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Traumatic Brain Injury in Children

Dyah Kanya Wati

Abstract

Traumatic brain injury (TBI) in children occurs as a result of a sudden bump, roll, or jerk to the head or a penetrating injury to the head that interferes the normal brain function. Traumatic brain injury (TBI) is the leading cause of death and disability in children. More than half a million children present annually to the emergency department for TBI-related visits, and resulting in the death of >7,000 children annually in the United States, with highest incident rates seen in children aged 0–4 years and adolescents aged 15 to 19 years. In Indonesia, from Riskesdas data in 2013 shows the incidence of head trauma in children is about 0.5% of the population from other injury rates. Pediatric TBI is associated with an array of negative outcomes, including impaired cognitive and academic abilities, social impairments, and behavioral problems. The scalp is highly vascularized and a potential cause of lethal blood loss. Even a small loss of blood volume can lead to hemorrhagic shock in a newborn, infant, and toddler, which may occur without apparent external bleeding.

Keywords: TBI, injury, disability, children

1. Introduction

Advancement in knowledge regarding traumatic brain injury (TBI) is incessantly pursued, especially in terms of key terms definition establishment. The exertion of external force on the brain, whether directly or indirectly, which causes disturbance in its structural or functional aspect defines TBI [1–3]. While the former definition is generally accepted, significantly more heterogeneous definitions can be compiled for a term closely related to TBI named concussion.

Most definitions agreed to refer to the constellation of clinical symptoms measured by mainly neurologic and cognitive dysfunctions and not exclusively evident in mild TBI [4, 5]. However, other definitions associate concussion with sports [6, 7], and although there are fundamental overlapping parameters, several areas of multiplicity impede the development of a universally accepted definition. This issue may affect every aspect of TBI as it determines the case definition in any given research.

2. Epidemiology

The global incidence of TBI is estimated at 939 cases per 100,000 people – which translates to 69 million people sustain a TBI every year [8]. Decreasing incident (4.4%) and increasing prevalence (6.6%) of TBI in the United States during 1990 through 2017 are reported by the Global Burden of Disease Study, with the latest

prevalence and incidence reported are 2.104 and 0.961 million, respectively. TBI incidence is among the highest compared to other neurological disorders, even with age-adjustment at 285 cases per 100,000 people [9]. Pediatric TBI contributes to a global incident range of 47–280 per 100,000 children [10]. Changes in Coronavirus disease 19 (COVID-19) pandemic circumstances since 2020 seem to affect TBI epidemiology as implied by the significant decrease in mild TBI incident for children aged 0–5 years (44%) and 6–17 years (93%) in Canada [11].

The epidemiological characteristics are also invariably affected by geographical and sociodemographic features. Countries with the lowest and highest incidence are Sweden (12 cases per 100,000) and Australia (486 cases per 100,000). Higher incidence, severity, and mortality in pediatric TBI are observed in rural as opposed to urban areas [12]. Bimodal age distribution with peaks at 0–4 years and 14–18 years with male-gender preponderance in the pediatric population is observed [13]. Meanwhile, the role of race and socioeconomic status requires further confirmation [10].

The most common injury mechanisms are falls and motor vehicle accidents, although the relative proportions vary by age distribution. The majority of pediatric TBI cases are mild (70–90%), and severe TBI only accounts for 3–7% of all cases. Consistently, the hospitalization rate is 129 per 100,000 in pediatric population and over 90% recover [10, 13, 14]. Nevertheless, 88% of concussions are left undiagnosed and one-third of properly diagnosed cases may experience ongoing sequelae, which would remain undetected until the development reached frontal lobe maturity [1, 14].

3. Anatomical and physiological consideration

Before further consideration on injury mechanisms, it is important to appreciate the evolving anatomy and physiology in every stage of child development and its impact on injury biomechanics (Table 1). Note that this difference is also relevant to TBI diagnosis and management in children.

Children have a relatively higher head-body ratio and, consequently, greater relative head weight as opposed to adults. The large head size increases the possibility of experiencing head trauma, while the weight imposed results in distinct acceleration dynamics when exposed to external forces. Early-stage facial development is characterized by maximum craniofacial ratio, protruding forehead, and less developed paranasal sinuses. These unique properties subject increased likelihood of frontal trauma, especially with lesser capability of the sinuses to absorb the energy.

Younger children have thin calvarium rich in bone marrow with fontanels and sutures closing at different times. The pliable skull, along with open sutures and fontanels, allows for deformation and limited intracranial pressure (ICP) buffering. Hence, the existence of fracture should raise clinical suspicion for significant

Cranium	Head-body ratio, craniofacial ratio, fontanels and sutures patency, calvarium characteristics
Brain	Underdeveloped myelin sheaths, water content, pulsatility
Cervical	Relative position of the fulcrum of movement, underdeveloped neck muscles and ligaments, susceptible articulations

CSF, cerebrospinal fluid; ICP, intracranial pressure.

Table 1.
Age-dependent characteristics in TBI.

underlying parenchymal injury despite lacking evidence on imaging investigation. The downside of high skull plasticity with regards to cortical vessels and brain parenchyma is that it may cause stretching and shearing of these structures in response to the external force.

The craniocervical structures depend mainly on the ligaments and soft tissues for stabilization. Weaker neck muscles and ligaments, upper position of fulcrum of the vertebral body, and flexible articulations in younger children predispose to craniocervical instability particularly when combined with the disproportional head weight. Therefore, a high index of suspicion for concomitant spinal injury has to be maintained until proven otherwise.

Cerebral white matter is less myelinated and contains more water compared to that of adults. Although the nerve fibers are pliable and less likely to rupture, their pliability increases the risk of cerebral contusion and subdural hematoma. The unmyelinated areas are significantly more prone to injury. Cerebral compliance is also affected by other age-dependent factors, such as cerebral blood flow and volume and cerebrospinal fluid (CSF)-brain ratio [13, 15].

4. Biomechanics

The biomechanistic aspect of head trauma is composed of two forces: translation or linear (LA) and rotational (RA) accelerations. The former results from direct impact measured in gravitational force unit (g), whilst the latter results from indirect or whiplash impact and is measured as radians per second squared (rad/s^2). Upon sudden impact with a surface, the head experiences deformation and deceleration in the same direction as the initial force and result in LA. The bending of the skull produces a wave-like pattern which causes tension propagating from the outer to inner skull. Tension propagation magnitude and direction determine the ensuing fracture initiation.

Intracranial damage occurs as a consequence of either brain motion or pressure gradient established by the LA. Brain motion is proposed to potentiate focal hematoma directly. Other authors proposed that the focal site of an impact is exposed to positive gradient resulting in focal injury and the distal site is exposed to negative gradient resulting in shear stress and cavitation. Previous researches reported a strong correlation between LA and ICP, and ICP with subsequent neurologic dysfunction.

Holbourn was the pioneer researcher who stated that RA-mediated brain injury was caused by shear stress and strain. Impact duration should also be taken into account as different combinations of impact duration and magnitude result in different injury types. Longer duration at a lower magnitude of RA generates diffuse axonal injury, and the opposite generates subdural hematoma. Although LA and RA are often described separately, the inherent coupling of both forces is inevitable in reality [16, 17].

5. Pathophysiology

TBI pathogenesis involves primary and secondary injuries culminating in a temporary or permanent neurological deficit. Primary injury represents brain dysfunction as a direct result of brain deformation. Structural damage in focal, multifocal, or diffuse pattern in primary injury can only be prevented before the collision. Consequent molecular, chemical, and inflammatory cascades further extend the reversible secondary injury from minutes to days after the primary insult [18].

Theoretically, cerebral blood flow (CBF) and therefore, cerebral perfusion pressure (CPP) is determined by the difference between mean arterial pressure (MAP) and ICP. It is noteworthy that in TBI cases, studies demonstrated that the cerebral autoregulation mechanism impairment in the presence of normal CBF and CPP values. Decreased metabolic demand in coma or ischemic conditions may be the plausible explanation for cerebral hypoperfusion after TBI. Under such pathophysiologic conditions, CBF continues to decline to reach ischemic level thus exacerbating the impact of secondary injury [19]. CBF restoration may also cause reperfusion injury mediated by oxidative stress, leukocyte infiltration, and blood brain barrier dysfunction [20, 21].

Deterioration of CBF deprives the cells of their metabolic needs and forcing them to switch into anaerobic metabolism. Less energy and more lactate production in anaerobic metabolism give rise to failure in cellular functioning and generate an acidic milieu [20]. Moreover, the glial-neuronal uncoupling further enhances extracellular lactate production independent of ischemia, resulting in lactate storm in severe cases [22].

One of the main concern in the cerebral metabolic alteration is the failure of the sodium/potassium (Na/K) pumps. Massive sodium influx precipitates the cascades in secondary injury through neuron depolarization. Depolarized neurons release excitatory neurotransmitters, including glutamate and aspartate, which leads to intracellular calcium increase and enzymes and free radicals activation [20, 22, 23]. Neuronal cells degradation triggers neuroimmune responses and instigates BBB dysfunction, both of which add up to the cerebral edema progression [23, 24]. Vasogenic and cytotoxic edema in TBI is followed by raised ICP. According to the Monro-Kellie doctrine, the brain responds to the edema by displacing CSF and venous blood away. Failure of this compensation mechanism ultimately results in brain compression and death [20, 23].

6. Diagnosis and clinical manifestations

Amidst many classification systems and scales constructed for TBI diagnosis, this review focuses on definition generalizability since some recent studies focused solely on sports-related concussion. Disease severity is classified into mild, moderate, and severe based on GCS level and imaging findings.

6.1 Mild TBI

The mildest form of TBI, or concussion, conversely raises considerable concern because of its large proportion and rather unsettled recognition approach. This mild manifestation can coexist in more severe TBI cases. Despite vast heterogeneity in definitions provided, the common ground is that the patient is alert and experiencing any of the mild TBI clinical phenotypes after head injury. As observed in **Table 2**, no clear-cut definition for concussion and loss of consciousness alone is not a prerequisite in defining concussion. Some organizations focus on the clinical criteria, while others incorporate validated supplementary tools to objectify the assessment.

The most specific and systematic clinical criterias are provided by the Brain Trauma Foundation (BTF) [4] and Craton et al. [27] based on Concussion in Sport Group (CISG) guidelines. BTF clearly defined the clinical indicators and assigned specific time intervals for each in the first step of its guideline [4], whereas the second step of the guideline described clinical concussion subtypes and associated conditions [28]. Craton et al. on the other hand classified the symptoms into seven

	AAN [25]	BTF [4]	CDC [26]	CISG [7]	NINDS [2]
GCS	—	13–15	13–15 ≥ 30 minutes	—	—
Diagnostic criteria	Alteration in memory and orientation	Disorientation or confusion, impaired balance, slower reaction time, impaired verbal learning and memory	Confusion or disorientation, posttraumatic amnesia, focal signs, symptoms, or seizure	Symptoms, signs, balance impairment, behavioral changes, cognitive impairment, sleep/wake disturbance	LOC, headache, confusion, lightheadedness, dizziness, blurred vision, tinnitus, change in sleep patterns, behavioral or mood changes, and impaired memory, concentration, attention, or thinking
Supporting tool	PCSS, GSC, SAC		GSC, PCSS, HBI, PCSI	SCAT5, SAC	
Exclusion requirement	CT imaging in select cases		CT imaging in select cases (PECARN)	Clinical utility of advanced neuroimaging requires further validation	
Non-contributing factors	LOC		Hospitalization or neurosurgical intervention requirement	LOC	

AAN, American Academy of Neurology; BMI, body mass index; BTF, Brain Trauma Foundation; CDC, Centers for Disease Control and Prevention; CISG, Concussion in Sport Group; CT, computed tomography; GCS, Glasgow Coma Scale; GSC, Graded Symptom Checklist; HBI, Health and Behavior Inventory; LOC, loss of consciousness; NINDS, National Institute of Neurological Disorders and Stroke; PCSI, Post-Concussion Symptom Inventory; PCSS, Post-Concussion Symptom Scale; PECARN, Pediatric Emergency Care Applied Research Network; SAC, Standardized Assessment of Concussion; SCAT5, Sports Concussion Assessment Tool version 5.

Table 2.
Proposed definitions of concussion.

clinical phenotypes with COACH CV mnemonics and suggested specific testings in addition to supplementary tools for identifying each phenotype [27].

Supporting tools proposed by the guidelines are meant as a diagnostic adjunct to clinical indicators. The tools are validated and mentioned in the order of their priority as in the actual guideline. Each tool is age- and condition-specific, therefore careful consideration should be taken before administering and interpreting the results in decision making strategy. Computed tomography (CT) scan is indicated in select cases when more severe TBI or complication is suspected or as suggested by intermediate or high risk in Pediatric Emergency Care Applied Research Network (PECARN) decision rules.

6.2 Moderate and severe TBI

Detecting moderate and severe TBI cases are more straightforward with commonly accepted classification based on the level of consciousness measured in pediatric Glasgow Coma Scale (GCS) [29] and evidence of pathological imaging findings. GCS level lower than 9 is considered severe TBI, while GCS level within the range of 9–13 is considered moderate TBI. Based on anatomical structure

	Skull fracture	Intracranial bleeding	Cerebral contusion	Diffuse axonal injury	Abusive head trauma
Clinical findings	Subcutaneous swelling	Seizure (SAH)	—	Coma, decorticate or decerebrate posturing, neuropsychiatric impairment	Abnormal shaking behavior as mechanism of injury, seizure, retinal hemorrhage, rib fracture
Main radiological evidence	Linear, depressed, basal skull, or growing skull fracture	Hyperdense lesion in specific configurations	Mixed-density lesion surrounded by perilesional hypodense area	Foci of reduced diffusion and increased susceptibility	Coexistence of multiple hematomas with different onsets

SAH, subarachnoid hemorrhage

Table 3.
 Diagnostic features of primary TBI manifestations [5, 13, 30].

involvement, primary TBI clinically manifests as skull fracture, extraparenchymal injury, intraparenchymal injury, and vascular injury, while secondary TBI manifests as diffuse cerebral swelling [13]. TBI manifestations are summarized in **Table 3**.

The appropriate imaging modality choice according to the American College of Radiology appropriateness criteria [31] depends on TBI onset and severity, risk assessment by PECARN criteria, and cognitive and neurologic signs. This guideline requires the exclusion of abusive head trauma in all cases and posttraumatic seizure in chronic cases. CT scan is recommended for acute and subacute cases, whilst magnetic resonance imaging (MRI) is recommended in subacute and chronic cases.

7. Management

Management strategy contingent on the severity of TBI. Management of mild cases highlights the importance of gradual rehabilitation while maintaining strict adherence to injury prevention. Indispensable emergency and intensive care in more severe cases warrant separate management planning.

The general strategy to manage mild TBI cases begins with complete rest. Once the child advance to a gradual return to regular activity, it is imperative to avoid any movement or activity that would provoke symptoms. Each of the next steps should be taken for at least 24 hours long, and any worsening of symptoms would render the child retreat to the previous step (**Table 4**). Similar gradual progression should also be applied to cognitive activities, especially in cases where mental activities exacerbate the symptoms. General preventive measures in commuting and playing sports should be exercised regularly [5].

Unconscious pediatric TBI patients need emergent tracheal intubation is recommended, along with the appropriate sedative or analgesic agent. Benzodiazepines are proven for their antiepileptic, anxiolytic, and amnestic properties. The dosage of benzodiazepine and opiate administrated is guided by proper preservation of mean arterial and cerebral perfusion pressure. The risk of respiratory depression as

Step	Activities	Time	Goal
Rest	No activity	24–48 hours	—
Nonaerobic activity	Normal daily activities	—	School or work activities
Light activity	Exercises at slow pace	5–10 minutes	Mild increase in heart rate
Moderate activity	Light resistance activities	Reduced than usual	Limited movement
Heavy noncontact activity	Noncontact exercises	Near usual	Intense activity
Full contact activity	Normal activities	Normal	Return to usual full-contact activities
Competitive activity	Full competitive activities	Normal	No restriction

Table 4.
Step-by-step to achieve the return to play [5].

the side effect of sedative agents could be prevented by securing airway and optimizing ventilation. Controlled mechanical ventilation for initial support by FiO₂ titration to achieve target SpO₂ of 92–99% or PaO₂ 75–100 mmHg is recommended. The most recent proper ventilation goal involves preventing hyperventilation, hypocapnia, and hypoxia [32–34].

Optimal intravascular volume status encompasses central venous pressure (CVP) and urine output monitoring, blood urea nitrogen and serum creatinine assessment, fluid management, and nutrition therapy. Normovolemic status is achieved by administering normal saline as much as 75% of the maintenance requirement to maintain CVP between 4–10 mmHg and urine output >1 ml/kg/hour. Initial use of 5% dextrose in normal saline infusion may be necessary to avoid hypoglycemia in younger patients. Nutrition therapy should start as early as 72 hours. The core temperature should be maintained within >35 °C and < 38 °C [33].

The first tier after baseline care is maintaining ICP threshold below 20 mmHg. Levels above this threshold urge intervention by methods in the following order: CSF drainage, hyperosmolar therapy, analgesic and/or sedation escalation, or neuromuscular blocker initiation should be considered. Coupling nature of ICP and CPP means that the increase in ICP is often followed by CPP improvement. Permissive intracranial hypertension remains an option, although the second tier of maintaining the CPP threshold should be decided carefully due to precipitous herniation risk. Age-specific CPP threshold ranges between 40–50 mmHg in concordance with increasing pediatric age extremes. Refractory increase in ICP despite first tier treatment requires a repeat CT scan when surgical option is indicated. Surgical intervention to remove mass and/or decompressive craniectomy is indicated when new or expanding lesion is detected [33].

8. Future directions

Pediatric TBI poses a great challenge with wide-ranged prognosis. Both mild and severe extremes in the TBI severity spectrum necessitate thorough assessment and management strategies. Future endeavors should be directed to establish universal definition of concussion, more reliable biomechanical models, optimal treatment algorithm, and effective prevention strategies.

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