HEAD INJURIES

Dr. Mohammed altamimi
• Pathophysiology
• Local effects on brain tissue
• Systemic effects of TBI
• Management protocols
• Patient triage and classification
• mechanism
• Types of head injuries
**Triage: the following criteria suggest severe injury and urgency in care**

<table>
<thead>
<tr>
<th>Revised Trauma Score¹</th>
<th>Injury Pattern</th>
<th>Injury Mechanism</th>
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</thead>
<tbody>
<tr>
<td>Glasgow Coma Score &lt;13</td>
<td>Penetrating injury to head, neck chest, abdomen or groin</td>
<td>Fall &gt;6 metres (c. 20 feet)</td>
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<td>Systolic Blood Pressure (sBP) &lt;90 mmHg</td>
<td>Two or more proximal long bone fractures</td>
<td>Ejection of patient from a vehicle</td>
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<td>Respiratory Rate (RR) &lt;12 or &gt;29 bpm</td>
<td>Flail chest</td>
<td>Roll-over of vehicle</td>
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<td>Burn &gt;15% TBSA* or facial, airway or chest involvement</td>
<td>Death of occupant in same car</td>
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<td>Victim = pedestrian or motorcyclist</td>
<td>Rearward displacement of front axle</td>
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<td>History of ingestion of alcohol or drugs</td>
<td>Passenger compartment intrusion</td>
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</tbody>
</table>
• The Primary Survey and Resuscitation
• proceed in parallel
• A — Clear the Airway. Use chin-lift or jaw-thrust. Immobilize the cervical spine with collars, bags and tape until cleared.
• B — Check Ventilation. Administer oxygen at 15 litres per minute with tight-fitting mask with reservoir or use bag, valve and mask.
• C — Check for pulses, skin perfusion and consciousness. Identify obvious sources of blood loss;
• D — Assess the level of consciousness with A.V.P.U.:
  • A (alert); V (responds to verbal communication);
  • P (responds only to pain); U (unconscious);
• E — Expose and examine the patient thoroughly.
<table>
<thead>
<tr>
<th>Airway</th>
<th>Breathing</th>
<th>Circulation</th>
<th>Disability</th>
<th>Exposure</th>
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</thead>
<tbody>
<tr>
<td>Is the airway compromised?</td>
<td>Ventilate by bag and mask or tube, with oxygen.</td>
<td>Arrest obvious bleeding. Pre-empt shock with 2 large bore i.v. cannulae.</td>
<td>Assess level of consciousness with AVPU (see D. above)</td>
<td>Undress completely. Avoid hypothermia.</td>
</tr>
<tr>
<td>If ineffective, intubate with endotracheal or naso-tracheal technique.</td>
<td>The oximetry reading should be 95% or greater, on supplemental oxygen.</td>
<td>Drain any significant haemopericardium.</td>
<td>Can the patient move all 4 limbs?</td>
<td>Insert NG tube and urinary catheter. Beware of contraindications!</td>
</tr>
<tr>
<td>If unable, consider cricothyroidotomy.</td>
<td>Use cutdown or intraosseous lines if necessary.</td>
<td>Use cutdown or intraosseous lines if necessary.</td>
<td>Is there evidence of peripheral nerve disruption or upper motor neurone/intracranial injury (e.g. asymmetry of movement or up going plantar – Babinski or thumb – Hoffman-reflexes).</td>
<td>Blood specimens: plasma chemistry, arterial blood gases, FBC, and cross-match.</td>
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<td></td>
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<td></td>
<td>Get a urine sample (for urinalysis and toxicology etc).</td>
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</table>
• The Secondary Survey

• Recheck A, B, C and D. Proceed to next stage if the patient is stable and analgesia has been effective. The secondary survey is a head-to-toe examination of the patient’s body.

• A.M.P.L.E. - a simple mnemonic for key information

• A: allergies (e.g. penicillin or aspirin)

• M: medication (e.g. a beta-blocker or warfarin)

• P: previous medical history (e.g. previous surgery or anaesthetic mishap)

• L: last mealtime (i.e. drink versus major meal)

• E: events surrounding the incident (e.g. fell 5 metres with immediate loss of consciousness)
# Examine each body region meticulously

<table>
<thead>
<tr>
<th>Head</th>
<th>Face</th>
<th>Neck</th>
<th>Chest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Examine the eyes, and check for basal skull fracture.</td>
<td>Exclude mid-facial fracture or airway obstruction.</td>
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<thead>
<tr>
<th>Neurological</th>
<th>Abdomen</th>
<th>Orthopaedic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimate GCS* repeatedly (e.g. every 15 minutes). Perform a full CNS examination, in the co-operative patient.</td>
<td>Peritoneal and retro-peritoneal injuries may present with normal signs.</td>
<td>Check for limb or spinal malalignment, crepitus, and tenderness. Examine the back.</td>
</tr>
<tr>
<td>Falling GCS levels require urgent CT scan and neurosurgical consultation.</td>
<td>CT, ultrasonography or occasionally diagnostic peritoneal lavage may help in excluding abdominal injury.</td>
<td>Search for minor fractures, dislocations, glass or other foreign bodies.</td>
</tr>
</tbody>
</table>
• INITIAL MANAGEMENT OF SEVERE TRAUMA

• Most widely accepted system is that recommended in the American College of Surgeons’ Advanced Trauma Life Support (ATLS) course. The key elements of this approach are the primary survey/resuscitation phase, the secondary survey and the rapid implementation of definitive treatment, which may involve early surgery. The objective is to detect life-threatening conditions as quickly as possible, to stabilise the patient and to start definitive treatment as early as possible, in a prioritised fashion.
Intensive management of traumatic brain injury (roles and objectives)

- The primary focus of intensive management is directed towards patients suffering severe head injury (GCS of 8) following initial resuscitation and to prevent those suffering moderate injuries from deteriorating into unresponsive coma.

- It is universally agreed that a population of neurons is irreversibly damaged at initial impact but neuronal demise continues unchecked until brain resuscitation is initiated.

- It is also agreed that primary brain injury initiates a cascade of metabolic derangement affecting the cerebral vascular system, glial supporting cells, and neurons, which leaves brain vulnerable to secondary injury.
Head injury

Primary impact

Hypoxia
- Brain stem dysf.
- Ventilation prob.
- O2 delivery prob.

ischemia
- Raised ICP
- Vasoconstriction
- SOL

Traumatic brain (Secondary injury)
- Ion pumps dysfunction
- Deranged autoregulation
- Oedema (vsogenic, c. toxic)

acidosis
Herniation syndromes
**Systemic effect of T.B.I**

- **Cholinergic dysfunction**
  - bradycardia, hypotension

- **Adrenal hyperactivity**
  - MI
  - neurogenic pulmonary edema

- **Hypothalamic dysfunction**
  - DI
  - SIADH
  - CSWS
  - hypersecretion of ACTH, GH prolactin

- **Brain stem dysfunction**
  - apnoea, hypoventilation
  - Bradycardia
  - hypotension, hypertention
  - decrease COP
  - arrhythmias

increased secretion of catecholamines and enkephaline, cytokines leading to hypermetabolism, hyperglycemia hypercatabolism and organ dysfunction (lung, liver, heart, gut) through lactic acidosis
MECHANISMS OF INJURY

- inertial or contact mechanisms

- Inertial injuries are commonly called "acceleration" or "deceleration" injuries

- Contact injuries are commonly called coup & contrecoup injuries.
A concussion is a violent jarring or shaking that results in a disturbance of brain function.
Figure 1-16. Axonal shearing may occur in acceleration as well as deceleration injuries. The nerve fiber may be stretched or completely severed, producing the manifestations of diffuse head injury.
Initial management of acute closed head injury

**at accident site**
- Triage, resuscitation of ABCD
  - (35% incidence of hypoxia
    - (Apnea > 10 min carries high mortality)
- Controlled ventilation to maintain paco2 30-35mmhg
  - GCS less than 8 needs intubation and MVA
- Crystalloid solution infusion and treatment of hypotension (15% incidence of hypotension)
  - presence of one episode of shock > 3 min (mostly hypovolemic) increases mortality by up to 50%
- minimum of syst bp of 100mmhg to maintain adequate cerebral perfusion

**emergency room**
- Systemic approach
  - GCS and B S reflexes
- Management protocols
  - Immediate intubation and CT brain
  - lateral cervical xray, Cxr. us abd, pelvic xr. DPL for all severe injured
  - Surgery if CT shows a midline shift (Manitol if FND before CT)
  - Ur cath, NG, laboratory tests

**in the ICU**
- .cardio-respir. Parameters monitoring
  - .Controlled ventilation
  - .PEEP
  - .hyperventilation
  - .Tracheostomy
  - .Chest toilet, physiotherapy
  - .Neuromuscular paralysis monitoring
  - .B gasses, electrolytes monitoring
  - .Antiepileptics
  - .Raised intracranial pressure treatment
<table>
<thead>
<tr>
<th>GCS</th>
<th>6</th>
<th>5</th>
<th>4</th>
<th>3</th>
<th>2</th>
<th>1</th>
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<tbody>
<tr>
<td><strong>Best motor response</strong></td>
<td>Obey command</td>
<td>Localize the pain</td>
<td>Withdrawal to pain</td>
<td>Flexion to pain</td>
<td>Extention to pain</td>
<td>none</td>
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<tr>
<td><strong>Verbal response</strong></td>
<td>oriented</td>
<td>confused</td>
<td>Inappropriate word</td>
<td>Incomprehensible sound</td>
<td>none</td>
<td></td>
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<tr>
<td><strong>Eye opening</strong></td>
<td>Spontaneous</td>
<td>To verbal</td>
<td>To pain</td>
<td>none</td>
<td>none</td>
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<td>Low risk group</td>
<td>Moderate risk group</td>
<td>High risk group</td>
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<td>Asymptomatic</td>
<td>History of change of consciousness</td>
<td>Depressed level of consciousness not due to drugs or other causes</td>
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<td>Head ache</td>
<td>History of progressive headache</td>
<td>Focal signs</td>
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<td>Dizziness</td>
<td>Alcohol or drug intoxication</td>
<td>Decreasing LOC</td>
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<td>Scalp hematoma</td>
<td>Unreliable or inadequate history</td>
<td>Penetrating skull injury</td>
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<td>Scalp laceration</td>
<td>Age less than 2 years</td>
<td>Palpable depressed fracture</td>
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<td>Absence of moderate or high risk criteria</td>
<td>Post traumatic seizure or vomiting or amnesia</td>
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<td></td>
<td>Multiple trauma</td>
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<td>Serious facial injury</td>
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<td></td>
<td>Signs of basal fracture</td>
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<td>Possible skull penetration</td>
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<td>Depressed fracture</td>
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<td></td>
<td>Child abuse</td>
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**Observation alone**

- Observation for high risk signs
- Consider CT
- Neurosurgical consultation

**Consider urgent CT & Neurosurgical consultation & examination**
Scalp Injuries

1. Injuries severe enough to damage scalp may also damage the underlying skull
2. A complete examination of the whole patient is mandatory

Layers of scalp are:
- **S** Skin
- **C.T** C.T.
- **A** Aponeurotic (Galila)
- **L** Areolar tissue
- **P** Periosteum
Types

- Lacerations
- Avulsions
- Physical injuries
- Chemical injuries
Scalp Lacerations

- Small and linear either superficial one (bleeder) or deep one (less beeder) mostly closed under L.A in 2 layers (Galia and skin)

- Stellated and large either superficial one (bleeder) or deep one (less beeder) mostly closed under G.A especially in children, extensive debridement is unnecessary, all devitalised tissue should be derbided.
Reconstruction ladder

1. primary suturing in simple lacerations, sharp, without tissue loss and gross contamination

2. secondary intention in wounds with minimum tissue loss

3. debridement and a split thickness graft in wounds with extensive tissue loss and intact pericranium

4. local flaps in wounds with extensive tissue loss and stripped pericranium and a split thickness graft for donor area (if a defect is left)

5. Pedicellar flaps can be used if local flaps are not sufficient especially for occipital areas and small defects

6. Free tissue transplant is used for anterior and large areas
Scalp swelling and hematomas

1. **Caput succedaneum**

2. **Cephal hematoma**

3. **Subgial hematomas**
Skull fractures classification

1. by pattern (according to amount of energy, ratio of impact force to surface area)
   - Comminuted
   - Depressed
   - Linear
   - Diastatic
   - Basal fractures
   - Growing

2. by type
   - Opened
   - Closed

3. by anatomic location
   - Basal
   - convexity
Treatment roles

• **Comminuted** (replacement as a cranioplasty if not infected with treatment of other associated injuries)

• **Linear fractures** (no treatment but observation if closed, debridement if infected and compound)

• **Depressed** (replacement as a cranioplasty if not infected, otherwise good debridement and craniectomy)

• **Basilar skull** fracture (observation for two days, avoid irrigation of the nose or ear, avoid probing, detailed auditory and vestibular examination is performed at 6 weeks interval)

• **Frontal sinus fracture** (if in the posterior wall cranialization is necessary)
**Types of traumatic intracranial lesions**

- **Focal** (epidural hematoma - subdural hematoma - brain contusion - intracerebral hematomas - focal subarachnoid hematoma)

- **Diffused** (subarachnoid hemorrhage - diffused axonal injury - concussion)
Traumatic intracranial hematomas

• **Acute subdural**

• accelerate high speed impact
• tearing of bridging veins
• bleeding from cortical vessels, venous sinuses
• acute brain trauma may coexist
• altered level of consciousness and focal neurological deficit are common
• CT and rapid evaluation are necessary
• If no signs of rapid deterioration or progressive neurological deficit, no mass effect so observation and control of intracranial p. is necessary, otherwise surgery is the role)
Acute Subdural Hematoma

Another example of acute subdural hematoma with a midline shift (noncontrast CT)
• **Chronic subdural**

- Mostly in those over 50 years old
- ½ of patients have got no history of trauma
- If there is any history of trauma, it is trivial
- Alcoholism, epilepsy, coagulopathy are common
- Dementia is common presentation
- In minimal neurological deficit, medical management is the role
- If not successful, deterioration of neurological picture so surgery usually by burr hole evacuation.
Subacute Subdural Hematoma

Noncontrast CT—note the clot appears less dense in this subacute subdural hematoma.
Extradural hematomas

- Mostly resulting from meningial vessel tear (arteries > Veins > sinuses)
- Fractures are common associated injury
- Severe associated brain injury is rare
- Level of consciousness is variable
- If with mass effect must be evacuated within ½ hour
- if small follow up is recommended
- Prognosis depends on level of consciousness at time of presentation
Cerebral contusions

- Small and deep one needs follow up
- Large and with mass effect needs lobectomy
- Large one may herniate as late as 9 days post trauma
- Level of consciousness depends on size of contusion and location
SAH—more examples

- Subarachnoid hemorrhage in the right sylvian fissure
SAH—more examples

Blood in the sulci

Edema causing a midline shift
Posterior fossa hematomas

- Usually arise from venous sinuses hemorrhage
- Deterioration in level of consciousness is rapid and signs of brain stem and long tract compression are common initial finding
- Urgent evacuation is the role
Penetrating head injuries

- Sonic waves and cavitations and decavitation and secondary insults are the injurious mechanisms.
- Infection rates are high.
- Injury far away from site of entrance and exit is common.
- Control of homodynamic state is the initial management.
- Surgical interventions are limited to debridement and removal of mass hematomas and for selected cases.
- Prognosis is usually bad.
Questions

• What are the types of skull fractures
• What are the parameters of GCS
• Enumerate the types of traumatic scalp swellings
• Enumerate the signs and symptoms of raised intracranial pressure
• What are the types of traumatic intracranial hemorrhages
• Right about types of mechanisms of head injury
• What is the pathophysiology of head injury
THANK YOU