

# Advanced Nursing Perspectives on the Assessment, Early Recognition, and Clinical Management of Pediatric Abusive Head Trauma- An Updated Review

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## Abstract:

**Background:** Abusive head trauma (AHT) is the leading cause of traumatic brain injury and fatal physical abuse in infants, characterized by complex mechanisms, diagnostic challenges, and severe long-term consequences.

**Aim:** To provide an updated review of the assessment, early recognition, and clinical management of pediatric AHT from an advanced nursing perspective.

**Methods:** This review synthesizes current evidence on etiology, epidemiology, pathophysiology, clinical presentation, evaluation strategies, and multidisciplinary management, including the critical nursing role.

**Results:** AHT commonly results from shaking, impact, or combined forces, producing subdural hematomas, retinal hemorrhages, diffuse axonal injury, and cerebral edema. Early recognition requires careful history-taking, comprehensive physical assessment, neuroimaging, laboratory testing, and ophthalmologic evaluation. Effective management depends on ATLS-based stabilization, intracranial pressure control, seizure prophylaxis, surgical intervention when indicated, and coordinated multidisciplinary care. Nurses play pivotal roles in assessment, monitoring, documentation, and caregiver education.

**Conclusion:** Early identification, standardized evaluation, and evidence-based management significantly improve outcomes and enhance child safety.

**Keywords:** Abusive head trauma, shaken baby syndrome, pediatric brain injury, nursing assessment, intracranial pressure, retinal hemorrhage, child abuse.

## Introduction:

Abusive head trauma (AHT) is recognized as a severe and preventable form of inflicted injury in early childhood. The Centers for Disease Control and Prevention (CDC) defines AHT as damage to the intracranial contents or skull of a child from birth to five years of age that results from blunt impact, violent shaking, or a combination of these mechanisms [1]. This definition underscores the pathophysiological consequences of inflicted mechanical forces rather than limiting the diagnosis to a single mechanism of injury. AHT represents the most serious manifestation of physical child abuse and remains the leading cause of traumatic brain injury (TBI) in infants and toddlers, with substantial rates of mortality and long-term morbidity [2][3][4][5]. Historically, the term shaken baby syndrome (SBS) was employed to describe the constellation of injuries associated primarily with forceful shaking. Over time, advancements in neuroimaging, biomechanics, and forensic medicine have expanded the conceptual framework surrounding inflicted pediatric head injury. Contemporary terminology reflects a broader understanding that these injuries may arise from repetitive acceleration-deceleration forces, rotational stresses, blunt impact, or combined mechanisms acting upon the developing brain and cranial structures [6]. This evolution in language acknowledges the complexity of injury patterns and avoids constraining diagnostic reasoning to a singular etiology.

One of the principal challenges in diagnosing AHT lies in the nonspecific nature of its clinical presentation. Infants and young children may exhibit symptoms that resemble those associated with accidental trauma or common pediatric illnesses. Irritability, vomiting, lethargy, poor feeding, apnea, or altered responsiveness may initially appear indistinguishable from benign conditions. The absence of external signs of injury frequently complicates the diagnostic process, and external bruising or scalp trauma may be minimal or entirely absent [7]. Consequently, clinicians must maintain a high index of suspicion when evaluating infants presenting with neurological compromise, particularly in the absence of a plausible or developmentally consistent explanation. Accurate identification of AHT requires a meticulous and developmentally informed clinical history. Reported accounts are often vague, inconsistent, or subject to revision over time. Caregivers rarely acknowledge intentional harm, and explanations frequently include short-distance falls from cribs, beds, highchairs, or stairs. Such accounts may not align with the child's motor abilities or with established biomechanical evidence regarding the forces required to generate significant intracranial injury. In some cases, responsibility may be attributed to siblings whose developmental stage makes the proposed mechanism improbable. These discrepancies between reported history and observed injury patterns are critical components of clinical assessment and should prompt thorough investigation.

The neurological consequences of AHT are profound and stem largely from hypoxic-ischemic injury, diffuse axonal damage, and secondary cerebral edema. Infants may present with apnea, seizures, altered consciousness, or signs of increased intracranial pressure. Retinal hemorrhages are frequently observed and reflect the transmission of acceleration and rotational forces to the ocular structures [8]. The clinical course varies widely, ranging from complete neurological recovery in milder cases of TBI to permanent cognitive impairment, motor dysfunction, developmental delay, and epilepsy. In severe presentations, extensive cerebral swelling, herniation, or irreversible hypoxic injury may culminate in death. The long-term burden associated with AHT extends beyond immediate neurological sequelae. Survivors often require lifelong medical care, rehabilitative services, educational support, and psychosocial interventions. Persistent deficits may involve intellectual disability, behavioral dysregulation, visual impairment, and motor disabilities such as cerebral palsy. The economic and societal costs are substantial, encompassing direct healthcare expenditures, long-term disability support, and indirect impacts on families and communities [9]. These consequences reinforce the urgency of prevention and early detection strategies.

Effective prevention of AHT depends on multifaceted interventions that address caregiver stress, mental health challenges, substance use, and social determinants of health. Educational initiatives that inform caregivers about normal infant crying patterns and safe coping strategies have demonstrated measurable reductions in abusive injuries. Early identification of psychosocial risk factors, including intimate partner violence, financial instability, and untreated mental illness, enables targeted support services. Clinician education also plays a central role, as enhanced training in recognizing injury patterns associated with abuse promotes timely intervention and improved outcomes. Comprehensive risk assessment should incorporate

evaluation of the child's physical findings, family dynamics, and environmental context to inform protective strategies. The American Academy of Pediatrics and the CDC define AHT as intentional injury to the skull or intracranial contents of children aged 0 to 5 years resulting from blunt force, forceful shaking involving acceleration, deceleration, and rotational components, or a combination of these forces. This definition emphasizes the inflicted nature of the injury rather than restricting interpretation to a specific mechanical action. Several alternative terms have been used in clinical and legal contexts, including shaken baby syndrome, battered child [10], whiplash shaken infant syndrome [11], nonaccidental trauma, intentional head trauma, and inflicted head trauma. Although terminology has evolved, the underlying principle remains the recognition of deliberate harm resulting in structural brain injury.

Brain injury constitutes the leading cause of traumatic death among children younger than two years and remains the predominant cause of injury-related mortality during infancy [12]. The vulnerability of the infant brain is attributable to its high water content, incomplete myelination, and relatively large head-to-body ratio. Weak cervical musculature further predisposes infants to significant intracranial forces during acceleration and deceleration events. These anatomical and physiological characteristics amplify the destructive potential of violent shaking or blunt impact. Shaken baby syndrome, historically conceptualized as a subset of AHT, is characterized by a triad of cerebral edema, retinal hemorrhage, and subdural hematoma (SDH). Cerebral edema signifies severe parenchymal injury and contributes to elevated intracranial pressure and secondary ischemic damage. Retinal hemorrhages associated with accidental trauma are generally limited in extent and typically occur in the context of high-impact events such as motor vehicle collisions.

In contrast, extensive, multilayered retinal hemorrhages have demonstrated strong associations with inflicted injury. A study conducted in France reported that grade 3A and 3B retinal hemorrhages were highly specific for SBS, with a positive predictive value of 0.96. Hemorrhages extending beyond the posterior pole of the retina exhibit high specificity for severe head injury, particularly in cases of AHT. Subdural hematomas in the context of AHT often result from tearing of bridging veins due to rotational acceleration. These collections may be acute, subacute, or chronic, and the presence of mixed-density blood products on neuroimaging may indicate repetitive injury. The combination of subdural bleeding, diffuse cerebral swelling, and extensive retinal hemorrhage should prompt urgent multidisciplinary evaluation. Early neuroimaging, comprehensive ophthalmologic assessment, and involvement of child protection services are essential components of clinical management. In summary, abusive head trauma represents a critical pediatric health issue characterized by complex pathophysiology, diagnostic challenges, and devastating long-term consequences. Its recognition requires careful synthesis of clinical findings, imaging results, developmental assessment, and historical consistency. Continued emphasis on prevention, caregiver education, and clinician training remains central to reducing incidence, mitigating harm, and safeguarding vulnerable children.

### **Etiology**

Abusive head trauma arises from a complex interaction of child vulnerabilities, caregiver characteristics, and environmental stressors. No single factor causes inflicted injury. Instead, risk accumulates across biological, psychological, and social domains. Understanding these determinants strengthens prevention efforts, improves screening strategies, and supports early intervention before harm occurs. Although identifiable risk factors increase probability, their absence does not exclude the possibility of abusive head trauma. Clinical vigilance remains essential in all pediatric populations. Infant characteristics play a significant role in precipitating caregiver frustration that may escalate to violent responses. Excessive or inconsolable crying is among the most consistently identified triggers. Peak crying typically occurs between six and eight weeks of age, often described as the "purple crying" period [13]. During this developmental stage, crying may intensify without clear cause and resist soothing attempts. Caregivers who misinterpret persistent crying as defiance or illness may experience feelings of inadequacy, anger, or helplessness. Without appropriate coping strategies, this stress can culminate in forceful shaking or impact. Infantile colic further compounds this vulnerability. Colic is characterized by prolonged periods of unexplained crying in otherwise healthy infants. Although self-limited, it places substantial emotional strain on caregivers. Sleep deprivation, repeated soothing failure, and perceived loss of control increase the likelihood of impulsive

reactions. Education regarding the normalcy and temporary nature of colic reduces maladaptive responses and promotes safer coping mechanisms. Developmental delay and physical disabilities also elevate risk. Infants with neurodevelopmental impairments may cry differently, feed poorly, or fail to respond to social cues in expected ways. Caregivers may perceive these behaviors as rejection or noncompliance. Chronic medical conditions requiring frequent hospital visits, complex medication regimens, or specialized equipment add further stress. The burden of caregiving can intensify financial hardship, disrupt employment, and limit social engagement. Prematurity and low birth weight contribute additional strain. These infants often require prolonged neonatal intensive care, ongoing monitoring, and heightened vigilance for complications. Multiple births amplify caregiving demands and fatigue. Unplanned pregnancy has also been associated with increased risk, potentially reflecting reduced emotional preparedness or inadequate support systems [14].

Caregiver characteristics substantially influence the likelihood of inflicted injury. Young parental age correlates with higher rates of abusive head trauma, particularly when accompanied by limited social support. Adolescents and young adults may lack experience in managing infant behavior or regulating emotional responses under stress. Low socioeconomic status intensifies daily challenges, including housing instability, food insecurity, and restricted access to healthcare. Financial hardship erodes coping capacity and may increase interpersonal conflict within households. Limited parental education can impair understanding of normal child development. Misinterpretation of age-appropriate crying or sleep patterns fosters unrealistic expectations. Caregivers who believe infants cry intentionally or possess advanced self-regulatory abilities may respond with frustration when expectations are unmet. Absence of prenatal care may reflect broader disengagement from healthcare systems, reduced exposure to anticipatory guidance, and missed opportunities for early education regarding infant behavior. Lack of childcare experience increases vulnerability, particularly among first-time parents. Inexperienced caregivers may struggle to differentiate between normal developmental behaviors and signs of illness. Poor impulse control and low frustration tolerance are psychological factors frequently associated with abusive behavior. Individuals with difficulty managing anger or stress are more likely to respond with physical force during moments of heightened emotion. Mental illness and substance use disorders further elevate risk. Depression, anxiety, and personality disorders may impair judgment and emotional regulation. Substance intoxication lowers inhibition and increases impulsivity, while withdrawal states may intensify irritability.

Single-caregiver households face distinct challenges. Without shared caregiving responsibilities, stress accumulates rapidly. Sleep deprivation and social isolation reduce resilience. Prior involvement with child protective services signals historical patterns of family dysfunction or prior maltreatment. These histories require careful evaluation and ongoing support to prevent recurrence. An inaccurate understanding of child development remains a central contributor. Unrealistic expectations regarding toileting, sleep consolidation, feeding patterns, or behavioral control predispose caregivers to disappointment and anger when developmental milestones are not achieved prematurely. Community-level determinants exert powerful influence. Social isolation limits opportunities for emotional support, respite care, and shared parenting experiences. Families residing in neighborhoods lacking recreational spaces or community engagement may experience heightened stress. Poverty and unemployment compound financial insecurity and psychological strain. Economic instability correlates with increased rates of domestic conflict and reduced access to healthcare resources. Exposure to domestic violence, including intimate partner violence, significantly increases risk. Children living in violent households face both direct and indirect harm. Caregivers subjected to partner violence may experience depression, anxiety, and reduced coping capacity, while perpetrators of domestic violence may also perpetrate child abuse. Limited availability of supportive community services restricts early intervention. Absence of accessible mental health care, parenting education programs, and crisis support lines leaves caregivers without constructive outlets during periods of distress. The presence of an unrelated adult male caregiver in the home has been associated with elevated risk [15]. This factor may reflect complex family dynamics, reduced biological attachment, or increased household instability.

Perpetrator profiles vary widely. Male caregivers are most frequently implicated, particularly fathers, stepfathers, and the mother's boyfriend [16][17]. Female perpetrators, including babysitters and biological

mothers, are also represented. The diversity of perpetrators underscores that abusive head trauma does not arise from a single demographic profile. Instead, it emerges when caregiver stress intersects with vulnerability and inadequate coping strategies. Risk factor identification supports targeted prevention but cannot function as a diagnostic substitute. Many families with multiple risk factors never commit abuse. Conversely, abusive head trauma can occur in households lacking obvious indicators. Therefore, prevention strategies must balance universal education with selective interventions. Universal approaches include anticipatory guidance regarding normal crying patterns and safe soothing techniques. Selective strategies focus on families experiencing mental health challenges, financial hardship, or social isolation. Early screening for caregiver depression, substance use, and domestic violence during pediatric visits provides opportunities for referral and support. Community-based programs offering parenting education and stress management training reduce incidence rates. Evidence suggests that structured education regarding the purple crying period lowers hospitalization for abusive head trauma in regions where programs are implemented [13]. Integrating social services, mental health care, and pediatric follow-up strengthens protective networks. In summary, the etiology of abusive head trauma reflects the cumulative effect of infant vulnerability, caregiver stress, and environmental adversity. Each domain contributes to overall risk, yet no single element determines outcome. Prevention requires recognition of these interconnected influences, proactive caregiver education, and coordinated community support systems. By addressing both individual and structural determinants, healthcare professionals can reduce incidence and safeguard vulnerable children.

### **Epidemiology**

Determining the true incidence of abusive head trauma remains complex. No centralized national registry captures all confirmed cases, and reporting systems vary across jurisdictions. Clinical presentation ranges from subtle neurological changes to catastrophic collapse. Many cases go unrecognized during early encounters, particularly when symptoms are nonspecific. Repeated episodes of maltreatment often precede a definitive diagnosis, which further obscures accurate counting. Underreporting and misclassification contribute to persistent gaps in epidemiologic data.

In the United States, estimates suggest that the annual incidence of abusive head trauma among infants younger than one year ranges from 25 to 35 per 100,000 [18]. These figures likely underestimate the true burden. Surveillance studies relying on hospital discharge data capture only cases that reach medical attention and are correctly coded. Infants who die before evaluation or whose injuries are misattributed to accidental causes may not be included. Despite these limitations, abusive head trauma remains the leading cause of fatal physical abuse in children. The majority of fatalities occur during the first year of life, highlighting infancy as a period of extreme vulnerability [19]. Mortality rates reported in clinical series range from 10 percent to 20 percent. Survivors frequently sustain permanent neurological impairment. The combination of high mortality and high morbidity positions abusive head trauma as one of the most devastating forms of pediatric injury. Epidemiologic studies consistently demonstrate that brain injury accounts for the largest proportion of abuse-related deaths in early childhood. This pattern reflects both the fragility of the developing brain and the severity of forces applied during inflicted trauma.

Age-specific variation provides important insight into risk patterns. Incidence peaks between six and eight weeks of age. This interval corresponds with a well-described increase in infant crying behavior, often termed the “purple crying period.” During this stage, crying intensifies in frequency and duration, often without clear precipitating factors. Caregivers may struggle to console the infant despite appropriate feeding, diapering, and soothing. The temporal overlap between peak crying and peak incidence of abusive head trauma supports the association between caregiver frustration and inflicted injury. After the first year of life, incidence declines sharply to approximately 3.8 per 100,000 annually [20]. Improved head control, decreased crying intensity, and evolving caregiver adaptation likely contribute to this reduction. Although infancy carries the highest risk, abusive head trauma can occur beyond the first year. Toddlers remain vulnerable, particularly in environments characterized by high stress and limited supervision. Epidemiologic patterns emphasize the need for anticipatory guidance during early well-child visits, especially within the first two months of life. Prevention strategies that target this narrow developmental window may yield measurable reductions in incidence. Geographic variation within the United States has

also been documented. Higher incidence rates have been reported in the Midwest, whereas lower rates have been observed in the Northeast. The reasons for these regional differences remain uncertain. Variability in reporting practices, socioeconomic conditions, access to healthcare, and implementation of prevention programs may contribute. Differences in clinician awareness and screening protocols may also influence case identification. Without standardized national surveillance, distinguishing true epidemiologic variation from diagnostic inconsistency remains challenging.

Sentinel injuries represent a critical epidemiologic concept in abusive head trauma. These minor but concerning injuries frequently precede severe or fatal events. Nearly one quarter of children later diagnosed with abuse had prior healthcare encounters during which injuries were either missed or misinterpreted. Bruising and intraoral trauma are the most common sentinel findings, particularly in children younger than three years [21]. In nonambulatory infants, any unexplained bruise warrants careful evaluation. Studies suggest that up to 20 percent of sentinel injuries are not recognized during initial clinical assessment. Missed opportunities for intervention allow abuse to escalate. Early identification of these warning signs provides a window for protective action. Variation in clinical practice further complicates epidemiologic interpretation. Screening decisions may be influenced, consciously or unconsciously, by caregiver demographics, socioeconomic background, or ethnicity. Such bias can result in both overevaluation in certain populations and underrecognition in others. Inconsistent application of diagnostic imaging, skeletal surveys, and ophthalmologic examinations affects reported incidence. Standardized screening protocols have demonstrated improved detection rates and reduced disparities in care [22]. When clinicians apply uniform criteria for evaluation of head injury in infants, identification of abusive head trauma becomes more consistent across populations. You should consider how practice variation influences reported statistics. If one institution screens all infants with intracranial hemorrhage using a structured algorithm, detection rates will differ from those at centers relying solely on clinical suspicion. Epidemiologic figures therefore reflect both true disease burden and the quality of detection systems. Implementation of evidence-based guidelines narrows this gap and strengthens data reliability. Socioeconomic stressors, public health crises, and community instability may also influence incidence trends over time. Periods of economic hardship correlate with increased reports of child maltreatment in several observational studies. Monitoring temporal changes in abusive head trauma rates provides insight into broader social pressures affecting families. In summary, the epidemiology of abusive head trauma reveals a condition concentrated in early infancy, marked by significant mortality, and influenced by diagnostic practices and social determinants. Incidence estimates in the United States range from 25 to 35 per 100,000 infants annually, with mortality reaching up to one fifth of affected children [18][19]. Peak occurrence aligns with the early weeks of life, particularly between six and eight weeks [20]. Geographic variation and clinical practice differences shape reported rates, while missed sentinel injuries underscore gaps in early recognition [21][22]. Accurate surveillance, standardized screening, and early preventive education remain essential for reducing incidence and improving outcomes.

### **Pathophysiology**

Abusive head trauma results from complex biomechanical forces that disrupt the structural and functional integrity of the developing brain. The primary mechanisms include rotational acceleration, translational movement, and direct impact. These forces rarely occur in isolation. Violent shaking generates rapid angular acceleration and deceleration, while blunt impact introduces additional contact forces. When combined, these mechanisms amplify tissue deformation and vascular disruption within the cranial vault. Experimental research using computational simulations, biomechanical surrogates, animal models, and postmortem analyses has clarified how these forces translate into specific intracranial injuries. Infants possess unique anatomical and physiological characteristics that increase vulnerability to these forces. The head of an infant is proportionally larger relative to body size compared with older children and adults. Neck musculature remains underdeveloped and provides limited stabilization during rapid motion. Ligamentous structures exhibit increased laxity, allowing greater rotational displacement of the head. The infant skull is incompletely ossified and more compliant, altering how external forces are transmitted to intracranial contents. Additionally, the brain contains a higher proportion of water and reduced myelination, which affects mechanical resilience. These features collectively magnify the effects of acceleration-deceleration

forces and predispose the infant brain to diffuse injury patterns. Rotational forces are particularly damaging. Rapid flexion and extension during shaking produce angular acceleration that generates shear stress across brain tissue. Bridging veins traversing the subdural space are especially susceptible to tearing under these conditions. Disruption of these vessels leads to subdural hematoma and, in some cases, subarachnoid hemorrhage. Accumulating blood increases intracranial volume within the fixed cranial compartment. As hematomas expand, intracranial pressure rises. Elevated intracranial pressure reduces cerebral perfusion pressure, impairing oxygen and nutrient delivery to neural tissue. Secondary ischemic injury then compounds the primary mechanical insult [22][23].

Diffuse axonal injury represents another critical component of pathophysiology. Shearing forces disrupt axonal membranes and cytoskeletal elements, impairing neuronal communication. This process triggers a cascade of ionic imbalance, excitotoxic neurotransmitter release, and mitochondrial dysfunction. Cellular energy failure follows, leading to cytotoxic edema and further neuronal damage. The resulting impairment resembles patterns observed in severe traumatic brain injury. Clinical manifestations such as altered consciousness, seizures, and apnea reflect widespread disruption of neural networks. Cerebral edema develops through both cytotoxic and vasogenic mechanisms. Cellular swelling increases brain volume, while disruption of the blood-brain barrier permits extravasation of plasma components into interstitial spaces. Progressive edema exacerbates intracranial hypertension. If compensatory mechanisms fail, brain herniation may occur. Herniation compromises brainstem structures responsible for respiration and cardiovascular regulation, which can lead to rapid clinical deterioration. Direct impact contributes additional injury pathways. When the infant's head strikes a surface, focal contact forces may produce skull fractures or cortical contusions. Coup injuries occur at the site of impact, while contrecoup injuries may develop on the opposite side due to brain movement within the skull. In clinical scenarios, many cases involve both inertial forces from shaking and contact forces from impact. The combination intensifies tissue strain and vascular injury. Repeated episodes of abuse produce cumulative damage. Progressive hemorrhage, recurrent hypoxic events, and sustained elevations in intracranial pressure amplify neuronal loss over time. Each subsequent insult occurs in a brain already compromised by prior injury, reducing physiological reserve and worsening outcomes. This cumulative process explains why some infants present with mixed-age hemorrhages or imaging findings suggesting prior trauma. In summary, the pathophysiology of abusive head trauma reflects the interaction of rotational acceleration, translational displacement, and impact forces acting on a structurally vulnerable brain. Vascular disruption, diffuse axonal injury, cerebral edema, and intracranial hypertension interact to produce severe neurological compromise. The developing anatomy of infancy magnifies these effects, and repeated injury intensifies cumulative damage, leading to high rates of morbidity and mortality [22][23].

### **History and Physical**

The diagnosis of abusive head trauma requires careful clinical judgment, particularly in preverbal infants who cannot describe symptoms or provide context for injury. Presentation often begins with vague and nonspecific concerns such as irritability, decreased feeding, or altered sleep patterns [23]. These early manifestations can resemble common pediatric illnesses, which increases the risk of misinterpretation. A central element of evaluation involves determining whether the caregiver's explanation of events corresponds with the child's developmental abilities and the severity of observed findings. When the described mechanism fails to plausibly account for the injury pattern, concern for nonaccidental trauma increases. Caregiver narratives frequently lack clarity or consistency. Accounts may shift over time or omit critical details. Delayed presentation for medical evaluation without a reasonable explanation further heightens suspicion. Denial of trauma is common, even when objective findings indicate significant injury. In some cases, caregivers attribute symptoms to minor falls, choking episodes, or sudden illness without identifying a specific traumatic event. An absent or implausible history in the context of serious neurological compromise should prompt immediate consideration of inflicted injury. Historical features that raise concern include poor feeding, persistent vomiting, diminished responsiveness, or regression in developmental milestones. Loss of a previously present social smile or decreased interaction with caregivers may indicate evolving neurological dysfunction. Increased irritability, lethargy, or fluctuating mental status often precede more overt neurological signs. Episodes of apnea or seizure activity require

urgent evaluation. Hypothermia without environmental explanation may reflect central nervous system impairment. These symptoms, particularly when occurring together, warrant comprehensive assessment for intracranial pathology.

Indicators of neglect may coexist. Failure-to-thrive, downward crossing of growth percentiles, and chronic poor hygiene reflect broader caregiving deficits. Untreated diaper dermatitis, inadequate wound management, and severe dental caries suggest limited access to or engagement with healthcare services. Many affected children have prior healthcare encounters during which sentinel injuries were overlooked. Recognition of these missed opportunities is essential in preventing recurrent harm. Physical examination findings may be subtle. External signs of trauma are sometimes absent despite significant intracranial injury. Thorough inspection of the entire body is essential. Bruising in nonambulatory infants requires immediate attention. The concept often described as bruising before cruising highlights that infants who cannot independently ambulate rarely sustain accidental bruises. Any unexplained ecchymosis in this age group demands further investigation. Intraoral injuries provide additional diagnostic clues. Tears of the labial or lingual frenulum may result from forceful feeding or direct trauma. Lingual lacerations or unexplained oral bleeding should not be dismissed. Bruises distributed across multiple organ systems without a unifying accidental mechanism strengthen suspicion. Injuries in varying stages of healing indicate repetitive trauma. Patterned bruises that resemble handprints, belts, or objects provide further evidence of inflicted injury. Particular attention should focus on atypical locations such as the torso, neck, ears, and genital region. Neurological findings often reflect increased intracranial pressure or diffuse brain injury. Recurrent vomiting, apnea, seizures, and bradycardia may signal cerebral compromise. Macrocephaly or a rapidly increasing head circumference suggests expanding intracranial collections. A bulging or tense anterior fontanelle indicates elevated intracranial pressure in infants with open sutures. Scalp bruising or soft tissue swelling may accompany skull fractures. Palpation of the skull can reveal step-offs or tenderness consistent with fracture [23].

Structured clinical tools support assessment. The TEN-4-FACESp rule assists clinicians in identifying high-risk bruising patterns. Bruising on the torso, ears, or neck in children younger than four years is concerning. Additional high-risk areas include the frenulum, angle of the jaw, cheeks, eyelids, and subconjunctivae. Patterned bruising increases suspicion [24][25]. Consistent application of this rule improves detection and reduces reliance on subjective interpretation. Burn injuries may also signal abuse. Immersion burns often present with sharply demarcated borders and symmetric distribution, particularly involving the lower extremities or genital area [26]. The absence of splash marks or irregular margins suggests forced immersion rather than accidental exposure. Circular or oval bruises on the upper arms may represent grab marks and should prompt imaging to evaluate for underlying fractures. Thoracic and abdominal injuries may occur during violent squeezing or blunt impact. Rib fractures, especially posterior fractures, are strongly associated with compressive forces applied to the chest. Signs such as labored breathing, chest wall tenderness, abdominal guarding, or rigidity require further imaging to assess for pulmonary contusions or solid organ injury. These findings may coexist with intracranial pathology. Musculoskeletal injuries are common in inflicted trauma. Fractures in nonambulatory infants without a clear accidental history raise strong concern. Long bone fractures, metaphyseal corner fractures, and fractures at various healing stages indicate repetitive harm. After head injury, fractures represent the second most frequent manifestation of physical abuse in young children [27]. A comprehensive history and meticulous physical examination form the foundation of diagnosis. Recognition depends on integrating developmental assessment, historical consistency, and physical findings. Early identification enables timely intervention and reduces the risk of recurrent injury or fatal outcomes.

### **Evaluation**

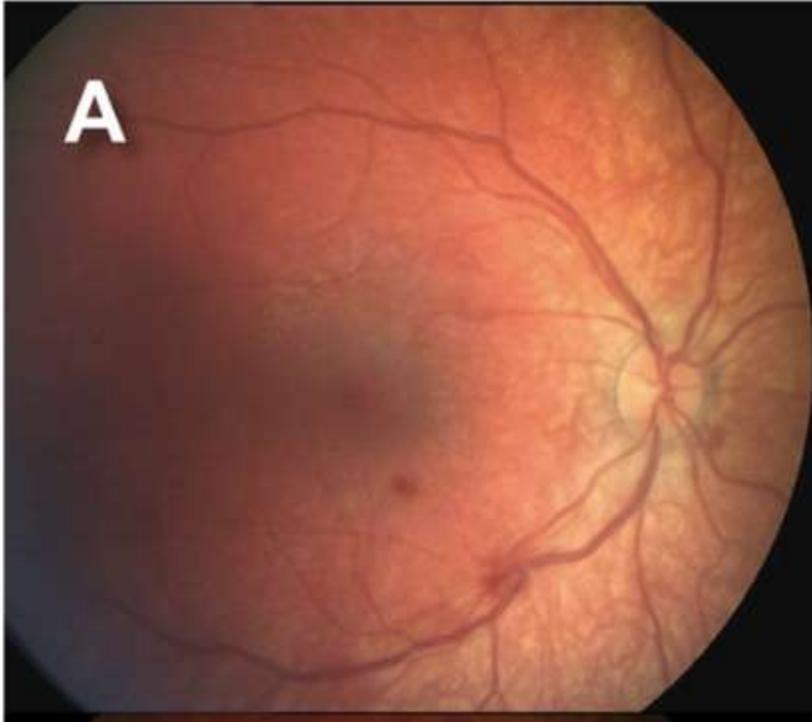
When abusive head trauma is suspected, evaluation must proceed in a structured and comprehensive manner. The goal is twofold. You must identify inflicted injury with accuracy, and you must exclude alternative medical explanations that could mimic trauma. Certain hematologic disorders, metabolic diseases, or congenital bone fragility conditions may present with bruising, bleeding, or fractures. Failure to consider these possibilities can lead to diagnostic error. At the same time, overlooking abuse places the child at continued risk. A balanced and systematic approach is essential. Many institutions have

implemented formal clinical practice guidelines for suspected abuse. These protocols standardize assessment, reduce variability in care, and promote early multidisciplinary involvement. Collaboration with child protection teams, social services, and law enforcement facilitates coordinated investigation. Before discharge, clinicians must ensure that the child's environment is safe. Structured pathways improve communication, enhance documentation, and reduce missed injuries [28]. Laboratory testing forms an important component of evaluation. A complete blood count provides information about anemia, thrombocytopenia, or infection. A comprehensive metabolic panel evaluates electrolyte disturbances, renal function, and hepatic injury. Elevated transaminases at or above 80 IU/L raise concern for occult abdominal trauma. In such cases, contrast-enhanced computed tomography of the abdomen and pelvis is indicated to assess for solid organ lacerations or hollow viscus injury. Abdominal injury may coexist with intracranial trauma and may not produce obvious external signs.



**Fig. 1:** Subdural Hematoma on Computed Tomography. The image shows a subdural hematoma (yellow arrow), indicating bleeding between the dura mater and the brain.

Measurement of troponin I should be considered when thoracic injury is suspected. Elevated levels may indicate myocardial contusion or associated chest trauma [29]. A coagulation profile is also required. Prothrombin time, partial thromboplastin time, and international normalized ratio help exclude bleeding diatheses. Although severe coagulopathies are uncommon in this context, targeted factor assays for II, VII, IX, X, XII, and XIII may be necessary when laboratory abnormalities or clinical findings suggest an underlying disorder. Consultation with hematology strengthens diagnostic accuracy. Urinalysis should assess for hematuria, which may indicate renal trauma. Abnormal creatinine or blood urea nitrogen values further support renal involvement. Neuroimaging is central to diagnosis. In acute settings, noncontrast head computed tomography is the first-line modality. It rapidly identifies skull fractures and detects intracranial hemorrhage, including subdural, subarachnoid, and epidural bleeding. Computed tomography provides immediate information that guides stabilization and neurosurgical consultation. Magnetic resonance imaging offers superior characterization of parenchymal injury. It differentiates acute from chronic subdural collections, evaluates diffuse axonal injury, detects cerebral edema, and identifies ischemic changes. MRI also clarifies ventricular compression and evolving injury patterns. Together, these modalities define both the presence and timing of injury. A skeletal survey is mandatory in children younger than two years when abuse is suspected. This imaging series includes radiographs of the skull, spine, ribs, pelvis, and long bones. The purpose is to identify occult fractures that may not produce symptoms. The American College of Radiology recommends a standardized protocol to ensure complete visualization [30][31][32][33]. Repeating the skeletal survey two to three weeks later increases sensitivity for healing fractures that were not visible initially. This follow-up imaging often reveals callus formation, confirming prior injury.



**Fig. 2:** Grade 1 Retinal Hemorrhage. Few intraretinal hemorrhages are observed in the posterior pole, confined to a limited area around the optic disc and macula.

Certain radiologic findings strongly suggest nonaccidental trauma. Multiple fractures at different stages of healing indicate repetitive injury. Posterior rib fractures are highly concerning because they often result from forceful anteroposterior chest compression. These fractures are rarely accidental in infants. Classic metaphyseal lesions, sometimes described as bucket-handle fractures, appear as curvilinear lucencies at the metaphysis and result from shearing forces near the growth plate. Spiral fractures of long bones reflect rotational mechanisms. Fractures of the scapula, vertebrae, or sternum are uncommon in accidental injury and require thorough investigation. When fracture patterns fail to correspond with the reported mechanism, suspicion increases. Ophthalmologic evaluation plays a critical role. Retinal hemorrhages are frequently observed in abusive head trauma, particularly when multilayered and extending to the peripheral retina. The distribution, depth, and number of hemorrhages assist in distinguishing inflicted injury from medical causes. While retinal hemorrhages may occur in conditions such as aneurysmal subarachnoid hemorrhage, the pattern often differs. Because retinal findings can resolve within seven to ten days, prompt examination is essential. Evaluation should be conducted by an ophthalmologist experienced in pediatric assessment. High-quality fundus photography ensures accurate documentation. The absence of retinal hemorrhage does not exclude abuse, but their presence in characteristic patterns strongly supports the diagnosis. Meticulous documentation underpins both clinical management and legal proceedings. Written descriptions must be precise and objective. Digital photographs capture injury characteristics and preserve visual evidence. Annotated body diagrams clarify location and distribution. Accurate records strengthen interdisciplinary collaboration and provide essential information for forensic review. Comprehensive evaluation demands clinical vigilance, diagnostic rigor, and coordinated teamwork. By integrating laboratory data, imaging findings, ophthalmologic assessment, and detailed documentation, clinicians can distinguish inflicted injury from medical mimics and implement protective measures that safeguard vulnerable children.

#### **Comprehensive Management Strategies in Pediatric Abusive Head Trauma**

The management of pediatric abusive head trauma requires immediate, structured, and evidence-based intervention to reduce mortality and long-term neurologic impairment. Early stabilization determines outcome. You must act without delay. The initial approach follows the trauma framework endorsed by the American College of Surgeons through the Advanced Trauma Life Support Program (ATLS) [34]. This

standardized method ensures systematic assessment and rapid correction of life-threatening conditions. The primary survey follows the ABCDE sequence. Airway protection remains the first priority, with strict cervical spine stabilization to prevent secondary spinal injury. Breathing is assessed through respiratory rate, oxygen saturation, chest wall movement, and auscultation. Circulation requires rapid evaluation of heart rate, blood pressure, capillary refill, and evidence of hemorrhage. Disability assessment focuses on neurologic status using the Glasgow Coma Scale (GCS). In preverbal children, an age-adjusted pediatric GCS improves accuracy [35]. This scale evaluates eye opening, verbal response, and motor response to quantify neurologic impairment [36]. Exposure involves complete undressing to identify occult injuries while preventing hypothermia. Clinical status can deteriorate rapidly. Repeat assessments are essential whenever changes occur. The secondary survey follows stabilization. It involves systematic inspection and palpation from head to toe. Careful evaluation of the scalp, skull, and maxillofacial region is required, with continued cervical protection. Examination then progresses to the thorax, abdomen, pelvis, and extremities to detect additional traumatic injuries. A detailed neurologic examination and further history gathering support ongoing clinical decisions.

Intracranial hypertension represents one of the most critical complications in abusive head trauma. Elevated intracranial pressure reduces cerebral perfusion and increases the risk of irreversible brain injury. Evidence-based recommendations published by the Brain Trauma Foundation in 2019 guide current practice [38][39]. Direct intracranial pressure monitoring remains supported when severe traumatic brain injury is suspected and imaging does not reliably reflect pressure dynamics. Treatment targets include maintaining intracranial pressure below 20 mm Hg and cerebral perfusion pressure between 40 and 50 mm Hg. These thresholds correlate with improved survival and neurologic outcomes. Medical management focuses on preventing secondary brain injury. Hyperosmolar therapy serves as a primary intervention. Hypertonic saline at 3 percent may be administered as intermittent boluses or continuous infusion, titrated to intracranial pressure goals. In refractory cases, higher concentration saline may be used cautiously. Seizure prophylaxis is recommended during the first seven days postinjury. Current data show no definitive superiority between levetiracetam and phenytoin. Adequate sedation reduces metabolic demand and agitation, yet large bolus doses of fentanyl or midazolam should be avoided due to risk of cerebral hypoperfusion. Controlled ventilation strategies may assist in managing refractory intracranial hypertension after the acute phase. Moderate hypothermia between 32 °C and 33 °C may offer benefit in select cases, though its application requires careful monitoring. Nutritional support plays a critical role in recovery. Early enteral feeding within seventy-two hours supports metabolic demands and may reduce complications. Immune-modulating formulas lack supporting evidence and are not recommended. Corticosteroids have shown no benefit in traumatic brain injury and should be avoided. In hemodynamically stable patients with refractory intracranial hypertension, high-dose barbiturate therapy may reduce cerebral metabolic demand.

Surgical intervention becomes necessary when medical therapy fails or when structural lesions demand correction. External ventricular drain placement allows cerebrospinal fluid diversion and intracranial pressure control. Decompressive craniectomy may be indicated for persistent, life-threatening intracranial hypertension. Skull fracture management depends on fracture type. Simple linear fractures without depression often require observation alone. Depressed or compound fractures exceeding skull thickness typically necessitate operative repair to reduce infection risk and prevent neurologic compromise [40]. Epidural hematomas may require surgical evacuation depending on size and neurologic status [41]. Subdural collections lack a universal treatment algorithm. Management decisions rely on clinical presentation and imaging findings, ranging from observation to surgical drainage or shunt placement. Effective treatment demands coordination among trauma surgeons, neurosurgeons, intensivists, and pediatric specialists. Every intervention aims to prevent secondary injury, preserve cerebral perfusion, and protect long-term neurologic function. Outcomes depend on speed, precision, and adherence to evidence-based protocols.

#### **Role of Nurses:**

Nurses play a pivotal role in the management of pediatric abusive head trauma, functioning at the intersection of acute clinical care, patient monitoring, and family support. They are often the first clinicians to identify subtle signs of abuse, including unexplained bruising, lethargy, vomiting, or changes in feeding

and behavior. Through meticulous assessment and documentation, nurses provide essential data that guide diagnostic investigations, including neuroimaging, laboratory tests, and skeletal surveys. Accurate recording of physical findings, injury patterns, and neurologic status is critical for both clinical management and potential legal proceedings. In acute care settings, nurses implement trauma protocols, ensuring airway protection, circulatory support, and neurologic monitoring, including Glasgow Coma Scale scoring. They administer medications, manage hyperosmolar therapy, provide seizure prophylaxis, and monitor responses to interventions. Nurses also coordinate care across multidisciplinary teams, communicating observations to physicians, social workers, and child protective services to facilitate timely intervention and safeguarding. Beyond clinical tasks, nurses support the psychosocial needs of the child and family, educating caregivers on safe handling practices, recognizing risk factors, and engaging in discharge planning. Their continuous bedside presence allows for early recognition of deterioration, advocacy for the child's safety, and promotion of optimal recovery and long-term outcomes.

### **Conclusion:**

Abusive head trauma remains a critical pediatric emergency with high morbidity and mortality. Its nonspecific presentation demands a high index of suspicion, meticulous assessment, and integration of clinical, radiologic, and ophthalmologic findings. Effective management relies on rapid stabilization, prevention of secondary brain injury, and adherence to evidence-based neurotrauma protocols. Long-term outcomes depend on timely intervention and coordinated multidisciplinary involvement, with nurses playing central roles in monitoring, documentation, and advocacy. Strengthening prevention programs, caregiver education, and standardized screening pathways is essential to reducing incidence, improving recognition, and safeguarding vulnerable children from devastating and preventable harm.

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### **References:**

1. Chaturvedi J, Goyal D, S R, Ruchika F, Mir MA. A Systematic Review of Pediatric Abusive Head Trauma: What a Surgeon Needs to Know Before Using a Knife. *Cureus*. 2025 Feb;17(2):e78419.
2. Elinder G, Eriksson A, Hallberg B, Lynøe N, Sundgren PM, Rosén M, Engström I, Erlandsson BE. Traumatic shaking: The role of the triad in medical investigations of suspected traumatic shaking. *Acta Paediatr*. 2018 Sep;107 Suppl 472(Suppl Suppl 472):3-23.
3. Vinchon M. Shaken baby syndrome: what certainty do we have? *Childs Nerv Syst*. 2017 Oct;33(10):1727-1733.
4. Rosén M, Lynøe N, Elinder G, Hallberg B, Sundgren P, Eriksson A. Shaken baby syndrome and the risk of losing scientific scrutiny. *Acta Paediatr*. 2017 Dec;106(12):1905-1908.
5. McNamara CR, Menchaca CI, Abel TJ, Horvat CM, Berger RP, Fink EL, Kochanek PM, Simon DW. Effectiveness of Fosphenytoin and Levetiracetam to Prevent Posttraumatic Seizures in Young Children with Accidental or Abusive Traumatic Brain Injury. *Neurocrit Care*. 2025 Apr;42(2):502-511.
6. Hung KL. Pediatric abusive head trauma. *Biomed J*. 2020 Jun;43(3):240-250.
7. Hoehn EF, Wilson PM, Riney LC, Ngo V, Bennett B, Duma E. Identification and Evaluation of Physical Abuse in Children. *Pediatr Ann*. 2018 Mar 01;47(3):e97-e101.
8. Burns J, Rohl S, Marth D, Proctor D, Amin R, Sekhon C. Which Clinical Features of Children on Initial Presentation to the Emergency Department With Head Injury Are Associated With Clinically Important Traumatic Brain Injury, Classification as Abuse, and Poor Prognosis? *Pediatr Emerg Care*. 2022 Jan 01;38(1):e254-e258.
9. Miller TR, Steinbeigle R, Lawrence BA, Peterson C, Florence C, Barr M, Barr RG. Lifetime Cost of Abusive Head Trauma at Ages 0-4, USA. *Prev Sci*. 2018 Aug;19(6):695-704.
10. KEMPE CH, SILVERMAN FN, STEELE BF, DROEGEMUELLER W, SILVER HK. The battered-child syndrome. *JAMA*. 1962 Jul 07;181:17-24.
11. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics*. 1974 Oct;54(4):396-403.

12. Narang SK, Haney S, Duhaime AC, Martin J, Binenbaum G, de Alba Campomanes AG, Barth R, Bertocci G, Care M, McGuone D., COUNCIL ON CHILD ABUSE AND NEGLECT. SECTION ON OPHTHALMOLOGY. SECTION ON RADIOLOGY. SECTION ON NEUROLOGICAL SURGERY. SOCIETY FOR PEDIATRIC RADIOLOGY. AMERICAN ASSOCIATION OF CERTIFIED ORTHOPTISTS. AMERICAN ASSOCIATION FOR PEDIATRIC OPHTHALMOLOGY AND STRABISMUS. AMERICAN ACADEMY OF OPHTHALMOLOGY. Abusive Head Trauma in Infants and Children: Technical Report. *Pediatrics*. 2025 Mar 01;155(3)
13. Barr RG, Trent RB, Cross J. Age-related incidence curve of hospitalized Shaken Baby Syndrome cases: convergent evidence for crying as a trigger to shaking. *Child Abuse Negl*. 2006 Jan;30(1):7-16.
14. Christian CW., Committee on Child Abuse and Neglect, American Academy of Pediatrics. The evaluation of suspected child physical abuse. *Pediatrics*. 2015 May;135(5):e1337-54.
15. Niederkröthaler T, Xu L, Parks SE, Sugerman DE. Descriptive factors of abusive head trauma in young children--United States, 2000-2009. *Child Abuse Negl*. 2013 Jul;37(7):446-55.
16. Bell E, Shouldice M, Levin AV. Abusive head trauma: A perpetrator confesses. *Child Abuse Negl*. 2011 Jan;35(1):74-7.
17. Esernio-Jenssen D, Tai J, Kodsi S. Abusive head trauma in children: a comparison of male and female perpetrators. *Pediatrics*. 2011 Apr;127(4):649-57.
18. Shanahan ME, Zolotor AJ, Parrish JW, Barr RG, Runyan DK. National, regional, and state abusive head trauma: application of the CDC algorithm. *Pediatrics*. 2013 Dec;132(6):e1546-53.
19. Piteau SJ, Ward MG, Barrowman NJ, Plint AC. Clinical and radiographic characteristics associated with abusive and nonabusive head trauma: a systematic review. *Pediatrics*. 2012 Aug;130(2):315-23.
20. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. *JAMA*. 2003 Aug 06;290(5):621-6.
21. Sheets LK, Leach ME, Koszewski IJ, Lessmeier AM, Nugent M, Simpson P. Sentinel injuries in infants evaluated for child physical abuse. *Pediatrics*. 2013 Apr;131(4):701-7.
22. Rangel EL, Cook BS, Bennett BL, Shebesta K, Ying J, Falcone RA. Eliminating disparity in evaluation for abuse in infants with head injury: use of a screening guideline. *J Pediatr Surg*. 2009 Jun;44(6):1229-34; discussion 1234-5.
23. Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC. Analysis of missed cases of abusive head trauma. *JAMA*. 1999 Feb 17;281(7):621-6.
24. Pierce MC, Kaczor K, Lorenz DJ, Bertocci G, Fingarson AK, Makoroff K, Berger RP, Bennett B, Magana J, Staley S, Ramaiah V, Fortin K, Currie M, Herman BE, Herr S, Hymel KP, Jenny C, Sheehan K, Zuckerbraun N, Hickey S, Meyers G, Leventhal JM. Validation of a Clinical Decision Rule to Predict Abuse in Young Children Based on Bruising Characteristics. *JAMA Netw Open*. 2021 Apr 01;4(4):e215832.
25. Thackeray JD. Frenet's tears and abusive head injury: a cautionary tale. *Pediatr Emerg Care*. 2007 Oct;23(10):735-7.
26. Maguire S, Moynihan S, Mann M, Potokar T, Kemp AM. A systematic review of the features that indicate intentional scalds in children. *Burns*. 2008 Dec;34(8):1072-81.
27. Raynor E, Konala P, Freemont A. The detection of significant fractures in suspected infant abuse. *J Forensic Leg Med*. 2018 Nov;60:9-14.
28. Higginbotham N, Lawson KA, Gettig K, Roth J, Hopper E, Higginbotham E, George TM, Maxson T, Edwards G, Garcia NM. Utility of a child abuse screening guideline in an urban pediatric emergency department. *J Trauma Acute Care Surg*. 2014 Mar;76(3):871-7.
29. Riney LC, Frey TM, Fain ET, Duma EM, Bennett BL, Murtagh Kurowski E. Standardizing the Evaluation of Nonaccidental Trauma in a Large Pediatric Emergency Department. *Pediatrics*. 2018 Jan;141(1)
30. Bradford R, Choudhary AK, Dias MS. Serial neuroimaging in infants with abusive head trauma: timing abusive injuries. *J Neurosurg Pediatr*. 2013 Aug;12(2):110-9.
31. Kleinman PK, Nimkin K, Spevak MR, Rayder SM, Madansky DL, Shelton YA, Patterson MM. Follow-up skeletal surveys in suspected child abuse. *AJR Am J Roentgenol*. 1996 Oct;167(4):893-6.

32. Zimmerman S, Makoroff K, Care M, Thomas A, Shapiro R. Utility of follow-up skeletal surveys in suspected child physical abuse evaluations. *Child Abuse Negl.* 2005 Oct;29(10):1075-83.
33. Bennett BL, Chua MS, Care M, Kachelmeyer A, Mahabee-Gittens M. Retrospective review to determine the utility of follow-up skeletal surveys in child abuse evaluations when the initial skeletal survey is normal. *BMC Res Notes.* 2011 Sep 12;4:354.
34. Galvagno SM, Nahmias JT, Young DA. Advanced Trauma Life Support® Update 2019: Management and Applications for Adults and Special Populations. *Anesthesiol Clin.* 2019 Mar;37(1):13-32.
35. Borgialli DA, Mahajan P, Hoyle JD, Powell EC, Nadel FM, Tunik MG, Foerster A, Dong L, Miskin M, Dayan PS, Holmes JF, Kuppermann N., Pediatric Emergency Care Applied Research Network (PECARN). Performance of the Pediatric Glasgow Coma Scale Score in the Evaluation of Children With Blunt Head Trauma. *Acad Emerg Med.* 2016 Aug;23(8):878-84.
36. Jain S, Margetis K, Iverson LM. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jun 23, 2025. Glasgow Coma Scale.
37. Kjaergård H, Nordkild P. [Bilateral compartment syndrome in a drug addict]. *Ugeskr Laeger.* 1985 Mar 11;147(11):959-60.
38. Kochanek PM, Tasker RC, Carney N, Totten AM, Adelson PD, Selden NR, Davis-O'Reilly C, Hart EL, Bell MJ, Bratton SL, Grant GA, Kissoon N, Reuter-Rice KE, Vavilala MS, Wainwright MS. Guidelines for the Management of Pediatric Severe Traumatic Brain Injury, Third Edition: Update of the Brain Trauma Foundation Guidelines, Executive Summary. *Pediatr Crit Care Med.* 2019 Mar;20(3):280-289.
39. Kochanek PM, Tasker RC, Bell MJ, Adelson PD, Carney N, Vavilala MS, Selden NR, Bratton SL, Grant GA, Kissoon N, Reuter-Rice KE, Wainwright MS. Management of Pediatric Severe Traumatic Brain Injury: 2019 Consensus and Guidelines-Based Algorithm for First and Second Tier Therapies. *Pediatr Crit Care Med.* 2019 Mar;20(3):269-279.
40. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, Servadei F, Walters BC, Wilberger J., Surgical Management of Traumatic Brain Injury Author Group. Surgical management of depressed cranial fractures. *Neurosurgery.* 2006 Mar;58(3 Suppl):S56-60; discussion Si-iv.
41. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, Servadei F, Walters BC, Wilberger JE., Surgical Management of Traumatic Brain Injury Author Group. Surgical management of acute epidural hematomas. *Neurosurgery.* 2006 Mar;58(3 Suppl):S7-15; discussion Si-iv.