Head Injury

10

Bindey Kumar, Anil Kumar, and Arun Prasad

10.1 Introduction

Head injury is one of the leading causes of death in the pediatric population with a mortality rate of 0.5 per 1000 children reporting to emergency rooms according to one Canadian review. However most head injuries are mild in nature and have normal recovery without sequelae with only <1% that requires neurosurgical intervention.

Head injuries in children are perhaps more significant than equivalents in adults because:

- Relative ↑ head size,
- ↓ thickness cranial bones
- Unstable gait and reduced control of neck (toddlers)
- ↓ subarachnoid space, providing less buoyancy.

Age Variation

- Infants
  - Non-accidental injury

B. Kumar (✉)
Department of Paediatric Surgery, All India Institute of Medical Sciences, Patna, Bihar, India

A. Kumar
Trauma and Emergency and General Surgery, All India Institute of Medical Sciences, Patna, Bihar, India

A. Prasad
Trauma and Emergency and Paediatrics, All India Institute of Medical Sciences, Patna, Bihar, India

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- Preschool age
  - Falls
- Adolescence
  - Road traffic accident, recreational activities, and violence by assaults and firearms
  - ↑↑ male teenagers

10.2 Classification and Pathophysiology

- Fractures
  - Linear
  - Stellate
  - Depressed
- Base of skull ± CSF leak
  - Periorbital hematoma (Battle's sign)
  - Retroauricular hematoma (Raccoon eyes)
  - hemotympanum
- Intracranial hemorrhage can be classified as:
  - Extradural (arterial)
    "talk and die," classically this is due to laceration of the middle meningeal artery by a temporal bone fracture.
  - Subdural (venous)
    The hematoma covers the entire hemisphere.
  - Intracerebral
- Diffuse Brain Injury
  - Mild concussion
    Confusion ± amnesia
  - Classic Concussion
    Loss of consciousness (LoC)
  - Diffuse Axonal Injury
    LoC, coma, autonomic dysfunction (e.g., labile blood pressure).

Acceleration–deceleration of the pediatric brain inside the skull leads to mechanical trauma to the brain focally (*coup*) and at the opposite pole (*countercoup*) giving rise to focal injuries.

Following the **primary brain injury** there is release of inflammatory mediators such as the amino acids glutamate and aspartate that can cause neuronal cell death by apoptosis. This is followed by cerebral edema that can be further aggravated by poor cerebral perfusion due to hypoxia, hypotension, and hyperthermia leading to an extensive **secondary brain injury.** Active treatment of primary brain injury (e.g., avoidance of hypoxia, acidosis, and appropriate fluid management) may minimize this **secondary brain injury.**

Monro–Kellie Doctrine
(Volume of intracranial content is fixed in a rigid box.)
An expansile mass can only move out the liquid component (e.g., venous blood, CSF).
Compensated: → Intracranial pressure (ICP)
Uncompensated: ↑↑ ICP
Cerebral Perfusion Pressure (CPP) = Mean Blood Pressure – ICP
Normal ICP ~ 10 mmHg (=13.6 cmH_2O)

Hematoma and cerebral edema reduce intracranial content leading to net outflow of blood and CSF and a rising ICP and falling cerebral perfusion. Ultimately, brain herniation occurs through the foramen magnum.

10.3 Clinical Assessment

Repeated neurological assessments using the **Glasgow Coma Scale (GCS)** may differentiate traumatic brain injury from clinically important traumatic brain injury (ciTBI) (Table 10.1).

In addition to mode of injury and presence of LoC, it is important to be aware of persistent vomiting, abnormal behavior, headache, and any seizures. Complete neurological examination including assessment of the cervical spine should be repeated frequently to assess changing status. The pupils should be assessed for their reaction to light and whether they match the other side.

Table 10.1 Glasgow coma scale

	Child	Infant	Score
Eye(s) opening	Spontaneous	Spontaneous	4
	To Speech	To speech	3
	To Pain	To pain	2
	No response	No response	1
Verbal response	Oriented, appropriate	Coos and babbles	5
	Confused, inappropriate	Irritable cries	4
	Inappropriate words	Cries to pain	3
	Inappropriate sounds	Moans to pain	2
	No response	No response	1
Best motor response	Obeys commands	Spontaneous, purposeful	6
	Localizes pain	Withdrawal from touch	5
	Withdrawal from pain	Withdrawal from pain	4
	Flexion to pain	Abnormal flexion to pain	3
	Extension to pain	Abnormal extension to pain	2
	No response	No response	1

10.3.1 S-100B Protein

This may be a specific marker of brain parenchymal injury. It is found in glial cells and released into circulation after head injury. Its best use might be to identify those children with mild injury to avoid a CT Scan head. The serum level above which S - 100 B protein becomes pathological is more than 0.105 mg/L.

10.3.2 Imaging

- Plain skull X-rays
  - Have a limited role in evaluating head injury although it may show extent of a skull fracture.
  - Skeletal growth centers in the skull and spine can resemble fractures, hence should be interpreted carefully.
- Cervical spine imaging
  - Both AP and lateral views.
  - Severe head injury may be associated with a neck injury in 10% of cases.
  - **Spinal cord injury without radiographic abnormality (SCIWORA)** may be seen in two-third of children who had spinal cord injury.
- CT scan, may be indicated in:
  - Penetrating or depressed skull fracture.
  - Seizure.
  - Persistent vomiting.
  - Retrograde amnesia of more than half an hour duration.
  - Localized neurological features.
  - Base of skull fracture.
  - GCS scale <13 at any point since the injury.
  - Children under 2 years
    GCS = 14 with altered level of consciousness or behavior or basilar skull fracture or loss of consciousness for ≥5 s.
  - Children above 2 years of age
    GCS = 14 with altered behavior or basilar skull fracture, history of vomiting, severe headache, or severe mechanism of injury (Figs. 10.1 and 10.2).

Severity Assessment and Management

- Always begin with an assessment of **Airway, Breathing, and Circulation**.
- After ensuring a clear airway, protect the cervical spine.
- The pediatric **Glasgow Coma Scale (GCS) for under 2 years of age** or **Glasgow Coma Scale Score** still forms the basis for defining the severity of head injury.
  - GCS 13–15: Mild Head Injury.
  - GCS 9–12: Moderate Head Injury
  - GCS 3–8: Severe Head Injury.

The goal of management is to treat the primary brain injury and prevent or minimize secondary brain injury.

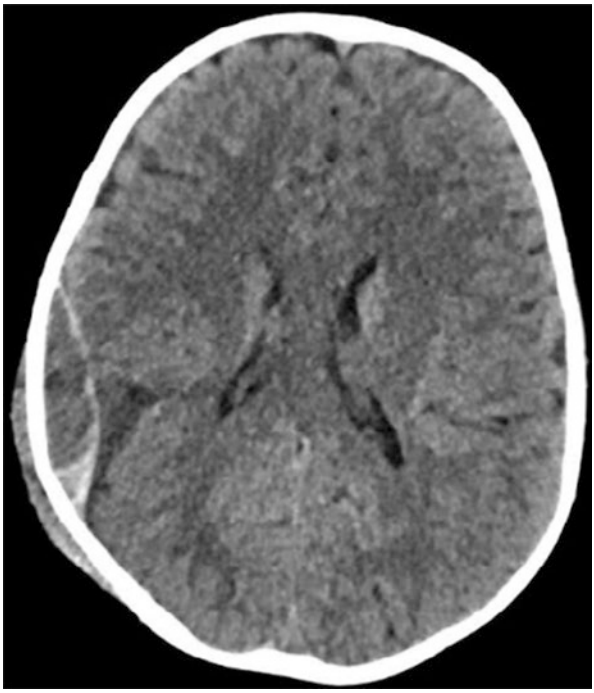

Fig. 10.1 A CT scan is showing rt parieto-temporal subacute extra dural hematoma after head injury

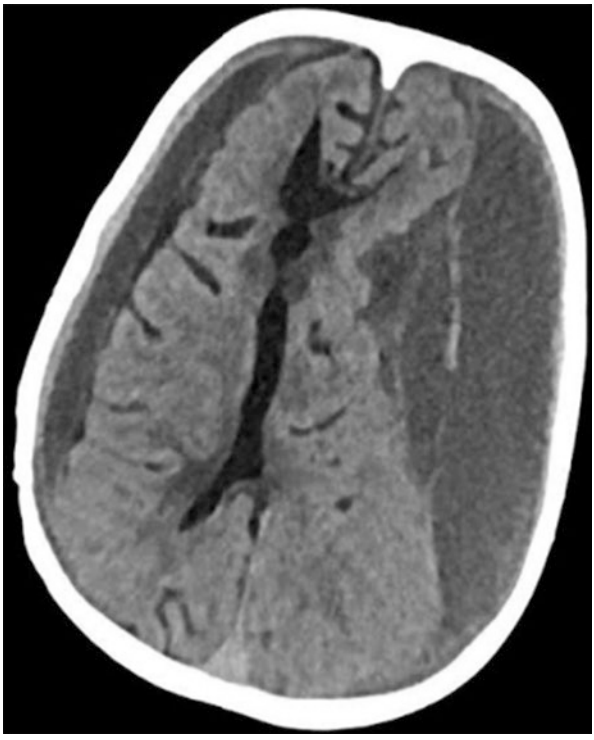

Fig. 10.2 A CT scan is showing bilateral subdural hematoma after head injury

The basic principle of management of the head injury is based on measures to ensure adequate cerebral blood flow and perfusion to ensure proper oxygenation of brain tissue, bearing in mind the Monro–Kellie doctrine.[1]

Proper oxygenation of the brain may be achieved by giving **assisted ventilation** in those who have severe head injury (GCS score < 8), mid-facial trauma, or with evidence of hypoxia. There is no advantage of endotracheal intubation over bag-mask ventilation.

Hemostasis should be achieved by medical or surgical means and is mostly extracranial. Adequate crystalloid solution should be given to maintain a normal blood pressure and ensure cerebral blood flow. Hypotonic fluids should be avoided for fluid resuscitation or maintenance fluids and normal saline or Ringer's lactate solution should be used instead. Blood products should be transfused to restore intravascular volume, if needed.

Measures to reduce intracranial pressure includes:

- Hyperosmolar therapy
  - 20% mannitol solution
  - Hypertonic saline
- Hyperventilation
  - Aim for a $PaCO_2$ ~ 35 mm Hg (=4.7 KPa).
  - Hyperventilation should be considered only for a limited period.
- Sedation (barbiturates),
- CSF drainage
  - Lumbar puncture or via a fontanelle.
- **Decompressive surgery**
  - Includes evacuation of expanding hematoma by frontal craniectomy.

Look for physical signs of raised intracranial pressure:

- Cushing[2] reflex
  - ↑systolic blood pressure, ↓ heart rate and ↓ respiratory rate may give warning of impending brain herniation.
- Ipsilateral dilated fixed pupil may show third (Oculomotor) nerve compression especially of the ipsilateral parasympathetic fibers.

Intracranial pressure monitoring plays an important role in management of cerebral edema and prevention of brain herniation.

Seizures should be controlled by anticonvulsant such as phenytoin, but prophylactic anticonvulsants are not recommended.

[1] Alexander Monro Secundus (1733–1817) and George Kellie (1770–1829). Both from Edinburgh with the latter a pupil of the former.

[2] Harvey Williams Cushing (1869–1939) Archetypal American neurosurgeon working in Boston and Yale.

10.4 Prevention of Head Injuries in Children

Head injuries can be prevented in children by the following measures-

- Installing window guards to prevent falls out of open windows
- Using safety gates at the top and bottom of stairs
- Keeping stairs clear of clutter
- Using a nonslip mat in the bathtub or shower floor
- Using playgrounds with shock-absorbing materials on the ground.
- Using a seat belt.

Further Reading

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