Assessment and Management of Head and Spinal Cord Injuries

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Assessment and Management of Head and Spinal Cord Injuries

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A. Head Injury

Case Study
An 18-year-old boy fell and hit his head against the goal post when he was tackled playing football. He was drowsy initially after the fall but soon regained full consciousness and was able to complete the match. However, he started getting headache soon after and hence was taken to the Emergency Department. Whilst waiting to be assessed, he collapsed suddenly and became unrousable.

- What type of head injury has this boy probably sustained?
- What is the term used to describe his fluctuating level of consciousness?
- What will the CT Brain most likely show?

Introduction
The majority of head injuries are a consequence of road traffic accidents, assaults, injuries at home, workplace or during sports. Excessive alcohol consumption is frequently implicated and young males are most commonly involved.

Epidemiology
Head injuries are a major cause of morbidity and mortality in the community. In the UK, it has been estimated that between 200 and 300 per 100,000 of the population are admitted to hospital with head injuries. Of these, 9 per 100,000 are fatal. In Malaysia, head and neck injuries resulting from road traffic accidents account for 85% of major trauma cases, of which approximately 60% require admission to intensive care unit.

Pathophysiology of Head Injuries
The brain is a very vulnerable organ encased in a rigid protective skull and cushioned by cerebrospinal fluid (CSF). Trauma to the brain can occur via translational acceleration or deceleration forces, direct focal sharp penetrating or blunt forces. In abrupt deceleration, injuries can be divided into coup and countercoup. A coup injury results from trauma at the site of impact whereas countercoup injury is the resulting remote injury away from the site of impact (i.e., the force of injury propelling brain parenchyma to hit the opposite interior surface of the skull). This is why most cerebral contusions occur without skull fractures. On the contrary, patients with skull fractures that look significant usually only sustain minor neurological dysfunction as most of the force is absorbed by the skull after an impact.

Indications for CT Scan
The advent of CT scanning has had a huge impact on the treatment for traumatic brain injury. It is rapid, non-invasive and allows identification of surgically treatable lesions (acute injury) as well as pathological chronic injury.

The following are the criteria for immediate request for CT scan of the head in adults as recommended by the NICE (UK) clinical guideline:

a. GCS less than 13 on initial assessment in the emergency department.
b. GCS less than 15 at 2 hours after the injury on assessment in the emergency department.
c. Suspected open or depressed skull fracture.
d. Any sign of basal skull fracture (haemotympanum, ‘panda’ eyes, cerebrospinal fluid leakage from the ear or nose, Battle’s sign).
e. Post-traumatic seizure.
f. Focal neurological deficit.
g. More than one episode of vomiting.
h. Amnesia for events more than 30 minutes before impact

A patient with minimal external signs of injury who is fully alert & orientated with a normal neurological examination and no symptoms other than headache may not need a CT scan. However, they do need close observation for the next 24 hours.

Neurological assessment
Head injuries require thorough on going assessment of the patient. The AVPU scale is a quick and easy method to assess level of consciousness as shown in Box 1:

<table>
<thead>
<tr>
<th>Eye Opening Response</th>
<th>Spontaneous-opens with blinking at baseline</th>
<th>4 points</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Opens to verbal command</td>
<td>3 points</td>
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<tr>
<td></td>
<td>Opens to pain</td>
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<td></td>
<td>None</td>
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<tr>
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<td>Oriented</td>
<td>5 points</td>
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<tr>
<td></td>
<td>Confused conversation, but able to answer questions</td>
<td>4 points</td>
</tr>
<tr>
<td></td>
<td>Inappropriate response, words discernible</td>
<td>3 points</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible speech</td>
<td>2 points</td>
</tr>
<tr>
<td></td>
<td>None</td>
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</tr>
<tr>
<td>Motor Response</td>
<td>Obey commands for movement</td>
<td>6 points</td>
</tr>
<tr>
<td></td>
<td>Purposeful movement to painful stimulus</td>
<td>5 points</td>
</tr>
<tr>
<td></td>
<td>Withdraws from pain</td>
<td>4 points</td>
</tr>
<tr>
<td></td>
<td>Abnormal (spastic) flexion, decorticate posture</td>
<td>3 points</td>
</tr>
<tr>
<td></td>
<td>Extensor (rigid) response, decerebrate posture</td>
<td>2 points</td>
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<tr>
<td></td>
<td>None</td>
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Box 1: The AVPU scale

A more objective way of recording a patient’s state of consciousness is by using the Glasgow Coma Scale (GCS). This is usually performed in the ‘Disability’ component of the primary survey once airway, breathing and circulation of the patient have been secured. Table 1 describes the GCS scale in detail.

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Table 1: GCS scale
Scalp laceration
The scalp has a rich blood supply in the dense fibrous layer (Figure 1) and hence severe blood loss can result from a scalp laceration. In infants, blood loss from scalp can actually lead to hypovolemic shock. Although scalp lacerations or bruising confirms the presence of a head injury, their absence does not exclude an underlying intracranial haematoma.

Scalp lacerations should be repaired in two layers, with opposition of the galea prior to closure of the skin. This should be done after meticulously debriding the wound. Routine use of antibiotics is not indicated in a clean wound but may be required in a dirty or contaminated wound.

Figure 1: Scalp laceration

Base of skull fracture
Diagnosis is made based on clinical evidence as it is often occult radiologically. Signs of base of skull fracture include periorbital haematomas (‘Raccoon eyes’), mastoid haematoma (Battle’s sign), anosmia, otorrhoea, rhinorrhoea, or VII and VIII cranial nerve palsies. On CT scan, there is presence of pneumocranium or air fluid levels in the sinuses. Prophylactic antibiotics may help in preventing meningitis. Nasal secretions post head-injury should be screened for beta transferrin (‘tau’ protein) for CSF. To prevent further leakage due to fistulas, surgery may be required.

Box 2
In elderly, alcoholic and patients on anti-coagulant medications, even minor head trauma can result in devastating intracranial bleeding.

Intracranial haemorrhage
Bleeding within the skull is a life-threatening emergency. Brain damage resulting from accumulated blood volume which in turn increases intracranial pressure (ICP) can lead to permanent neurologic deficit or death. Intracranial haemorrhages (ICH) can be classified into:

a. Subdural hematoma
b. Extradural hematoma
c. Subarachnoid haemorrhage

Subdural Haematoma
This results from tearing of bridging veins between the cerebral cortex and draining venous sinus. They are classified into:

- acute <24hours
- sub-acute 24hours – 2weeks
- chronic >2weeks

Acute subdural haematomas are associated with high-velocity trauma and thus have a poor outcome. The blood follows the subdural space over the convexity of the brain and appears as a concave hyperdense collection (Figure 2). Acute subdural haematoma are rapid evolving lesions and early evacuation is mandatory.

Chronic subdural haematomas are most common in infants and elderly. They present with progressive neurological deficit after trauma.

CT appearance varies depending on the age of the lesion. In the acute phase (Days 0-3), the lesion appears hyperdense. As it liquefies (Days 3-14), the lesion is isodense or hypodense as a result of fibrinolysis occurring within the clot. Chronic subdural hematomas, older than 2 weeks, are usually hypodense.
SDH collections can either resolve or increase in size from osmotic effects or repeated bleeds. These clots are evacuated by drilling burrholes over the skull.

**Extradural Haematoma**
Extradural haematoma (EDH) is the accumulation of blood between the dura matter and skull, typically occurring after significant blunt head trauma. Fractures of the temporal bone can disrupt the middle meningeal artery and dural venous sinuses leading to high-pressure bleeding within the cranial vault. The potential space between the dura and bone is developed by the expanding haematoma taking on the convex lens configuration inside the cranium (**Figure 2**). Cushing’s triad describes the physiologic response to the rapidly increasing intracranial pressure and imminent brain herniation.

EDH is more likely to occur in younger age groups as the dura is able to strip more readily off the underlying bone. Patients will have brief loss of consciousness often accompanied by a lucid interval, headache, drowsiness, dizziness, nausea and vomiting. Rapid clinical deterioration is a significant criterion. An extradural haematoma is a surgical emergency where an urgent craniotomy and decompression is required.

**Subarachnoid haemorrhage**
Subarachnoid haemorrhage (SAH) is bleeding in the area between the brain and the thin tissues that cover the brain (subarachnoid space). Subarachnoid haemorrhage can be caused by bleeding from an arteriovenous malformation (AVM), bleeding disorders, cerebral aneurysms and use of anti-coagulants.

The main symptom is a severe headache that starts suddenly and is typically occipital or unilateral. Patients often describe it as the “worst headache ever” or also known as ‘thunderclap headache’ which reaches maximum intensity within seconds. Patients may experience photophobia, agitation, drowsiness or become comatose.

Acute bleeding in SAH appears bright in CT scans (**Figure 3**). Blood is usually seen in the ventricles, sulci and cisterns. The overall sensitivity of CT is best within the first 12 hours. Sensitivity declines with time. Approximately seven percent of acute SAH will not be visualized on initial head CT, typically because there is a small volume of bleeding. CSF analysis from a lumbar puncture can help with the diagnosis even if the CT scan is normal.

The principal goal of treatment is to prevent re-bleeding by surgical clipping or endovascular techniques. Without treatment, re-bleeding occurs in 50% of patients with ruptured aneurysm within six months.

**Box 3**
The features of Cushing’s triad include:
- Hypertension
- Bradycardia
- Abnormal respiratory patterns

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![Figure 2: SDH on CT scan](image1)

![Figure 3: EDH on CT scan](image2)
require nursing home, hospital care or other institutionalised settings rather than their own homes. Around 20% of patients leave Spinal Cord Injury Centres clinically depressed.

Common mechanism
The spinal cord can be injured by transection, distraction, compression, bruising, haemorrhage, or ischaemia of the cord or by injury to blood vessels supplying it. These injuries can all result in permanent cord injury and may be complete or incomplete.

Presentation
A complete cord syndrome is characterized clinically as complete loss of motor and sensory function below the level of the traumatic lesion.

Incomplete cord syndromes have variable neurologic findings with partial loss of sensory and/or motor function below the level of injury. These include the anterior cord syndrome, the Brown-Séquard syndrome and the central cord syndrome.

Signs & symptoms of acute spinal cord trauma
- Flaccid paralysis below level of injury
- Loss of spinal reflexes below level of injury
- Loss of sensation (pain, touch, proprioception, temperature) below level of injury
- Loss of sweating below level of injury
- Loss of sphincter tone with bowel and bladder dysfunction

Assessment
History
A high index of suspicion of spinal cord injury is necessary in any major accidents, unconscious patients, falls from a height, sudden jerk of neck after rear end car collision, facial injuries or head injuries. Enquire about neck or back pain, numbness, tingling, weakness and ability to pass urine.

Examination
A logroll is performed to assess the patient’s spine. Inspect for bruising then palpate for spinal deformity or tenderness. Repeat the neurological examination to determine neurological damage (complete/incomplete) and its progression. Perform rectal examination to assess anal tone. Thorough overall examination for fractures at other sites must be performed to rule out other distracting injuries.

Complete neurological examination
The aims include:
- To determine level of lesion - counted as the lowest level at which neurological function is intact bilaterally

B. Spinal Cord Injury

Case Study
A 23-year-old lady is brought to the Emergency Department after a road traffic accident. She was the driver of a 3-door hatchback car that was hit from the back by a lorry whilst driving to her workplace. On examination, she is noted to have bruising over her anterior chest wall and tenderness at the upper cervical region. Her GCS is 15 and other physiological parameters are stable. Further neurological assessment reveals weakness in her upper limbs. PR examination is normal.

- What type of spinal cord injury she may have sustained?
- What is the best form of imaging which may be used to diagnose her condition?
- What drug(s) should be given to her during the initial phase of injury?

Introduction
Due to the increase in road traffic accidents, unfortunately, spinal cord injuries have become more common in the modern society. Despite advances in the understanding of the pathogenesis and improvements in early recognition and treatment, it remains a devastating injury, often producing severe and permanent disability. With the peak incidence in young adults, traumatic spinal cord injury leads to widespread impact to the society and the economy.

Epidemiology
In the UK every year, there are around 1,200 people paralysed from spinal cord injuries. There are currently thought to be approximately 40,000 people in the UK living with paralysis. This may be a conservative estimate as this only takes into account patients who have been treated in a specialist spinal cord injury unit and does not include those who have been treated in a district general hospital. It is estimated that the current annual cost of caring for people paralysed by spinal cord injury is more than £500 million. Twenty-one percent of people discharged from Spinal Cord Injury Centres
To determine whether damage is complete or incomplete  
To determine prognosis  
This may be difficult until period of spinal shock (flaccidity, areflexia) is over i.e., 24-48 hours after injury.

**Imaging**  
**X-rays**  
- Cervical spine: AP, lateral including C7/T1, open mouth view of odontoid, Swimmer’s view or pull arms down view.  
- AP and lateral view of other tender areas of spine.

**Box 5**  
Period of spinal shock usually resolves within 48 hours and return of bulbocavernous reflex signals termination of spinal shock.

**CT scan**  
- Used to show bony injuries  
- Provides better visualization of vertebral arches, facet joints and neural canals.  
- Differentiation of neural elements from other soft tissues requires intrathecal administration of contrast medium.

**MRI scan**  
To show soft tissue involvement. Any extramedullary compression by disc, haematoma and bone may also be readily apparent.

**Management**  
**Initial management**  
If there is presence of neurological damage:  
- Catheterise  
- Take note of reduced blood pressure and bradycardia due to neurogenic shock (temporary generalised sympathetic)  
- Rule out hypotension due to haemorrhage elsewhere  
- The patient may need treatment with vasopressors, not fluid resuscitation  
- Invasive monitoring is required.

Give intravenous methylprednisolone (solumedrol) to reduce inflammation and preserve blood flow to the spinal cord. The dosage regimen is as follows:  
- 30 mg/kg over 15 min

**Definitive management**  
The objectives are to preserve neurological function and relieve reversible nerve or cord compression. This is done by stabilizing the spine with surgery via posterior instrumentation or fusion (Figure 4). After surgery, patients need to undergo rehabilitation as part of the recovery process.

**Box 6**  
Use of high-dose methylprednisolone therapy for spinal cord injury patients remains controversial. Clinicians should carefully weigh the potential benefits versus the risks of this treatment.

**Figure 4:** Posterior instrumentation of L3 - L5

**Conclusion**  
Head and spinal cord injuries result in significant morbidity and mortality. Since it commonly affects young individuals, the resulting paralysis and permanent brain damage has a devastating impact socially and economically. Prompt recognition, careful stabilisation and referral to the appropriate specialist unit is thus essential to enhance the possibility of a successful functional outcome.
References:


Laparoscopic Colorectal Surgery Course & Master Class
(a course endorsed by the Royal College of Surgeons of Edinburgh)

22nd and 23rd April 2013 | Prince Charles Hospital, Merthyr Tydfil, Wales

Course Convenor: Professor. P.N. Haray

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