Management of pediatric head injury

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Introduction

Exact incidence of head injuries in India is not available, but in the USA incidence is 200 cases/10000 population. Children younger than 4 years of age have been reported to have poor prognosis similar to adults while better outcomes have been reported in 5 to 15 year age group.¹

Open fontanelle and sutures predispose a child to a higher incidence of subdural hematoma (SDH). A possible explanation may be that in an skull that can expand, clinical significant volume of blood can easily gets accumulated and, absence of arachnoid adhesions and pacchioniann granulation's predispose to tearing of bridging veins and accumulation of blood along the vertex.²

There is characteristic pathognomical brain injury suffered by infants < 3 months of age due to lack of glial tissue, increased brain matter and movable squamosal bones leading to tears of cerebral cortex, mesencephalic dysfunction from cerebral herniation, tears of subcortical white matter and orbito-frontal and temporal lobes.

Older infants suffer from tears at pontomedullary junction, petechial hemorrhages and cellular necrosis. By one year of age sutures are closed and skull becomes rigid. Glial cell proliferation, synaptic connections, and dendrites arborization progress logarithmically till two years of age. Myelinization begins at first year and progress till 10th year of life.³ Hence contusions, subarachnoid and extradural (epidural) hematoma, and axonal injuries are commonly seen. Diffuse axonal injury is a common cause of persistent coma despite absence of cerebral edema.

Pathogenesis of brain injury

After severe traumatic brain injury reduction in cerebral blood flow (CBF) begins almost immediately after injury lasting as long as 24 hours. This happens due to neurochemically mediated vasospasm, astrocyte swelling with compression of the microcirculation. This early hypoperfusion with normal metabolic requirement is a high risk setting and any associated hypotension or hypoxia leads to further hypoxic ischemic injury to the brain. As the injury evolves, blood brain barrier disruption occurs, vasogenic edema occurs. In addition cytotoxic edema is a key factor to secondary cerebral swelling. Enlarging hematoma also contributes to decreased cerebral perfusion pressure (CPP) by increasing the intracranial volume and intracranial pressure (ICP) and decreasing cerebral blood flow. Loss of cerebral autoregulation occurs frequently. Normal cerebral blood flow regulation in response to changes in blood pressure and cerebral vascular tone is absent. Eventually cerebral edema progressively increase the ICP further reducing the CPP (mean arterial pressure (MAP)—intracranial pressure). Aldrich et al⁴ reported that incidence of diffuse cerebral swelling was 41% in children as compared to 26% frequency in adults, possibly due to a hyperemic response (luxury perfusion). Lang et al⁵ reported incidence of diffuse cerebral swelling at 4 to 5 times that of adults.

Clinical evaluation and management in emergency

In any child with multiple trauma, a quick primary and secondary survey is performed with prompt attention to airway, breathing and circulation (see chapter on multiple trauma). Pediatric patient with head injury may be brought unconscious, posturing (decerebrate or decor-
ticate), or actively convulsing. All patients should be presumed to be full stomach and oxygen therapy should be initiated. Comatose patients need to be intubated with rapid sequence intubation technique, with due attention to cervical spine stabilization. Jaw thrust maneuver can be performed during bag mask ventilation. Head tilt and chin lift maneuvers should be avoided. A cervical spine collar should be placed until cervical spine X-rays are obtained to rule out a fracture or dislocation.

Glasgow coma scale (GCS): For clinical evaluation, as much as possible, GCS for adults should not be used considering the anatomical, physiological and developmental differences of pediatric age group, a modified GCS is available (Table 1).

Indices of good outcome

Single most reliable examination for evaluating outcome in children less than 3 years of age is oculomotor examination, as oculomotor functions are fully developed by two months of age, while cortico spinal myelination and optic pathway myelination develop much later. Child with open fontanel and ocular score of 3-4 generally has good outcome. Similarly children with motor score of 4 and closed fontanel will have good outcome. Those with closed fontanel and verbal score of 3 have good outcome.

Indices of poor outcome

Evidence of retinal hemorrhage indicates poor outcome.

Radiographic evidence of post-traumatic splitting of suture indicates poor outcome and high incidence of seizures. A bilateral linear skull fracture correlates with poor outcome. Incidence of post-traumatic seizure is 10% in children with depressed fracture and 7% without depressed fracture. Occurrence of generalized tonic clonic seizure indicates poor outcome to the extent of 27 to 66%.

Irrespective of closed or open fontanel, children with motor neurological deficit like hemiparesis have poor outcome in about 25% of cases.

The child has suffered a severe head injury irrespective of child coma score, if any of the following is present:
1. Unequal pupils (a difference of 1 mm)
2. Asymmetric motor examination
3. Open head injury with CSF leak or exposed brain
4. Neurological deterioration (GCS decreased by 2 points)
5. Depressed skull fracture

External examination

Scalp: subgaleal/subperiosteal haematoma should be looked for Fontanel: To be examined preferably in upright position when child is quiet.

Cranial Suture: Metopic, coronal, lambdoid sutures need to be examined for suture width (normally no palpable gap, except for sagittal suture), for mobility, and overlap (normally no overlap is seen).

Heads Circumference: Any measurement greater than 2.5 cm, above or below normal value for age is abnormal.

Types of injuries

1. Injuries to scalp: These range from abrasion, subgaleal or sub-periosteal hematoma to laceration of various size, shape and depth, depending upon mode and nature of injury. Major lacerations should be sutured as soon as possible, to prevent further blood loss.

2. Fractures

a. Linear fracture: Single fracture line passes through entire thickness of skull. A simple linear fracture in itself does not require any intervention. Patient should placed in PICU observation specially when the fractures lines are crossing over trajectory of meningeal artery or dural sinus. These innocuous looking fractures may lead to epidural hematoma and gradual deterioration due to oozing from bone or rapid deterioration when arterial

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Table 1: Modified Glasgow Coma Scale

<table>
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<tr>
<th>Ocular response</th>
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<tbody>
<tr>
<td>4. Pursuit</td>
</tr>
<tr>
<td>3. EOM(Extra ocular muscles) intact, reactive pupils</td>
</tr>
<tr>
<td>2. Fixed pupils or EOM impaired</td>
</tr>
<tr>
<td>1. Fixed pupils and EOM impaired</td>
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<table>
<thead>
<tr>
<th>Verbal Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. Cries</td>
</tr>
<tr>
<td>2. Spontaneous Respiration</td>
</tr>
<tr>
<td>1. Apneic</td>
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<table>
<thead>
<tr>
<th>Motor Response</th>
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<tbody>
<tr>
<td>4. Flexes and extends</td>
</tr>
<tr>
<td>3. Withdraws from painful stimuli</td>
</tr>
<tr>
<td>2. Hypertonia</td>
</tr>
<tr>
<td>1. Flaccid</td>
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bleeding accumulates or severe bleeding from venous sinus occurs.

Acute epidural hematoma in contrast to adults is as common in posterior meningeal basal territory as it is from middle meningeal artery.

b. Diastatic fracture: Linear, Comminuted, Stellate
Common in children < 3 years
A diastatic linear fracture is break in skull continuity, more specifically sutures, with coarse definite separation of bone edges by > 5 mm over distance of 2 cm. They are potential source of epidural or subdural hematoma or meningocele.

c. Basilar skull fracture: Basilar skull fracture may present as raccoon eyes (anterior fracture) or battle sign (petechial hemorrhage over mastoid in middle cranial fossa fracture). Basilar skull fractures may lead to cerebrospinal fluid (CSF) leak or cerebrocele. With CSF leak chances of meningitis are very high and appropriate meningeal coverage with antibiotics (ceftriaxone and vancomycin) should be considered.

d. Depressed skull fracture: Outer table of one or more of the fracture edges lie below normal anatomic level of the inner table as compared to surrounding intact skull. It could be closed or open. An open depressed fracture is surgical emergency.

Surgery for closed depressed fracture is usually cosmetic but if radiological appearance suggest dural laceration, brain penetration, mass effect, or underlying subdural hematoma, extradural hematoma or Intracranial hematoma exploration required.

In neonates and infants depressed fracture are typically green stick fracture located in frontal or parietal areas. They are called “Ping-Pong” fracture because they show indented bone without fracture in cortical areas. They may resolve spontaneously, fracture > 5 mm may require surgical intervention.

e. Compound skull fracture: This is a neurosurgical emergency. Once diagnosed clinically, a skull X-ray and CT scan of the head is mandatory. Appropriate management can be undertaken based on findings.

2. Intracranial hematoma
Extradural (epidural), subdural, intradural, contusions may be encountered. CT scan of the head is diagnostic and the investigation of choice.

Epidural hematoma
Epidural hematoma occurs mainly at the convexity or posterior fossa. It could occur due to middle meningeal arterial bleed or tearing of bridging veins. It occurs due to compression/decompression injury, acceleration/deceleration injury or direct trauma and usually present as:
1. Initially normal child coma score followed by progressive deterioration.
2. Initial loss of consciousness, followed by recovery (Lucid interval), followed by loss of consciousness.
3. Continuous progressive deterioration.

Subdural hematoma (SDH)
SDH can be acute, subacute or chronic. Acute subdural hematoma is associated with poor outcome and is clinically characterized by severe depression of level of consciousness with focal deficit and cerebral injury.

Subacute subdural hematoma presents as progressive deterioration of consciousness and/or neurological function, associated with good outcome with appropriate and timely neurosurgical intervention.

Chronic subdural hematoma is the most common entity in pediatric traumatology, and is amenable to surgical treatment.

Intra cerebral hematoma and contusions
These have variable and multiple presentations ranging from perfectly normal to mildly depressed level of consciousness, to severe neurological deficit, seizures and coma. They may or may not require surgical intervention, may or may not respond to surgical management depending upon extent and nature of damage.

3. Diffuse brain injury
Depending upon severity of impact, head injury could lead to minor concussion or diffuse axonal injury. A concussion is temporary loss of neurological function immediately after trauma with no radiological findings and
associated with complete recovery. While diffuse axonal injury is usually due to shearing movements of the brain during acceleration deceleration injuries, it is almost always associated with poor outcome. Patient with poor neurological status whose clinical status does not correlate with CT scan findings should be subjected to MRI which usually will reveal lesion in corpus callosum, rostral part of pons or in the internal capsule.

4. Other injuries
Some of the other injuries commonly seen are CSF leak, growing fracture of skull or cerebral vascular injury.

Radiological investigations
1. Skull X-ray: lateral view and AP view of skull should be taken.
   It can reveal fracture, diastasis and other bony abnormalities. If a CT scan is easily available X-ray skull can be avoided as with a good CT all the relevant information obtained X-ray can be obtained with bone windows detecting any fractures.

2. CT Scan: CT is the investigation of choice. It reveals bony injury, hematoma (appears hyperdense when compared to brain parenchyma), evidence of cerebral edema (hypodense compared to normal brain parenchyma and isodense compared to CSF) or mass effect (midline shift).
   CT is necessary for operative planning.

3. MRI (Magnetic resonance imaging) Scan: MRI had no role in acute management of a patient with head injury. It can be used in cases of diffuse axonal injury and in follow up for prognostication. Some time a small lesion in vital areas of brain may not be seen on CT but seen on MRI. MRI is usually reserved for later detailed evaluation after acute problem has been addressed.

4. Cervical Spine (Lateral View): A lateral view of the cervical spine is obtained in the emergency room to rule out cervical bony injury or dislocation. This must be followed by full cervical spine series with anteroposterior and oblique views to rule out any fracture or dislocation. Cervical spine collar should be kept on until a fracture or dislocation of the cervical spine has been ruled out.

Neurosurgical management
Neurosurgical management includes operative removal of extradural (epidural) hematoma or subdural hematomas as soon as possible after the diagnosis is made. ICP monitor needs to be placed in most cases for ICP and CPP monitoring and further management in the pediatric intensive care unit (PICU).

Non Surgical Management
Management in the PICU
The basic aim is to minimize the primary injury and prevent secondary insult to the brain. Treatment remains supportive. As discussed in pathogenesis the key to management of a head injured patient with cerebral edema or diffuse axonal injury is to maintain cerebral perfusion pressure by control of ICP and hemodynamic status.

Primary cerebral injury is due to trauma per se, accumulation of blood clot and tissue damage leading to decreased cerebral blood flow, increased cerebral blood volume, increased intracranial pressure and decreased cerebral perfusion pressure.

Secondary injury is mainly due to failure of auto regulation and biochemical changes, which lead to cerebral edema (vasogenic, cytotoxic, hydrostatic or osmotic.) Tissue damage is mainly due to direct injury or apoptosis changes.

Seizure control
Midazolam may be used to control seizures in patients with status epilepticus. Propofol or Thiopentone may also be used, however hypotension associated with the use of these agents should be treated with fluid therapy.

Phenytoin should be initiated in patients with post traumatic seizures, although use of prophylactic phenytoin in all head injured children is not supported by clinical evidence. In patients who are sedated and muscle relaxed, EEG should be monitored and nonconvulsive seizures should be controlled to reduce the cerebral oxygen consumption.

Management of Intracranial hypertension and cerebral perfusion pressure (CPP)
Intracranial hypertension is defined as ICP >20 mm
Recommendations for adults include maintaining an ICP of less than 20 mmHg and perfusion pressure (CPP) of >60 mmHg. These may be applicable for older children. Although no normal data is available through clinical evidence, in infants with severe traumatic brain injury it would be logical to suggest targeting for an ICP of <15 mm Hg and a CPP of >45 to 50 mm Hg. Careful use of inotropic agents such as dopamine may be necessary to maintain these parameters, however induced hypertension with phenylephrine is not recommended since cerebral autoregulation is lost and further increase in cerebral blood flow may exacerbate edema formation by hydrostatic effect.25,26

CSF drainage by ventriculostomy has been shown to be as effective as mannitol therapy in reducing ICP.27

Hypoxia and hypotension at any point of management is associated with poor outcome, should, therefore be avoided or promptly treated.

**Mechanical ventilation**

In all children with Child Coma Score is less than 8, intubation and mechanical ventilation is indicated, head should be kept in midline and 30 degrees elevated position.28 Arterial blood gases should be monitored to maintain pCO₂ levels between 31 to 32 rather than 26 to 27 mm Hg for better outcome 6 months after trauma.29 Hypercarbia must be avoided. Positive endexpiratory pressures (PEEP) in the range of 4 to 5 cm of H₂O are harmless as far as ICP is concerned and should be used to prevent atelectasis.

**Sedation and muscle relaxation**

Sedation and muscle relaxation is recommended for adequate control of ICP. Fentanyl, midazolam and vecuronium infusion is a good combination as long as hypotension is avoided. Subject to availability, pentobarbital, phenobarbitone, propofol may be used. Intermittent thiopentone and Intravenous lidocaine is recommended to blunt raised ICP response while suctioning the endotracheal tube. Alternatively instillation of lidocaine in the endotracheal tube may be as effective.

**Fluid therapy**

Initial resuscitation should be done with normal saline or ringers lactate solution to support hemodynamic status in a hypotensive patient. A central line should be placed to guide fluid therapy by central venous pressure monitoring. Glucose should be maintained at normal level. Hypoglycemia should be avoided in infants and neonates. More commonly, due to stress of the head injury, serum glucose is high, glucose containing fluids should be avoided initially. Ringers lactate or half normal saline may be used. Hypertonic saline has been used by some centers in presence of hyponatremia due to cerebral salt wasting syndrome. Hypertonic saline used for reducing refractory ICP, has however not been shown to affect survival in clinical studies.30

If hyponatremia is due to SIADH (syndrome of inappropriate antidiuretic hormone secretion) fluid restriction may be required. Blood and plasma are used as guided by the hematocrit and coagulation profile.

**Mannitol**

Mannitol can reduce ICP by two mechanisms. Reduction of viscosity which is transient, and dependent upon autoregulation being intact. More potent action, however is by its osmotic effect.

Mannitol in dosages of 0.5 to 1 gm/kg may be used intravenously at 6 hourly interval with monitoring of serum osmolality (to be kept under 320). mannitol should not be used if serum osmolality is >330, patient is hypertensive, patients with renal failure. Rapid pushes of mannitol can transiently increase ICP by causing transient systemic hypertension, therefore should be avoided. Mannitol has a theoretical risk of enlarging a hematoma by rapid shrinkage of brain and tearing of bridging veins. Therefore a CT may be necessary to rule out a hematoma before mannitol therapy is initiated.

**Induced hypothermia**

Prevention of iatrogenic hyperthermia and prompt treatment for fever is very important to keep cerebral metabolic oxygen requirements down.

In patients with refractory ICP induced hypothermia may be used as a last resort, however clinical studies have shown this to be of no benefit.

**Steroids**

Always controversial, but there is no clinical evidence
to use in cerebral edema due to head injury. Steroid use should be reserved for patients with brain tumors.

**Nutrition**

Early institution of enteral feeds is recommended if there is no associated intraabdominal injury to major organs such as liver, spleen, or duodenal hematoma.

**Rehabilitation**

Tracheostomy may be indicated in patients in prolonged comatose state who cannot protect their airway or require long term ventilation.

Once patient is recovered from acute injury in the PICU, early physical therapy for prevention of deep vein thrombosis and prevention of contractures may be necessary. Arm, leg, hand, and feet splints should be used as indicated.

**Evidence based international guidelines**

The guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents were published on June 6, 2003 as special supplements to Pediatric Critical Care Medicine, Critical Care Medicine, and the Journal of Trauma, ensuring a multidisciplinary audience of more than 30,000 specialty physicians. According to brain injury association of America, more than 1 million children in the US sustain brain injuries each year, of whom 250,000 are admitted to hospitals, 7000 die, and 30,000 are permanently disabled. Indian statistics though not available should be comparable at the least. Following is a summary of available evidence in the literature. This information could be used to apply to care of head injuries infants and children in the Indian scenario.

1. Pediatric patients with traumatic brain injury should be treated in a pediatric trauma center or, failing that, a tertiary care hospital with pediatric trauma care capability.

2. Hypoxia must be treated appropriately; however, there is no evidence to support endotracheal intubation versus bag-mask ventilation during transfer to the hospital.

3. Prophylactic treatment with mannitol or mild hyperventilation is usually unnecessary but should be used in patients with evidence of cerebral herniation or worsening neurological function.

4. Intracranial pressure monitoring is indicated for children with a Glasgow coma score of less than 8, but it may also be employed for children in whom serial neurological examination is not feasible.

5. Treatment for increased intracranial pressure should be initiated when the pressure rises more than 20-25 mm Hg.

6. The sensitivity of ventricular catheters, external guage transducers, or catheter tip pressure transducers in monitoring intracranial pressure appears equal. Subarachnoid, subdural, epidural and externally placed monitors are less accurate.

7. Cerebral perfusion pressure should be maintained at more than 40 mm Hg. Further research is needed to determine an optimal cerebral perfusion pressure range.

8. The routine use of sedation and neuromuscular blockade in severe pediatric traumatic brain injury is not supported by scientific evidence. Coughing and bucking on the tube and suctioning maneuvers leading to increased intracranial pressure may necessitate their use, in practice.

9. Cerebrospinal fluid drainage via ventriculostomy is a first line option for refractory elevated intracranial pressure; lumbar drainage may be added in patients with open cisterns on imaging and without major mass lesions or midline shift.

10. Mannitol or hypertonic saline are both acceptable agents for lowering intracranial pressure.

11. Hyperventilation should only be used as a second line method to reduce refractory intracranial pressure. In this setting, the PaCO2 should be kept at 25-30 mmHg.

12. High dose barbiturates may also be employed in the treatment of refractory increased intracranial pressure. Patients receiving this therapy require extremely close monitoring for hypotension.

13. Hyperthermia has been postulated to increase secondary mechanisms of brain injury in adults. Therefore, hyperthermia should be avoided in younger patients. Hypothermia on the other hand, may be beneficial, and when intracranial hypertension is refractory, the authors recommend that it be considered, despite lack of evidence.

14. Decompression craniectomy may also be considered to improve refractory intracranial hypertension. Surgical interventions may be more successful in patients with reversible brain insults.

15. There is no evidence to recommend steroid therapy in children with traumatic brain injury.

16. Although research had not directly addressed out-
comes in pediatric patients with traumatic brain injury, nutritional support should be strongly considered with a goal of 130%-160% of resting metabolic expenditure.

17. Prophylactic antiepileptic medications are not recommended.

Complication and sequelae of head injury
Various sequelae can be seen as follows:

**Early**
- Transient cortical blindness
- Seizures
- Cranial Nerve palsy
- Diabetes insipidus
- Syndrome of inappropriate secretion of ADH
- Cortical venous occlusion
- Hemiparesis

**Late**
- Post traumatic epilepsy
- Post traumatic aneurysm
- Meningitis
- Hydrocephalus
- Memory loss
- Disability
- Muscle contractures

Outcome after pediatric head injury

Child outcome score has been described based on various parameters such as neurological and cognitive deficit.

**Child outcome score**
I. Child Outcome Score Excellent recovery  
II. Mod but non disabling deficit  
III. Either a secure motor or cognitive deficit  
IV. Vegetative  
V. Death

I and II – Good outcome  
III to V – Poor outcome

Indices of good outcome and poor outcome have already been described earlier in this chapter.

Summary

Young children under 4 years of age have been shown to poor outcomes after severe head injury.

Severe traumatic brain injury remains a common problem with significant morbidity and mortality. A proper medical management needs an organized team approach ranging from prehospital care, emergency room and pediatric intensive care management. Prompt detection of mass lesions (hematomas) and neurosurgical intervention is very important. In non neurosurgical patients with cerebral edema, control of ICP and cerebral perfusion pressure by ICP monitoring, mechanical ventilation, hemodynamic support, mannitol and judicious use of fluids and blood products have been helpful in successful management of these children.

Use of helmets, seat belts, infant seats and restraints are probably most important measures to prevent severe injuries in case of an accident. In addition, child and adult education regarding road safety, following traffic signals, care during road crossing can go a long way.

Conclusions

1. Prompt recognition and transfer of pediatric victims of traumatic brain injury is paramount to good outcomes
2. Intracranial pressure should be monitored and treated if >20 mm hg in children with severe brain injury
3. Mannitol, hypertonic saline, ventricular drainage, hyperventilation, barbiturates and hypothermia should be considered in refractory intracranial hypertension.
4. Steroids and prophylactic antiepileptics have no role in traumatic brain injury in children.

References