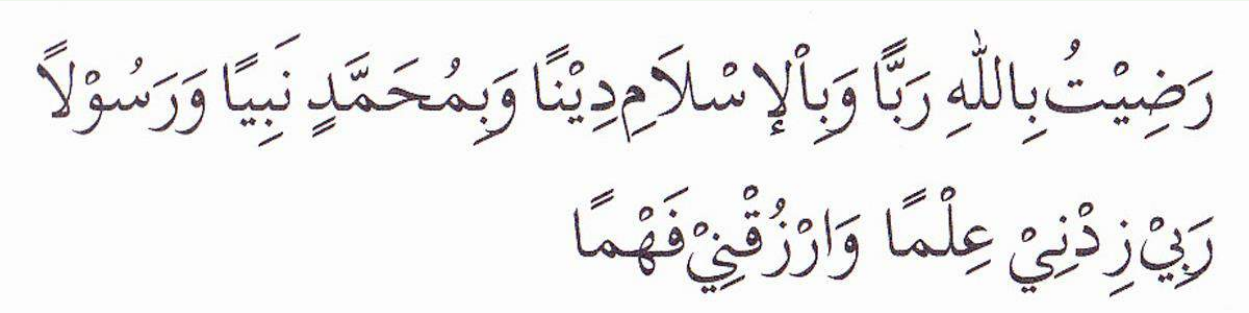
Universitas 'Aisyiyah
Yogyakarta

HEAD TRAUMA

Dwi Prihatiningsih, M.Ng.

Dua before Studying



"I've accepted Allah as my Lord And Islam as my way of life
And Muhammad As Allah's Prophet and Messenger. O Allah! Advance me in Knowledge and true understanding"

Learning Outcome

At the end of this topik, students will be able to show understanding of:

1. Clasification
2. Phatophysiology
3. Treatment/Management

Introduction

- Every year in the United States 1.7 million people suffer Traumatic Brain Injury (TBI)
- TBI is listed as a contributing cause in approximately one third of injury-related deaths
- Head trauma is more common in children, adults up to 24 years, and those older than 75 years
- TBI is 3 times more common in males than females.
- Although only 10% of TBI occurs in the elderly population, it accounts for up to 50% of TBI-related deaths

Clasification

Based on the Glasgow Coma Scale (GCS) score, it is classified as:

- ❑ Mild = GCS 14 to 15, also called concussion
- ❑ Moderate = GCS 9 to 13
- ❑ Severe = GCS 3 to 8

Universitas 'Aisyiyah Yogyakarta

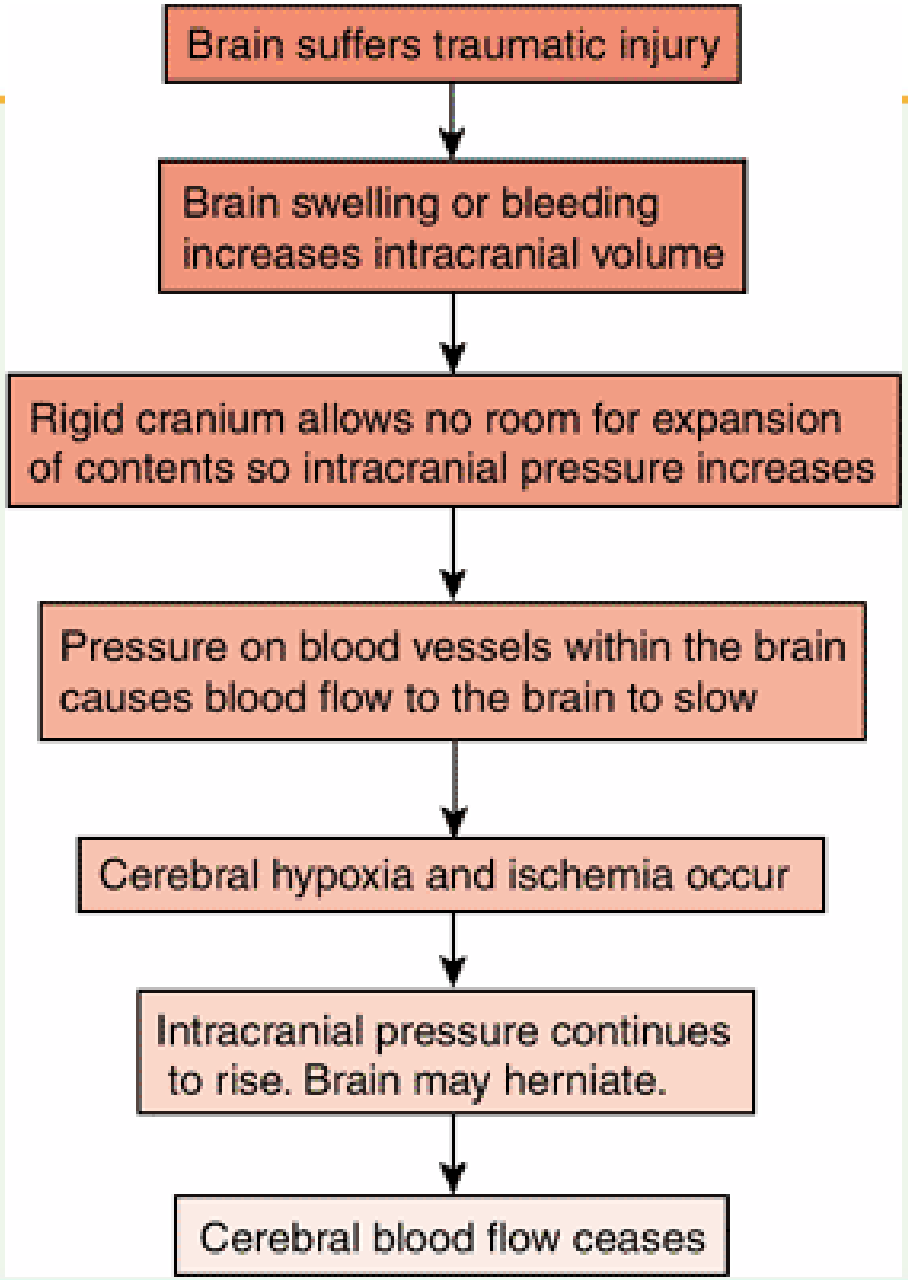
GCS

BEHAVIOR	RESPONSE	SCORE
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place, and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
Best motor response	Obeys commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1

Classification

Primary Injury	Secondary Injury/Secondary Neurotoxic Cascade
Primary injury includes injury upon initial impact that causes displacement of the brain due to direct impact, rapid acceleration-deceleration, or penetration.	Secondary injury consists of the changes that occur after the initial insult. It can be due to:
• Contusion (bruise on the brain parenchyma)	• Systemic hypotension
• Hematoma (subdural, epidural, intraparenchymal, intraventricular, and subarachnoid)	• Hypoxia
• Diffuse axonal injury (stress or damage to axons)	• Increase in ICP

Pathofisiology



Treatment / Management

The most important goal is to prevent secondary brain injuries. This can be achieved by the following:

- ❑ Maintain airway and ventilation
- ❑ Maintain cerebral perfusion pressure
- ❑ Prevent secondary injuries (by recognizing and treating hypoxia, hypercapnia, or hypoperfusion)
- ❑ Evaluate and manage for increased ICP
- ❑ Obtain urgent neurosurgical consultation for intracranial mass lesions
- ❑ Identify and treat other life-threatening injuries or conditions (if they exist)

A relatively higher systemic blood pressure is needed:

- ❑ Increase in intracranial pressure
- ❑ Loss of autoregulation of cerebral circulation

Priorities remain the same: the ABC also applies to TBI. The purpose is to optimize perfusion and oxygenation.

Management: Airway & Breathing

- ❑ Identify any condition which might compromise airway, such as pneumothorax.
- ❑ For sedation, consider using short-acting agents having minimal effect on blood pressure or ICP:

  Induction agents: Etomidate or propofol

  Paralytic agents: Succinylcholine or Rocuronium
- ❑ Consider endotracheal intubation in the following situations:
- Inadequate ventilation or gas exchange such as hypercarbia, hypoxia, or apnea
- Severe injury (GCS score of = 8)
- Inability to protect airway
- Agitated patient
- Need for patient transport

Management: Airway & Breathing

- The cervical spine should be maintained in-line during intubation.
- Nasotracheal intubation should be avoided in patients with facial trauma or basilar skull fracture.
- Targets:

  Oxygen saturation > 90

  PaO2 > 60

  PCO at 35 - 45

Circulation

- Avoid hypotension. A normal blood pressure may not be adequate to maintain adequate flow and CPP if ICP is elevated.
- Target
  Systolic blood pressure > 90 mm Hg
  MAP > 80 mm Hg
- Isolated head trauma usually does not cause hypotension. Look for another cause if the patient is in shock.

Increased ICP

- Increased ICP can occur in head trauma patients resulting in the mass occupying lesion. Utilize a team approach to manage impending herniation.
- Signs and symptoms:
  Change in mental status
  Irregular pupils
  Focal neurologic finding
  Posturing: decerebrate or decorticate
  Papilledema (may not be apparent with rapid elevation of ICP)
- CT scan findings:
  Attenuation of sulci and gyri
  Poor gray/white matter demarcation

General Measures

- **Head Position**: Raise the head of the bed and maintain the head in midline position at 30 degrees: potential to improve cerebral blood flow by improving cerebral venous drainage.
- **Lower cerebral blood volume (CBV)** can lower ICP.
- **Temperature Control**: Fever should be avoided as it increases cerebral metabolic demand and affects ICP.
- **Seizure prophylaxis**: Seizures should be avoided as they can also worsen CNS injury by increasing the metabolic requirement and may potentially increase ICP. Consider administering fosphenytoin at a loading dose of 20mg/kg.
- Only use an anticonvulsant when it is necessary, as it may inhibit brain recovery.

General Measures

- **Fluid management**: The goal is to achieve euvolemia. This will help to maintain adequate cerebral perfusion. Hypovolemia in head trauma patients is harmful. Isotonic fluid such as normal saline or Ringer Lactate should be used. Also, avoid hypotonic fluid.
- **Sedation**: Consider sedation as agitation and muscular activity may increase ICP.
- **ICP monitoring:** Normal ICP in adults is 5–15 mmHg and in TBI an ICP of >20 mmHg is widely accepted as ICH
- **Hyperventilation:** Normocarbia is desired in most head trauma patients. The goal is to maintain PaCO between 35-45 mmHg. Judicious hyperventilation helps to reduce PaCO2 and causes cerebral vasoconstriction. Beware that, if extreme, it may reduce CPP to the point that exacerbation of secondary brain injury may occur. Avoid hypercarbia: PaCO > 45 may cause vasodilatation and increases ICP.

General Measures

Mannitol

- A potent osmotic diuretic with net intravascular volume loss
- Reduces ICP and improves cerebral blood flow, CPP, and brain metabolism
- Expands plasma volume and can improve oxygen-carrying capacity
- Onset of action is within 30 minutes
- Duration of action is from two to eight hours
- Dose is 0.25-1 g/kg (maximum: 4 g/kg/day)
- Avoid serum sodium > 145 m Eq/L
  Serum sodium > 145 m Eq/L
  Serum osmolality > 315 mOsm
- Relative contraindication: hypotension does not lower ICP in hypovolemic patients.

References

Shaikh, F & Waseem, M (2018). Head Trauma. NCBI Bookshelf, available at https://www.ncbi.nlm.nih.gov/books/NBK430854/

unisa

Universitas 'Aisyiyah
Yogyakarta