Traumatic Brain Injury

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Traumatic Brain Injury

- 8% of all deaths in USA
- 200 per 100,000 /year
- 32,000 head injury/year in Saudi Arabia
- Mortality rate: 15-30 /100,000 /year
- 10,000/year in Saudi Arabia
Traumatic Brain Injury

4,800 Death
24,870 Hospitalized
131,600 medically attended

- Based on 1990 USA estimates. Corrected No. for Saudi Arabia based on a population of 24 M.
Traumatic Brain Injury
Scalp Lacerations:
Indicate more severe injury
• 3% of medically attended TBI cases
• 65% of admitted & 80% of fatal TBI cases
• Depressed: fragments of inner tables of the skull are depressed by thickness of the diploe (11%)
Traumatic Brain Injury

Skull Fractures:
**Compound:** associated scalp laceration

**Penetrating:** if tearing in the dura

Both are potential routes for infection, post traumatic Epilepsy

**Growing skull fracture**
Traumatic Brain Injury

Skull Fractures:

- Skull base fracture
- Infection
- CSF rhinorrhea
- Otorrhea
- Pneumocephalus
Traumatic Brain Injury

Cranial nerves injuries:

- Loss of sense of smell
- Orbital fractures; CNs 2, 3, 4 & 6
- Temporal # skull base: CNs VII & VIII
Traumatic Brain Injury

Extradural (Epidural) Hematoma:

- 2% of all types
- Concomitant # in 85% of patients
- 80% in the temporal area
- Torn meningeal blood vessels
- Younger age group
- Evacuation if >30mm or > 15mm thickness

If not evacuated:
50% increased in 2 wks (liquification)
Get smallers & resolved in 4-6 wks
Rupture of bridging veins in the subdural spaces
• Significant associated cortical injury
• Evacuation if >10mm or > 5mm shift
• Acute within 48hrs
Traumatic Brain Injury

Subdural Hematoma:

- Subacute in 2-14 days
- Chronic in >14 days
Traumatic Brain Injury

**Intracerebral Hematoma:**

- 16% of cases
- Often multiple
- Frontal and temporal lobes
- Rupture of intracerebral vessels
- Association with contusions
- Delayed appearance
Traumatic Brain Injury

Subarachnoid Hemorrhage:

- Most common cause
- Severity of injury
- Vasospasm!
Contusions: the contact between brain surface and the skull bony protuberances (Frontal, orbital, temporal area).

Coup: at the site of the injury

Counter-coup: diametrically opposite point of injury
Concussion of the deep structures of the brain
Leading to widespread neurological dysfunction
Impaired consciousness or coma.
Concussion is a mild form of diffuse axonal injury.
Diffuse Axonal Injury:

- 50% of all severe head injury
- 35% of all deaths
- Vegetative state and severe disabilities
- Shearing injury of the axon leading to degeneration of white matter:
  1- lesions at corpus callosum, brain stem, cerebellar peduncles.
  2- wide spread shearing injury (Micro. damage to axons) axonal retraction balls
Traumatic Brain Injury

Diffuse Axonal Injury:

- 50% of all severe head injuries
- 35% of all deaths
- Most common cause of vegetative state and severe disabilities
- Shearing injury that affects nerve fibers and leads to degeneration of white matter
  1- lesions at corpus callosum, brain stem, cerebellar peduncles.
  2- widespread shearing injury (Micro. Damage to axons) axonal retraction balls
Traumatic Brain Injury

Diffuse Axonal Injury:

- Generalized white matter damage
- Strains of tentorium and falx during accel/deceleration
- Could occur as a result of ischemia.
Diffuse Axonal Injury:

Shearing injury of the axon leading to degeneration of white matter:
- Lesions at corpus callosum, brain stem, cerebellar peduncles.
- Wide spread shearing injury (Micro. damage to axons)
  axonal retraction balls
Traumatic Brain Injury

Ischemic brain damage:

- much more common
- Occur soon after injury
- Hypoventilation, Hypo-perfusion
- Late with high ICP
Patho-physiology of Brain Injury

- **Cerebral Perfusion**
  - ↓ Cerebral Perfusion
  - ↓ Energy Stores
  - Cerebral Ischemia

- **Apoptosis**
  - Cell Shrinkage
  - Chromatin aggregation

- **Inflammation**
  - Cytokines
  - Chemokines
  - Adhesion molecules
  - Proteases

- **Excitatory AA**
  - Glutamate, Aspartate

- **Activation of NMDA & AMPA**

- **BBB Breakdown**
  - Protein phosphorylation
  - Proteolysis
  - Nitric Oxide
  - O₂ free radical production
  - Altered gene expression

- **Neuronal Cell Death**

- **↑ Intracellular Ca²⁺**
Traumatic Brain Injury Evaluation

ABC

Glasgow Coma Score
Traumatic Brain Injury
Transfer To a Neurosurgical Unit:

- By an anesthetist, even if not intubated.
- Physical disturbance during the journey.
- Ensure patient stability before transfer.
- Monitoring during the transfer.
- Formal handover to the neurosurgical team.

- A Copy of the CT scans.
Glasgow Coma Score

Used for:
- level of Consciousness
- Communication
- Management guideline
- Estimate prognosis

Glasgow Coma Scale for Head Injury

Eye opening
Spontaneous 4
To loud voice 3
To pain 2
None 1

Verbal response
Oriented 5
Confused, disoriented 4
Inappropriate words 3
Incomprehensible sounds 2
None 1

Best motor response
Obey 6
Localizes 5
Withdraws (flexion) 4
Abnormal flexion posturing 3
Extension posturing 2
None 1
Traumatic Brain Injury

Management:

Mild head injury (GCS 14-15):

• admitted to a ward
• Frequent neurological observations
• Observed until complete neurological recovery
• Discharged if a responsible adult can supervise
• All patients with a GCS <15 should have a CT scan
### Traumatic Brain Injury Monitoring:

<table>
<thead>
<tr>
<th>Date</th>
<th>HOSPITAL NUMBER</th>
<th>WARD</th>
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**Time (24 hour clock)**

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<thead>
<tr>
<th>Eyes</th>
<th>Spontaneously</th>
<th>To speech</th>
<th>To pain</th>
<th>None</th>
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**COM A SCALE**

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<thead>
<tr>
<th>Best verbal response</th>
<th>Orientated</th>
<th>Conversation disorganised</th>
<th>Inappropriate words</th>
<th>Incomprehensible sounds</th>
<th>None</th>
</tr>
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**Best motor response**

<table>
<thead>
<tr>
<th>Obey commands</th>
<th>Localise pain</th>
<th>Flexion to pain</th>
<th>Flexion abnormal</th>
<th>Extension</th>
<th>None</th>
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**GCS Score**

- Eyes closed by swelling: $C$
- Endotracheal tube or tracheostomy: $T$
- Usually record the best arm response
Traumatic Brain Injury

Indications for surgery:

- Focal lesion(s) surrounding oedema and midline shift
- Clotting deficiencies
- Cross-matched blood
- Exploratory burr holes!
- Compound depressed skull fracture
- Severely depressed fracture
- CSF otorrhoea and rhinorrhoea.
- Neurological status
Guideline for the Management of Severe Traumatic Brain Injury

3rd Edition

Brain Trauma Foundation
American Association of Neurological Surgeons,
Congress of Neurological Surgeons,
Joint Section on Neurotrauma and Critical Care.
Traumatic Brain Injury

Management:

- BP < 90 mmHg systolic should be avoided
- Hypoxia Pa O2 < 60mmHg or O2 saturation < 90% should be avoided
- Hypothermia?
- Antiepileptic Drugs: Dilantin
Use of Steroids: **NO**

Hyperventilation: PaCO2 ~30-34 mmHg

Avoid: 1st 24hrs or <25mmHg

IV fluid: **NaCl**
Traumatic Brain Injury

Use of Mannitol:

- Severe head injury
- Buy time during transfer
- Up to 1g/kg of intravenous mannitol (20% solution) over 30 minutes to reduce associated cerebral edema.
- Rebound phenomena
- Hyper tonic saline
Indications: GCS < 8
Unable to monitor
Types:
Readings:
Decompressive Craniectomy
Traumatic Brain Injury

Management:

Decompressive Craniectomy
Traumatic Brain Injury

Outcome:

Moderate head injury (GCS: 9-12): fare less well.
  50% good recovery
  25% moderate degree of disability.
  15% severe degree of disability
  7-10% Death or a persistent vegetative state

Severe head injury (GCS: 3-8): worst outcomes.
  20% good outcomes.
  20% moderate disability
  20% severe disability
  40% Death or a persistent vegetative state
Traumatic Brain Injury
Rehabilitation:

- Expedite Recovery
- Outpatient therapy
- Constant vigilance to prevent problems; joint mobility, skin integrity, respiratory status etc.
Traumatic Brain Injury

Prevention:

- Speeeeeeed
- Automobile seatbelts and child restraints
- Air bags ?
- Helmets by cyclists
Thank you