Traumatic Brain Injury: Management of Increased Intracranial Pressure (ICP)

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Traumatic injuries due to traffic accidents, sports or violence induced incidents, represent one of the major causes of morbidity and mortality nowadays. Significant portion of all trauma cases include head trauma, which can be divided into fractures, brain traumatic injuries, hematomas (both extra axial and parenchymal) and diffuse axonal injuries (DAI). Recognition of the signs and symptoms is of uttermost importance, for head trauma can be as mild as every day bruises and bumps to the head, or severe and life threatening. Rapid recognition, dedicated diagnosis and adequate imaging which lead to proper treatment - both conservative and surgical - are essential for the best possible, complication- and sequel-free, outcome.

Definition of Head Trauma

Head trauma includes a wide spectrum of injuries to the head, including bones, brain, meninges and intracranial vascular structures. But mostly, the term head trauma is synonym for traumatic brain injury (TBI).

Increased intracranial pressure is every rise above the threshold of 20 mm Hg independently of reason.

Classification of Head Trauma

Head trauma can be divided:

1. By the clinical presentation:
   - Mild: Loss of conscience shorter than 30 minutes, normal CT and/or MRI scan. Patient may have cognitive problems like headache, memory
problems, attention and mood changes.

- **Severe:** Loss of consciousness longer than 30 minutes, memory loss longer than 24 hours. Neurological deficit, that may be nonreversible, is presented as impairment of movement, higher level cognitive functions, with potential progression to comatose state.

2. By **type of injury**:
   - **Open:** Injury that involves fracture of head bones, brain tissue is directly injured by fragments of bone, bullets, knife, etc.
   - **Closed:** Intact head bones and dura mater, brain is damaged due to acceleration-deceleration, can be both focal and diffuse.

3. By **time of presentation**:
   - **Primary:** Occurs at the moment of injury, and could be of pure mechanical nature (object passing through brain tissue and meninges and causing direct trauma), and brain hitting the bone on/opposite to the impact site such as coup and counter coup lesion; or acceleration-deceleration injury when unrestricted movement of head leads to strains and causes hematomas, vascular or stalk injury.
   - **Secondary:** Injuries that occur hours/days/weeks after trauma, and are not a consequence of direct trauma, but are considered to be consequence of impaired cerebral blood flow (CBF), resulting in local of diffuse brain edema, hemorrhage or increased intracranial pressure (ICP) complicated by electrolyte disbalance potentially resulting in neuronal and/or patient’s death.

Pathophysiology of Head Trauma

**Fractured bones** (linear, displaced, impacted) and lesion of underlying dura or arachnoid membrane resulting in epidural or subdural hematomas; brain injuries which consist of intraparenchymal hematomas, contusion of underlying brain tissue and diffuse axonal injuries.

**Epidural hematoma** is, potentially rapidly expanding blood collection between the bone and dura mater originating from meningeal arteries. It is a life threatening condition that usually requires surgical procedure.

**Subdural hematoma** is blood collection between dura and arachnoid membrane that originates from injured bridging veins. It can be both acute and chronic, and requires prompt, usually surgical care.

**Intraparenchimal contusion** (areas of swollen and damaged brain tissue) and hematomas can occur on the contact site (coup lesions), as well as on contralateral site (known as counter coup lesion).

**Diffuse axonal injury (DAI)** is one of the most important pathologic features of brain trauma. Microscopic damage of the brain tissue, resulting from rotational acceleration of the brain and subsequent unrestricted head movement, is complicated to recognize both on clinical exam and on imaging studies (MRI is gold standard for recognition of DAI).

Pathophysiological exam shows multiple swollen and disconnected axons, leading to changes in normal neuron function. Mainly decline in cerebral blood flow, both local and global, is the reason for this. It results in edema, hemorrhage and increased intracranial pressure. Impaired cerebral blood flow and raised intracranial pressure lead the cascade of electrolyte disorders eventually resulting in neuronal death.
Following **Monro-Kellie doctrine** it’s known that the adult skull, because the sutures and fontanels are closed, is a rigid nonexpandable vault. Its volume remains constant regardless of the proportion of individual constituents, presented as cerebral parenchyma (80 %), cerebrospinal fluid (CSF) (10 %) and blood (10 %). Also, the rise of volume in one compartment raises intracranial pressure.

**ICP** is the pressure inside the cranial vault, with physiological range between 10-20 mmHg in adults and 3-7 mmHg in children.

ICP depends on the brain, CSF and cerebral blood interaction. It depends gravely on CSF production volume, system resistance to reabsorption of CSF and venous pressure in superior sagittal sinus.

**Raised ICP can lead to intracranial herniation**

- Transtentorial (either lateral or central)
- Tonsillar
- Subfalcine

Physiologically, **pressure variations** are rapidly compensated by **CSF displacement** into the subarachnoid space, spinal canal and lumbar cistern that leads to cerebral blood flow (CBF) decrease.

**ICP is regulated in few stages:**

- Firstly, increased intracranial volume doesn’t affect ICP because CSF is displaced in the ventricular system and spinal canal, thus compensating volume change.
- Next level is volume overload, when the system becomes decompensated and finally ceases to function which leads to prompt and rapid changes in ICP, even after minor volume increase.
- In long standing, high pressure situations brain parenchyma undergoes reductive changes and neurons and glial cells are lost.

So when **buffer mechanisms** fail, increased intracranial pressure (ICP) leads to **decrease in blood supply** and **decreased cerebral perfusion pressure** (CPP).

- CPP is calculated as a result of subtraction of mean arterial pressure (MAP) and intracranial pressure (ICP) values.

Cerebral blood flow (CBF) is represented by 15 % to 25 % of the cardiac output, and approximately 40 to 50 mL/100g of brain tissue/min. But blood flow volume isn’t the only thing that regulates oxygen supply to the brain. Oxygen supply is inadequate in cases of underlying anemia and hypoxia.

**Cerebral autoregulation**

**Vasodilatation** or **vasoconstriction** are mechanisms that ease the cerebral autoregulation function in order to maintain CBF at an appropriate level and fulfill the brain’s metabolic oxygen requirements.

Cerebral autoregulation is a multifactorial issue in first line sensitive to MAP and partial pressure of arterial carbon dioxide (PaCO2), and to a lesser extent to partial pressure of arterial oxygen (PaO2) and other parameters. Nevertheless, once the upper or lower limits of autoregulatory mechanisms are exceeded, CBF becomes solely dependent on MAP, which makes **arterial pressure monitoring and therapy** essential in head trauma
Clinical Features of Head Trauma

Knowing that head trauma may present itself in a very wide spectrum of clinical scenarios, and always having in mind that minor injury appearing patients may be the ones with rapidly fatal outcome, one must very seriously and carefully evaluate every trauma patient.

Signs and symptoms are nonspecific; primary estimation is roughly based on **Glasgow Coma Score** that constitutes of measured motor response, verbal response and eye opening response with the following values:

1. **Motor response**
   - 6 – Obeys commands fully
   - 5 – Localizes to noxious stimuli
   - 4 – Withdraws from noxious stimuli
   - 3 – Abnormal flexion, i.e., decorticate posturing
   - 2 – Extensor response, i.e., decerebrate posturing
   - 1 – No response

2. **Verbal response**
   - 5 – Alert and Oriented
   - 4 – Confused, yet coherent, speech
   - 3 – Inappropriate words and jumbled phrases consisting of words
   - 2 – Incomprehensible sounds
   - 1 – No sounds

3. **Eye opening**
   - 4 – Spontaneous eye opening
   - 3 – Eyes open to speech
   - 2 – Eyes open to pain
   - 1 – No eye opening

The final score is made by adding the values of 1, 2 and 3, and the sum has the following meaning:

**Mild (13-15):**
- Reversible symptoms, loss of consciousness shorter than 30 minutes.

**Moderate disability (9-12):**
- Loss of consciousness greater than 30 minutes;
- Physical or cognitive impairments which may or may not resolve;
- Benefit from rehabilitation.

**Severe disability (3-8):**
- Coma: unconscious state, without meaningful response and voluntary activities.

**Vegetative state (1-3):**
- Sleep wake cycles;
- Arousal, without interaction;
- No localized response to pain.
Persistent vegetative state:
- Vegetative state lasting longer than one month.

Brain death:
- No brain function;
- Specific criteria needed for making this diagnosis (neurological, biochemical, CT or MRI scan).

Mild traumatic brain injury (GCS 13-15)
Also known as concussion, this is the most frequent form of TBI and often missed at initial examination. It constitutes of brief change in mental status namely confusion, disorientation, short loss of memory and/or loss of consciousness for less than 30 minutes.

There are some common, but unspecific symptoms such as headaches, fatigue, poor attention/concentration, irritability-emotional disturbances, and also nausea, loss of smell, mood changes, getting confused.

Potential problem makes the fact that symptoms may not, and often are not, present right after injury, taking days and weeks to develop. In older individuals, slow growing subdural hematoma with confusion, memory loss and subtle balance disorders is the most overlooked trauma sequel that can lead to a range of serious outcomes, fatal end included.

Severe traumatic brain injury (GCS below 13)
This is a serious medical condition presenting as a spectrum of cognitive function disorders such as longstanding or irreversible loss of memory, loss of consciousness for more than 30 minutes; motor function disorders such as extremity weakness, impaired coordination and balance; sensation disorders namely hearing, vision, impaired perception and touch; and very often emotion alterations like depression, anxiety, aggression, loss of impulse control, personality changes.

Clinical staging and diagnosis consist of detailed neurological examination and use of various trauma score charts (GSC is a common option, but there are many other systems); brain imaging is indispensable, routinely in first line CT scan.

- CT scan can visualize bone fractures, epidural or subdural hematomas, intraparenchymal hematomas or contusion of brain parenchyma, vascular structure lesions.

- MRI examinations are reserved for specific situations, primarily to assess DAI. In rare occasions, SPECT and PET scan may be useful.

In a non-acute setting, cognitive evaluation done by a neuropsychologist is essential, and depending on the patients’ needs, physical, occupational and speech therapy should address specific deficits.

Differential Diagnosis
Due to diverse signs and symptoms, and in scenarios of inadequate anamnesis, present TBI must be differentiated from the following:
Management of Head Trauma and Increased Intracranial Pressure

Management of head trauma requires a **multidisciplinary team**. Depending on specific conditions and the severity of injury, outdoor clinic to ICU patient treatment may be necessary.

Hematomas, epidural and subdural, require **surgical decompression** depending on the neurological status and elevation of ICP.

Contusion and DAI injuries usually require **aggressive conservative treatment** to prevent raised ICP and its complications.

Firstly, that includes **elevation of the head of the bed** to improve venous drainage; maintenance of $\text{PaO}_2 > 100 \text{ mmHg}$ and $\text{PaCO}_2 30-35 \text{ mmHg}$ by adequate ventilation and oxygenation; use of the **lowest possible AW pressures** to avoid obstruction of venous drainage; keeping $\text{MAP}$ at pre-intubation level.

Also **manitol** (or 3 % saline) in 20-30 min intervals is used to reduce ICP by reducing **cerebral parenchymal cell water** (although it is well known that afterwards drug enters CSF and increases ICP). This neuromuscular blocking drug reduces ICP by blocking the coughing reflex. **Sedation** by decreasing anxiety, fear and response to pain block the increase of ICP. **Steroids** reduce edema in **traumatic brain injury**, but their effect is much more beneficial in the setting of intracranial tumors.

In 2016, the brain trauma foundation gave **recommendations for specific treatment options** and desired **threshold values**:

- **Blood pressure** thresholds should not exceed 100 mmHg for patients 50 to 69 years old or 110 mmHg or above for patients 15 to 49 or above 70 years as it is considered to decrease mortality and improve outcomes.
- **Intracranial pressure** thresholds: treading ICP higher than 22 mmHg is recommended because values above this level are associated with increased mortality.
- A combination of ICP values and clinical and brain CT findings may be used to make / alter management decisions.
- **Cerebral perfusion pressure**: recommended target CPP value for survival and adequate outcomes is 60 – 70 mmHg (although minimum optimal CPP threshold may depend upon the autoregulatory status of the patient).
- Avoiding aggressive attempts to maintain CPP higher than 70 mmHg with fluids and pressors because of the risk of adult **respiratory failure**.
- **Advanced cerebral monitoring**, such as jugular venous saturation lower than 50% may reduce mortality and improve outcomes.
Complications of Traumatic Brain Injury

Both mild and severe traumatic brain injury can lead to complications, the most common being post traumatic seizures. These frequently occur after severe TBI, communicative hydrocephalus, deep vein thrombosis that occurs in half of the patients, spasticity, gastrointestinal and genitourinary complications that present most common in sequels, post traumatic longstanding agitation and heterotopic ossification which are clinically significant in one fifth of the patients.

References

Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury, J Neurotrauma, 2007


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