CLOSED HEAD INJURY IN PEDIATRICS

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1. Background

Trauma is the leading cause of death in children over 1 year of age. 80% of deaths due to trauma are due to head injury. Head injury in the pediatric population is most often due to motor vehicle collisions. A large percentage of head injuries are due to child abuse, falls and recreational activities. Two things that must be considered in every pediatric patient with a head injury are the possibility of associated cervical spine injury and the possibility of abuse.

The pediatric brain is more susceptible to injury and complications of head injury than the adult brain. There is increased water content in infants and young children's brains 88% versus 77% in adults. This makes them softer and more likely to be injured in acceleration-deceleration mechanisms. Also they have decreased myelination which increases the susceptibility to shear injury. The compliant infants skull is also easily deformed leading to underlying brain parenchymal injury. This also can be pretective in that very young children with open sutures can better tolerate increased intracranial pressure.

2. Diagnosis

a) Types of closed head injuries

Concussion

- transient loss of consciousness
- vomiting, amnesia, pallor
- waxing and waning mental status but no focal deficit
- □ improve over time

Contusion

- □ from coup or contre-coup mechanism
- most often frontal or temporal lobes
- see progressive deterioration secondary to cerebral edema, hematoma and infarct to area.
- focal deficits depending on area involved
- decreased LOC

Epidural Hematoma

- bleeding between dura and skull
- often due to middle meningeal artery laceration
- □ reaches peak size in 6-8 hours
- characteristic lucid interval seen in adults may not happen in pediatric patients
- mass effect leading to herniation symdromes

Sudural hematoma

- between dura and arachnoid
- usually due to tearing of bridging veins
- acceleration-deceleration injuries or shaken baby
- often have associated parenchymal injury
- □ if large see profound progressive deterioration

Subarachnoid Hemorrhage

- due to damage to small vessels over cortex
- □ headache, nausea and vomiting and neck stiffness

Diffuse Axonal Injury

- rapid acceleration-deceleration injury
- basal ganglia, thalamus, corpus callosum most often affected
- altered mental status and prolonged vegetative state
- minimal findings on imaging
- poor prognosis for recovery

3. Questions to Ask

- a) Was the injury witnessed
- b) Mechanism of injury and forces involved (speed of vehicles, seat belt?)
- c) Loss of conciousness, lucid period
- d) neurological deficits noticed: vision, balance, motor or sensory function
- e) seizures
- f) nausea or vomiting
- g) amnesia, disorientation
- h) medications, alcohol or drugs involved
- i) history of coagulation disorders and other medical problems

^{***}Note: In the situation of acute head injury history may become secondary to initial resuscitation efforts

4. Physical examination

Often head injury occurs in the setting of the multi-trauma patient. So the head injury has to be assessed along with other injuries

a) Primary survey

- Airway
- Breathing
 - Head injury patients often have apneic spells and hypoventilation
- Circulation
 - Hypotension should not be attributed to head injury alone until other causes have been ruled out
 - Cushing's Triad (↓HR; ↑BP; irregular respiration): seen with increased ICP (link to signs of inc_ICP)
- Neurological Assessment
 - Pediatric GCS (link pediatric GCS)
 - AVPU (alert, verbal, pain, unresponsive)
 - Pupils (Unilateral, dilated, unreactive pupil suggests actual or imminent uncal herniation and is a neurosurgical emergency. Bilateral dilated unreactive pupils is even more ominous)
- Cervical spine stability

b) Secondary Survey

Examine head to toes, paying special attention to the following:

- Cervical spine
- Head: deformities, lacerations, Battle's sign, Raccoon eyes, hemotympanum, otorrhea, rhinorrhea, bulging fontanelle
- Neurological: Pupils, eye deviation, retinal hemorrhage, papilledema, decreased venous pulsations, reflexes (motor, corneal, gag and oculovestibular), motor and sensory function.

5. Investigations

- a) <u>Laboratory investigations</u>
 - □ CBC
 - □ PTT/INR
 - Blood type and cross
 - Arterial blood gases
 - Toxicology screen if history of head injury is unclear

b) ECG

Head injury can lead to cardiac dysrhythmias

c) Imaging studies

- □ **CT** is the gold standard for initial assessment of head injury, and is indicated in the following circumstances:
 - LOC >5 min
 - Deterioration of mental status
 - Focal deficit
 - Seizure
 - Vomiting > 6 hours
 - Bulging fontanelle
 - Suspicion of skull fracture
 - Other injury requiring general anaesthetic
- Ultrasound may be used in infants with open fontanelle

6. Management

- □ Airway
 - Intubate if GCS <8 (link to pediatric GCS)
- Breathing
 - Maintain normal ventilation
 - PaO2 >90
- Circulation
 - Normotensive, euvolemic is the goal
 - CPP=MAP-ICP
 - Cardiac monitoring
- Increased ICP (link to signs of inc_ICP)
 - Elevate head of bed 30 degrees
 - Mannitol or Furosemide
 - Sedate and paralyze if needed
 - Hyperventilation to PaCO2 35
 - Barbituates and hypothermia as last resort
 - Extraventricular drain for CSF
- Seizure management
- Bleeding management: one third head injury patients develop DIC
- Neurosurgical definitive treatment
 - Burr holes
 - Craniotomy
 - Drains

7. Disposition criteria

- Minor injury
- Responsible adult caregiver
- □ Awake every 2 hours
- No focal deficit
- No LOC or normal CT
- □ No signs or symptoms after 2 hours observation
- Return to ED if increasingly sleepy, unarousable, unequal pupils, decreased motor function, seizure, protracted vomiting, severe head ache, change in mental status

8. Complications of Head Injury

- Seizures
- Cortical blindness (transient)
- Hydrocephalus
- Neurogenic pulmonary edema
- DIC- permanent
- Neurological defect

References

Acknowledgement

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