of arterial blood pressure (ABP) and ICP enables real-time assessment of CA. The pressure reactivity index (PRx) serves as a key marker of CA; values near zero or negative indicate preserved or favourable autoregulation, while a positive PRx indicates impaired autoregulation and poor prognosis [24]. PRx-guided CPP optimisation (CPPopt) differs from traditional CPP targets and deviations from CPPopt are associated with poorer outcomes [24]. The STARSHIP study, conducted in the UK, monitored CA-guided treatment in paediatric sTBI [27]. Innovative strategies such as wavelet PRx improve CPPopt precision, while RAP and the pulse amplitude index (Pax) are under investigation for establishing specific ICP thresholds [24]. These evolving approaches may improve paediatric TBI care by moving away from fixed CPP targets.

The TBI highlights the requirement of enabling sufficient cerebral oxygenation after injury. Evaluating parameters such as PbtO₂ assists in monitoring critical ischemic thresholds, with values below 8-10 mmHg [28]. Rapid, combined detection of decreased oxygenation with PbtO₂ is linked to prompt interventions, thereby enhancing outcomes [25].

Treatment

Intracranial Pressure (ICP) and Cerebral Perfusion Pressure (CPP) management Increased ICP is an important complication associated with acute neurological catastrophes, particularly in severe paediatric TBI (sTBI), as it influences secondary injury progression. paediatric TBI guidelines recommend maintaining ICP below 20 mmHg and achieving a minimum CPP of 40-50 mmHg, with age-dependent thresholds for CPP [26].

Woods KS et al., revealed that mean ICP, age-adjusted CPP and diagnosis predicted in-hospital mortality among children; the ICP threshold was lower in non TBI patients (15 mmHg) than in TBI patients (18 mmHg). Furthermore, a mean CPP of <67 mmHg was the sole predictor of mortality and exhibited age-dependent

modifications. In TBI cases, the mean CPP thresholds associated with mortality exceeded guideline targets (under 2 years: 45 mmHg; 2-8 years: 57 mmHg; 8 years and older: 68 mmHg) [29]. These findings highlight the need for accurately measuring physiological targets to improve outcomes.

Elevated ICP is observed in anoxic brain injury; however, its management is implemented to address post-anoxic oedema and is associated with outcomes regardless of whether it is a causal factor in neuronal death. Two paediatric studies indicated ICP as the sole predictor of mortality, rather than of neurological outcomes [30,31]. Another study in adults by Son SH et al., demonstrated an association between lumbar-puncture-measured ICP and neurologic outcomes after cardiac arrest; however, in paediatric populations, ICP thresholds related to outcomes have not been determined, nor has ICP-directed therapy efficacy been assessed [32]. This deficiency in defined ICP targets has stimulated interest in non invasive monitoring strategies.

Temperature control: Hyperthermia prevention is important for limiting brain injury; therefore, therapeutic hypothermia has been investigated as a viable treatment option in the paediatric population. Hutchison JS et al., demonstrated benefits with hypothermia; however, they reported that moderate hypothermia (32-33°C for 24 hours) increased mortality without improving secondary outcomes in paediatric sTBI [33]. Adelson PD et al., similarly reported no mortality benefit linked with prolonged hypothermia (48-72 hours) and slower rewarming, causing rapid trial futility in improving outcomes [34]. Tasker RC et al., in a meta-analysis, revealed no significant mortality advantage associated with hypothermia, while Bayesian analysis indicated a 33% probability of more than a 20% reduction in mortality risk [35]. Despite limited evidence, hypothermia is still used, underscoring the need for further research to determine potential benefits in paediatric TBI subgroups.

Hyperosmolar therapies: For addressing raised ICP in paediatric sTBI patients, intravenous osmotic therapy is used. Mannitol in 20% solution is given at a dosage of 0.5-1.0 g/kg, with additional doses guided by ICP levels. Nevertheless, there is a risk of hypovolaemia and hypotension, which should be avoided in paediatric sTBI patients [36]. A study by Kim JH et al., indicated that mannitol at a dose of 0.1 g/kg reduces ICP in 50% of paediatric patients with neurological conditions, whereas a dose of 1 g/kg was effective in 99% of cases [36]. Another study by O'Neill BR et al., indicated that mannitol decreases ICP and raises the pressure-volume index, linking its application to outcomes that surpassed predictions by trauma scores [28]. A systematic review by Stopa BM et al., included 11 studies and demonstrated that Hypertonic Saline (HTS) and mannitol can lower ICP and improve clinical outcomes in paediatric sTBI patients; however, the review lacks detail on treatment methods and outcome evaluation [37]. Considering such risks, HTS is the preferable strategy for increased ICP and CPP, with improved cerebral haemodynamics and ICP reduction in paediatric sTBI patients [29]. In a double-blind trial involving paediatric participants, Fisher B et al., indicated that 3% saline resulted in greater ICP reduction than 0.9% saline [38]. Chong SL et al., reported no significant difference in mortality or functional outcomes in paediatric patients with mild-to-severe TBI treated with either 3% HTS alone or 20% mannitol alone [39].

Pharmacologic therapies: Analgesics, sedatives and neuromuscular blocking agents are essential components of paediatric intensive care, aiding in managing ICP and facilitating mechanical ventilation. Opioids provide analgesia in conjunction with sedative effects, while sedatives decrease cerebral metabolic demands, reducing CBF and ICP. Gamma-aminobutyric Acid (GABA) agonists, including dexmedetomidine, benzodiazepines and barbiturates, increase inhibitory signalling and promote sleep induction. Propofol functions as a GABA agonist and NMDA antagonist with a short half-life; however, its use in paediatric populations is limited because of

infusion-related lactic acidosis [40]. Ketamine, previously avoided due to concerns about CPP and ICP through catecholamine release, but recent evidence indicates that it may not increase ICP and could enhance CPP; however, further investigation is required [40].

Neuromuscular blockers, in combination with sedatives, help decrease metabolic demands and reduce shivering during therapeutic hypothermia and require Electroencephalogram (EEG) monitoring to detect Seizures (SE). Pentobarbital, used to induce burst suppression on EEG, is employed for refractory intracranial hypertension and SE, though it carries risks including hypotension and respiratory depression and prolonged use increases the risk of ventilator-associated pneumonia [40].

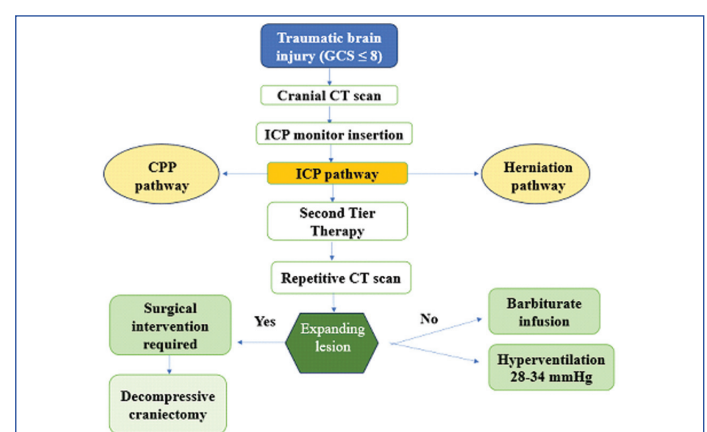
Limited evidence exists regarding comparative data in paediatric TBI; opioids provide transient relief from ICP but may contribute to neuroinflammation and deficits in myelination [20]. GABA agonists, particularly benzodiazepines, are associated with delirium and may impair neuronal recovery. Moreover, concerns about propofol-related neurotoxicity in paediatric anaesthesia may complicate its use [40]. Shein SL et al., determined that HTS is an effective agent for ICP reduction and CPP enhancement in comparison with fentanyl, revealing a higher failure rate [41].

Cerebrospinal fluid (CSF) diversion: For decades, CSF diversion has been utilised as an effective approach for managing sTBI in paediatric patients [40]. In a study involving 1,000 paediatric TBI patients, Bell MJ et al., indicated that 314 underwent CSF diversion, while 686 patients without intervention showed no significant variation in Glasgow Outcome Score-Extended for Paediatrics (GOS-EP) scores between groups. However, CSF diversion led to a significant reduction in overall ICP but did not improve outcomes at six months following TBI [42].

The CSF drainage occurred intermittently, with continuous drainage showing greater effectiveness in decreasing ICP [42]. Continuous CSF drainage is increasingly favoured for its proactive approach in managing intracranial hypertension. Timely initiation of treatment may reduce the need for additional ICP-directed strategies [29].

Decompressive craniectomy: Decompressive Craniectomy (DC), with or without duraplasty, is a controversial approach for addressing increased ICP or preventing cerebral herniation. Jaradat A et al., reported that DC yielded favourable outcomes (GOS 4-5) in 67% of paediatric sTBI cases; however, in-hospital mortality was 24%, with complications in 35.6% of patients [43]. In a randomised controlled trial, Thomale UW et al., revealed favourable outcomes in 92% of children undergoing DC [44], whereas Nagy L et al., reported similar outcomes in 75% of cases [45]. In contrast, Bruns N et al., observed high death rates in patients receiving DC treatment [46]. This highlights the potential of DC in paediatric sTBI, but variable outcomes require further studies to improve patient selection and determine optimal timing and duration.

Management strategies for Traumatic Brain Injury (TBI) are illustrated in [Table/Fig-2] [47].

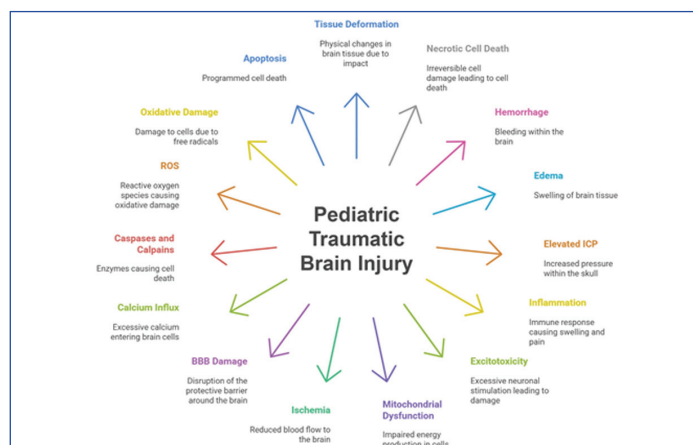


[Table/Fig-2]: Management strategies for Traumatic Brain Injury (TBI). CPP: Cerebral perfusion pressure, ICP: intracranial pressure [47].

Outcomes

Association between morbidity and mortality in paediatric TBI.

The association between morbidity and mortality in paediatric TBI is strongly influenced by injury severity and the effectiveness of measures taken to reduce secondary injury. Greater injury severity and a lower initial GCS score are associated with poorer outcomes and GOS scores decrease accordingly, with mortality in sTBI reaching up to 50% [Table/Fig-3] [40]. Sharples PM et al., observed a fourfold increase in mortality among paediatric patients experiencing hypotension and hypoxia [48]. Data from the Approaches and Decisions in Acute Paediatric TBI (ADAPT) trial reported a 19.1% mortality rate in sTBI children, suggesting a significant correlation between GCS and mortality risk [49]. Moreover, greater TBI severity is frequently associated with emotional and cognitive impairments, leading to neurological deficits, behavioural disorders and overall disability [50].



[Table/Fig-3]: Overview of paediatric TBI outcomes (BBB: Blood brain barrier, ICP: Intracranial pressure; ROS: Reactive oxygen species) [40].

In a retrospective study, Hwang SY et al., revealed that children under two years of age with TBI were more likely to experience vomiting and Post-Traumatic Epilepsy (PTE) as common findings, whereas children aged over two years primarily experienced confusion and disorientation. Nevertheless, children with TBI often show improvement with rehabilitation strategies [51]. Overall, rapid intervention and higher functional status may lead to shorter rehabilitation stays and better recovery outcomes. Paediatric rehabilitation utilises an interdisciplinary approach involving physical therapists, psychologists and allied services to foster resilience and enhance neural plasticity for long-term adaptation [52].

Cognitive and Neurodevelopmental Outcomes (Learning, Emotional and Behavioural): Cognitive assessments show that 60% of children with TBI score more than one standard deviation below the mean, while 40% have minimal adaptive behavioural performance. Better outcomes are associated with higher GCS scores (≥ 13), absence of seizures, higher socioeconomic status and greater social support [53]. Children with severe TBI exhibit more pronounced cognitive impairments, while those with mild injuries experience difficulties in functioning and visual memory deficits. Some disabilities may persist or evolve over time; however, a supportive family environment improves cognitive outcomes regardless of injury severity [54].

Neumane S et al., reported that despite initial recovery, 80% of children with TBI had moderate-to-severe impairments at 24 months, with long-term disabilities spanning cognitive, socio-emotional, physical, neurological and behavioural domains and injury severity served as a prognostic factor for outcomes [50]. Moreover, children with sTBI exhibit impairments in communication and motor skills; however, social support enhances communication and problem-solving abilities, thereby improving outcomes [53].

Kumar B et al., reported a significant positive correlation between GCS and GOS scores among children with TBI due to brain

contusions, extradural haematoma and skull fractures, with higher GCS scores linked to better recovery [55].

Chaitanya K et al., revealed a significant association between residual deficits and TBI severity; however, no significant association was observed between mode of injury and outcome [56]. Kapapa T et al., reported that children with mTBI often experience emotional, cognitive and behavioural concerns developing after hospital discharge, leading to social withdrawal, prolonged absence from school, aggressive behaviour and family tension [57]. Moreover, Keenan HT et al., reported that children experiencing sTBI, particularly from inflicted injuries, often had persistent cognitive and adaptive impairments, with outcomes influenced by family characteristics and injury severity [53].

Subcortical lesions are commonly identified in both accidental and non accidental TBI, with nearly one-third of children exhibiting motor and visual impairments, while more than 50% display cognitive impairments. Cognitive decline can occur without visual MRI lesions, as subcortical injury is linked with poor outcomes and delayed motor development [58].

Risk of epilepsy: Epilepsy has a substantial effect on outcomes after TBI in children. The risk of Post-Traumatic Epilepsy (PTE) is correlated with TBI severity, long-term developmental delays and negative outcomes [59]. Södal HF et al., in a large cohort study, reported an aggregate epilepsy incidence of 3.1% at two years, increasing to 4.0% at five years post-TBI [60]. Keret A et al., reported that children with sTBI have a 2.9-fold heightened risk of PTE compared with children with mTBI and that this is associated with longer hospitalisation and poorer recovery [3]. Laing J et al., depicted significant risk factors for early post-traumatic seizures following mTBI to sTBI, including extended hospitalisation, the need for mechanical ventilation and poorer 24-month outcomes, with increased mortality and PTE occurrence [59]. Moreover, Amonkar P et al., demonstrated significant risk factors and immediate outcomes associated with patients experiencing epilepsy and requiring Paediatric Intensive Care Unit (PICU) admission [61]. Mariajoseph FP et al., demonstrated risk factors for PTE and the significance of intracranial haemorrhage in the occurrence of later epilepsy [62]. Elsamadicy AA et al., reported that prolonged loss of consciousness post-TBI was associated with a lower incidence of PTE compared with those who returned to baseline more rapidly [63].

Sleep disturbances: The TBI can significantly affect recovery and quality of life in children, with manifestations including daytime sleepiness, increased sleep latency and circadian rhythm disturbances [64]. Luther M et al., reported that nearly 20% of children with TBI experience sleep disturbances such as Sleep-Wake Disturbance (SWD), fatigue and nightmares, with a negative association with cognitive function and overall quality of life [65]. Williams CN et al., demonstrated SWD in 56% of children, with 46% classified as critical cases and 68% of these children showing multiple SWD phenotypes [64].

Children with mTBI, compared with sTBI, exhibit higher scores on the Sleep Disturbance Scale for Children (SDS-C), suggesting that sleep-related problems may be more prominent in milder injuries relative to normal data [40]. Chronic sleep disturbances linked to memory impairments and increased fatigue highlight the need to address sleep problems in this vulnerable group [48].

Seizure Prophylaxis

Post-traumatic Seizures (PTS) are a significant complication of paediatric TBI, contributing to increased metabolic demand, Cerebral Blood Flow (CBF), Intracranial Pressure (ICP) and excitotoxic neuronal injury. Liesemer K et al., reported an incidence of PTS of 12% in a cohort of 275 paediatric patients, with heightened risk in mTBI to sTBI, children under two years and abusive TBI [66].

Pease M et al., in a systematic review and meta-analysis, found that seizure prophylaxis after mTBI to sTBI was associated with a small but statistically significant reduction in early PTS risk [67].

Prevention of secondary TBI from PTS is essential and phenytoin has traditionally been used for seizure prophylaxis [68]. The paediatric TBI guidelines include a level III recommendation for phenytoin use in preventing early PTS [69]. A retrospective study reported an incidence of PTS of 15% in children with sTBI receiving phenytoin prophylaxis versus 53% without treatment [70]. Literature suggests antiepileptic drugs, including phenytoin, fosphenytoin and phenobarbital, offer protection against early PTS [71]. However, adverse effects associated with phenytoin reduce the therapeutic index, with varied pharmacokinetics in vulnerable patients, leading many centres to restrict its use [71].

Seizure prophylaxis practices vary across trauma centres, with agents ranging from phenobarbital to levetiracetam, the latter preferred due to its wide therapeutic index and minimal monitoring needs [40]. Chung MG and O'Brien NF reported a 17.6% PTS incidence with levetiracetam use [68]. Al Jayyousi O et al., observed levetiracetam to be more effective against early PTS, while fosphenytoin depicted lower PTS rates and is preferred in high-risk cases [72]. Gupta N et al., demonstrated that surgical intervention including craniotomy was required in 28.66% of patients with PTS, with the majority achieving good recovery [73].

CONCLUSION(S)

Paediatric TBI carries a high risk of mortality and long-term disabilities. Effective treatment includes normalising physiological parameters, regulating ICP and CPP, using osmotic agents, antiseizure medications and applying brain monitoring tools. Early and multidisciplinary neurocritical care is essential for preventing secondary injury and optimising recovery. Current therapeutic strategies show efficacy, but novel treatments and improved protocols are still required. Continued research is necessary to enhance clinical outcomes in children affected by TBI and seizure disorders.

Acknowledgement

The authors would like to thank Dr. Vikas S. Sharma (MD), CEO, Maverick Medicorum® (India), for creating illustrations and medical writing services in the preparation of this article.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Jun 29, 2025
- Manual Googling: Aug 05, 2025
- iThenticate Software: Aug 21, 2025 (8%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? No
- Was informed consent obtained from the subjects involved in the study? NA
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: Jun 28, 2025

Date of Peer Review: Jul 14, 2025

Date of Acceptance: Aug 23, 2025

Date of Publishing: Dec 01, 2025