Head Trauma in the Pediatric Population

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10.1 Introduction

Traumatic Brain Injury (TBI) among young patients may lead to detrimental neurological and cognitive deficits, causing an everlasting burden. Mechanisms of TBI alter depending on age category [1], and vary from domestic abuse, and falls, to motor vehicle accidents. TBI Pathology also differs depending on age groups. The younger the child is, the more likely he will endure a subdural hematoma (SDH) and diffuse brain edema compared to contusions and diffuse axonal injury among adolescents [2]. Examining the toddlers and small-aged category is further a difficult task. The clinical presentation is variable depending on the mechanism of trauma, and the pediatric Glasgow Coma Scale is mainly used as an assessment tool [3]. Special care to details should be given to the pediatric population; even scalp bleeders can be a source of significant blood loss and hemorrhagic shock in infants.

Initial emergency management always entails several measures including avoiding hypotension, running primary surveys to rule out another organ injury, head elevation to allow proper jugular venous drainage, and careful manipulation of the cervical spine [4]. In addition, interviewing witnesses and family members is important for understanding the mechanism of injury.

The pediatric skull is different from the adult bone. It has higher plasticity properties, hence leading to better absorptive forces and clinical manifestations [5]. The presence of open sutures in infants can avert an increase in ICP. However, these open suture lines themselves are a risk for growing skull fractures [6]. With respect to skull fractures, linear fractures are more observed over the parietal area, followed by the occipital, frontal, and temporal bones [7]. The presence of extracranial subcutaneous swelling is an indirect diagnosis of such fractures [8].

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When it comes to the site of brain injury, head-to-body volume or head-to-torso ratio in the pediatric population is higher than in the adult counterpart [9]. This puts the pediatric population at higher risk of head injury [10] due to the bigger forehead protuberance and a higher risk of frontal bone trauma, and frontal lobe contusions.

In the setting of significant head injury, primary objectives in management include maintaining adequate oxygen delivery and proper cerebral perfusion pressure, as well as reduction of intracranial pressure (ICP). In general, ICP in adults ranges normally between 5 and 15 mmHg, however, these values can vary depending on patient age, such as 2–7 mmHg in infants and young children [11].

By and large, the treatment includes two tiers. The first tier approach entails medical management, along with sedation or anesthetics to decrease the metabolic brain demands. This is usually accompanied by ICP monitoring. In case of failure of these strategies, hyperosmolar agents along with controlled ventilation are implemented to decrease the ICP. In the event of medical treatment resistance, escalation to second-tier therapies such as decompressive craniotomies can be used as protective measures to decrease the ICP.

10.2 Mechanisms of Injury

Falls are the most common mechanism in children suffering a head injury, followed by motor vehicle crashes, pedestrian and bicycle accidents, assaults, sports-related trauma, abuse, and projectiles in war conflict areas [12, 13]. These mechanisms cause isolated head trauma in the majority of patients [12]. Infants in general endure more falls and are at increased risk for inflicted injury. Moreover, children who remain in the care of the perpetrator are at significant risk of being injured again.

10.3 Imaging

The aim of neuroimaging is to acquire a quick and sensitive diagnostic assessment of injury extent. A Skull X-ray is not of any value anymore in the presence of other imaging modalities. It only depicts skull fractures but gives no hint regarding the brain parenchyma and extra-axial presence of blood collections. Ultrasound (US) is a bedside modality that is radiation-free and considered a cheap technique. It can accurately diagnose pediatric skull fractures when compared to CT scans, nonetheless, its use is restricted to the neonatal period only [14]. CT scan correlates well with the clinical presentation [15] and is considered the gold standard for TBI diagnosis [16]. MRI is mainly used in the acute stage after head injury in stable patients when CT findings do not explain the neurological symptoms and in the subacute/chronic phases. However, it cannot be utilized in case of foreign bodies penetration, has a longer acquisition time when compared to CT and US, and got a lower sensitivity rate to detect bone fractures and fragments.

10.4 Treatment: Medical

Most cases of mild to moderate TBI may be managed conservatively by simple but important measures. Head elevation is crucial in reducing traumatic congestion of the brain and is imperative in addition to neurologic observation and avoidance of excessive hypotonic fluids. Intracranial pressure (ICP) monitoring is not recommended for most of these cases, but rather in the setting of severe head injury when the Glasgow Coma scale (GCS) is less than 8.

10.5 ICP Monitoring

The intraventricular catheter is the gold standard for ICP measurement. It offers both monitoring and the ability for CSF drainage [17]. The implication of CSF drainage via ventriculostomy or lumbar drain is not well investigated in the literature and is considered level III evidence [18]. Unfortunately, the placement of such catheters is impossible in cases of severe edema and collapsed ventricles.

Intraparenchymal intracranial pressure sensors or intraparenchymal probes can be used as equally sensitive methods for ICP measurement [19]. They were introduced for the first time in the Brain Trauma Foundation Guidelines for TBI in children in 2003 [20, 21]. The main indication was early detection of high ICP among patients with severe TBI, patients with traumatic mass lesions, or in whom the serial neurologic examination is precluded by sedation, neuromuscular blockade, or anesthesia [22]. The presence of an open fontanel and/or sutures or a normal CT scan in an infant with severe TBI does not prevent the development of high ICP or exclude the use of intracranial pressure monitoring. However, ICP monitoring is not regularly suggested in infants/children with mild or moderate head injury [23].

10.6 Cerebral Perfusion Pressure

Although Cerebral Perfusion Pressure (CPP) is currently mentioned in the adult TBI guidelines [24], no large studies have been conducted for the pediatric population. Despite the lack of level I and II evidence regarding the relationship between cerebral perfusion pressure (CPP) and clinical outcome, it is still highly recommended in intensive care unit medical practices to manage both ICP and CPP in pediatric patients with severe TBI [21]. A study conducted by Chambers et al. on a sample of 235 TBI pediatric patients found a correlation between CPP and clinical outcome, after age-stratified critical levels of CPP, among age groups of 2–6, 7–10, and 11–16 years, CPP values of 43, 54, and 58 mmHg, respectively, were associated with good outcomes [25].

10.7 Temperature Control

Hyperthermia should be avoided in order to decrease the brain's metabolic demands, lipid peroxidation, the increase in parenchymal inflammation, and more importantly, to decrease seizure incidence. Hence the importance of a hypothermic state induction, as part of the intracranial hypertension treatment modality, that is considered level II evidence. However, no clear benefit was noted regarding the outcome at 6 months post-severe TBI in the pediatric population [26].

10.8 Hyperosmolar Therapy

Hyperosmolar therapy is the standard treatment to reduce ICP in severe TBI adult patients. Hypertonic saline (HS) and mannitol are the most widely used agents. Common HS solutions are 3%,5%, and 7.5% [27]. There is a difference in the suggested mechanism between mannitol and HS. Mannitol has both a rheological and a vasoconstrictive effect, both of which help in the decrease of ICP [28]. On the other hand, HS acts by creating an osmotic gradient, which is a shift from the intracellular space to the interstitial volume [29]. Despite that, both arms are commonly used in the daily practice among pediatric TBI, where level II and III evidence recommend the use of HS 3% boluses in pediatric patients with high ICP, due to the lack of sufficient data and papers on the use of mannitol [30].

10.9 Anticonvulsants

The pediatric population has lower seizure thresholds when compared to their adult counterparts [31]. What makes the situation worse, is the subtle nature of the seizures, making their detection more difficult to spot [32]. Post-traumatic seizures can be labeled as early seizures when they occur within 7 days of the insult, and late whenever occurrence is after 1 week of the trauma. It is considered a good idea to prevent such seizures, however, the side effects of antiepileptic drugs have an undesirable impact (impaired learning, behavioral changes, skin rash, hematological abnormalities, ataxia, Stevens-Johnson syndrome) and should be avoided [33]. When it comes to early post-traumatic seizures, some data show a benefit in starting antiepileptic drugs (AED) early on especially when GCS is <8. Moreover, level II and III evidence indicates that prophylactic anticonvulsant therapy after 7 days from TBI, did not prevent later post-traumatic seizures [34]. Moreover, no specific guidelines have been approved about the duration of the AED [35, 36]. Further studies should be carried out to assess the outcome in terms of mortality and the effect of the anticonvulsant therapy on different age categories.

10.10 Corticosteroids

The use of steroids in children with traumatic brain injury lacks literature evidence and might inflict harm such as infectious complications, and suppression of endogenous cortisol levels. In addition, its use was not associated with improved outcomes, nor reduction of ICP or mortality rate [37].

10.11 Treatment: Surgical-Decompressive Craniectomy

During trauma, physiologic equilibrium is breached and compensatory mechanisms operate to keep ICP constant [38]. This autoregulation may be disturbed in severe TBI, causing neurological deterioration and brain herniation. A normal ICP in adults is below 15 mmHg. When it reaches a level above 20 mmHg in TBI patients, it is considered pathological and appropriate adjustment is necessary. Once ICP is resistant to medical interventions, second-tier treatment modalities may become necessary.

Surgery is an option whenever ICP is refractory, and brain edema is not resolving. Kocher [39] and Cushing [40] were the first to use Decompressive surgery (DC) as a tool to decrease ICP in TBI cases. It provides a way to physically create more space for the brain allowing it to relax, helps in evacuating the hematomas, and decreases the risk of herniation. Consequently, cerebral blood flow and cerebral perfusion pressure will increase, improving brain tissue oxygenation.

Currently, the evidence describing the benefits of DC in pediatric TBI patients is limited; mostly level II and III evidence [21]. Different techniques have been described for decompressive craniectomies including bifrontal craniectomies, bitemporal, wide unilateral, and bilateral frontotemporoparietal craniectomy [41–43].

For the frontotemporoparietal craniectomy, the patient is placed in a supine position, with the head turned to the contralateral side. A reverse question mark incision is created from the root of the zygoma, around 1 cm in front of the tragus, going around the back of the ear helix reaching approximately 2 cm lateral to the midline (to prevent damage to the superior sagittal sinus) and anterior to the coronal suture. The bone flap should be extended inferiorly toward the floor of the temporal fossa [44]. Insufficient bony decompression will cause mushrooming of the brain parenchyma, and further damage to the brain cortex, and cortical veins engorgement [45]. The bifrontal craniectomy technique is mainly applied in the case of frontal lobe contusions and can be used for generalized cerebral edema. The incision is created from the zygomatic area bilaterally, to around 2 cm from the coronal sutures posteriorly. The craniotomy flap extends to the orbital roofs anteriorly. The superior sagittal sinus will need to be sacrificed anteriorly, by ligating it with stitches and cutting it with scissors. The falx should be also sectioned. This step will allow better brain expansion and prevents the brain from herniating against a tight dural edge. However, surgery comes with a risk of complications such as hydrocephalus,

subdural hygroma formation, new hematoma formation or hematoma progression, and wound infection [46].

10.12 Prevention, Future Considerations, and Conclusion

Preventive methods and anticipatory guidance are the most important measures against TBI. Law implementation, such as seat belts, car seats, and helmet use [47]. Education regarding seat use, with correct position, never in the front but in the back seat, and facing the rear of the car, use of head helmets during bicycling, skating, rollerblading, skiing, and practicing martial arts fights is of utmost importance. Future research should be conducted in order to stratify what is normal ICP according to age category. Although randomized controlled trials continue to be the gold standard to provide level I evidence for TBI, we are aware of the lack of sufficient understanding of many basic aspects of TBI which limits the standardization of TBI management [48].

10.13 Case Illustration

A 15-year-old male, previously healthy, endured a motor vehicle accident 24 hours prior to presentation. The patient was on ATV when he was hit by a car. He was not wearing a helmet. As per the family, he lost consciousness and was vomiting before being transferred to another hospital where he was rushed to the operating room for a right-sided decompressive craniectomy.

Upon presentation in the emergency department, he was agitated, moving all four extremities, and the pupils were equal and sluggishly reactive at 3 mm before being fully sedated. After admission to the Pediatric ICU, we recommended head of the bed elevation at 30 degrees, to keep sodium in the range of 145–150 by administering 3% hypertonic saline, PC02 in the range of 28–32, and a possibility of an ICP monitor placement based on the CT scan results.

A CT scan showed a left temporal hemorrhagic contusion with a neighboring subdural hematoma, measuring 18 mm in maximum diameter with associated surrounding vasogenic edema and effacement of the adjacent sulci and mass effect on the left hemisphere and left lateral ventricle with a 5-mm shift of the midline structures to the right. This was associated with left uncal herniation, and a hypodensity in the left cerebral peduncle and the genu of the left internal capsule as well as in the medial aspect of the right thalamus that may represent diffuse axonal injury (Fig. 10.1a, b). He was rushed to surgery for a left-sided decompressive craniectomy (Fig. 10.2). After the surgery, a Camino ICP monitor (Integra Life Sciences) was inserted denoting a normal ICP level ranging between 12 and 16 mmHg. One month after the bilateral surgeries, a repeat CT brain showed bilateral subdural hygromas which resolved spontaneously (Fig. 10.3). Two months post-bilateral decompressive surgeries, the patient underwent bilateral cranioplasties and replacement of the bone flaps (Fig. 10.4a, b). Four months post-TBI, the patient is

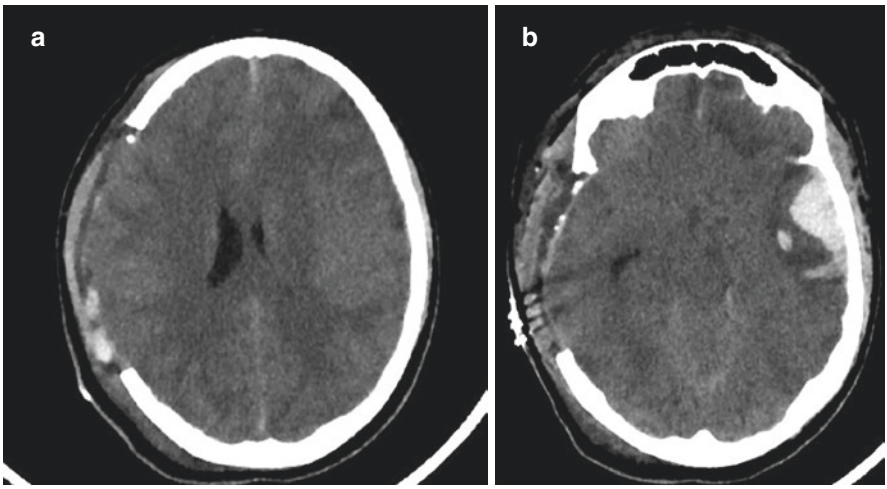

Fig. 10.1 (**a**) CT scan of the upper section of the brain showing right decompressive craniectomy, and left side edema with mass effect on the lateral ventricle. (**b**) lower CT scan cut showing left subdural hematoma and contusions with significant edema and effacement of basal cisterns

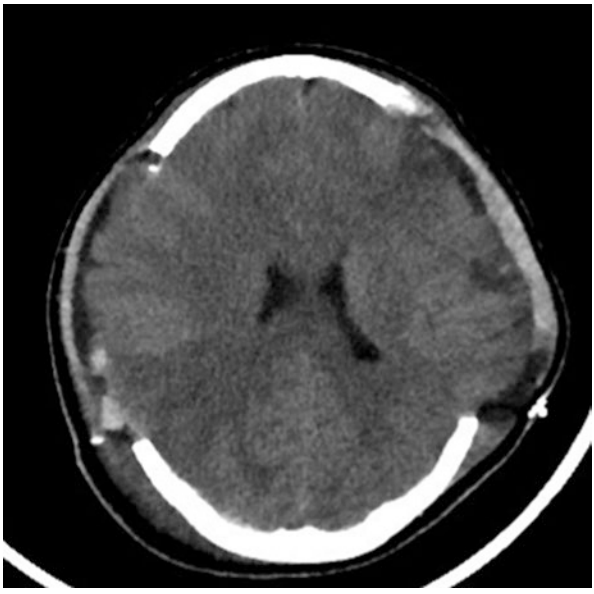

Fig. 10.2 CT scan section post-bilateral decompressive craniectomies with some bulging of the brain through the bony defects, but less cerebral edema

wheelchair bound, still on an NG tube, but able to swallow food, and the tracheostomy was reduced and closed. He wears a diaper, speaks a few words with repetitive anxious behavior, and still suffers from right spastic hemiparesis.

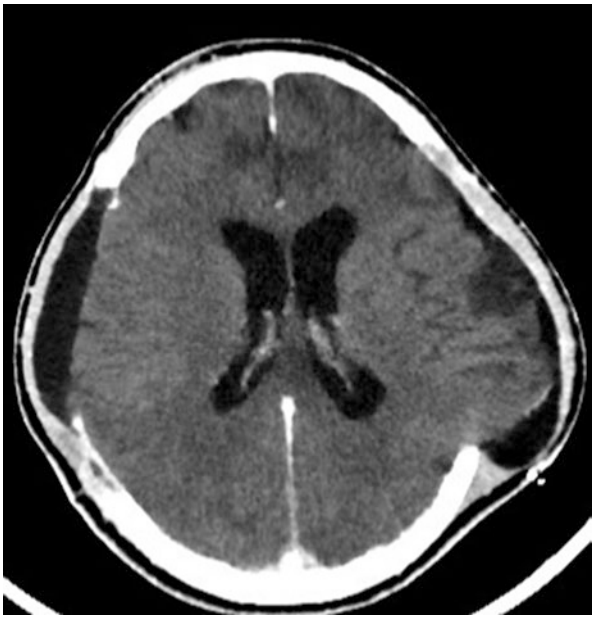

Fig. 10.3 CT scan at 1-month follow-up after craniectomies showing widening ventricles and bilateral subdural hygromas. The edema is markedly reduced

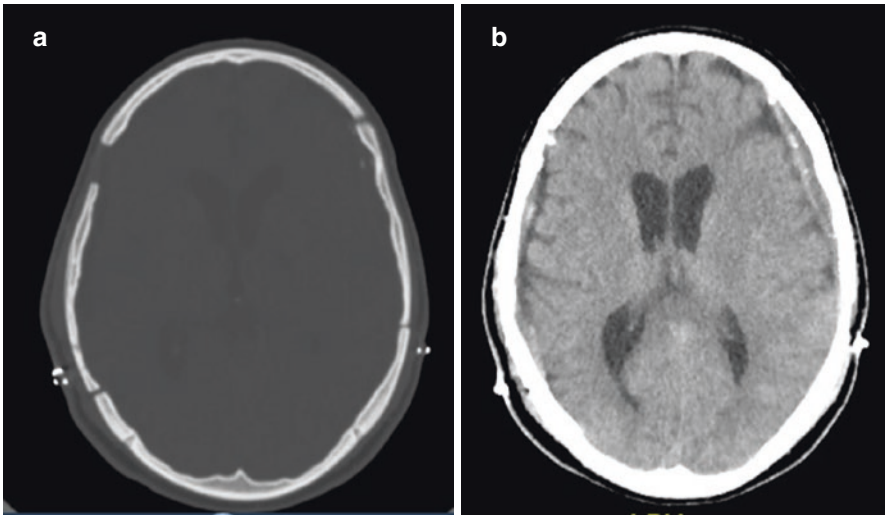

Fig. 10.4 CT scan at 2 months follows up. (**a**) Bone window setting showing replacement of bilateral bone flaps. (**b**) Brain setting showing resolved edema, and minor subdural hygromas

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