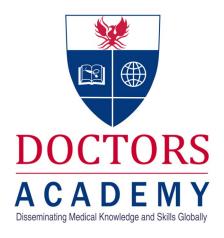
Assessment and Management of Head and Spinal Cord Injuries

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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 become 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 fatal¹. 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 unit⁹.

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 countercoup³. 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 guideline²:

- 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, Neurological assessment 'panda' eyes, cerebrospinal fluid leakage from the Head injuries require thorough on going assessment of 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.

the patient. The AVPU scale is a quick and easy method to assess level of consciousness as shown in **Box 1**:

A lert		
Responds to V oice		
Responds to P ain		
U nconscious		

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.

Eye Opening Response	Spontaneous-opens with blinking at baseline	4 points
	Opens to verbal command	3 points
	Opens to pain	2 points
	None	1 point
Verbal Response	Oriented	5 points
	Confused conversation, but able to answer questions	4 points
	Inappropriate response, words discernible	3 points
	Incomprehensible speech	2 points
	None	1 point
Motor Response	Obeys commands for movement	6 points
	Purposeful movement to painful stimulus	5 points
	Withdraws from pain	4 points
	Abnormal (spastic) flexion, decorticate posture	3 points
	Extensor (rigid) response, decerebrate posture	2 points
	None	1 point

Table 1: GCS scale

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Top

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 skin³. 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 haematomas 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 lesion⁴. 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 haematoma is a surgical emergency where an urgent osmotic effects or repeated bleeds. These clots are craniotomy and decompression is required. evacuated by drilling burrholes over the skull.



Figure 2: SDH on CT scan

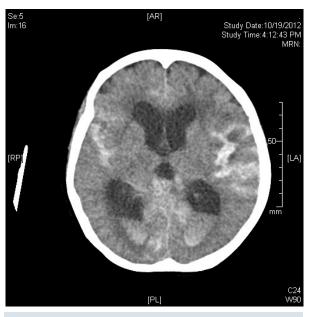
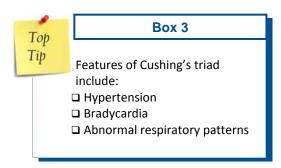


Figure 3: EDH on CT scan

Extradural Haematoma

Extradural haematoma (EDH) is the accumulation of Subarachnoid haemorrhage (SAH) is bleeding in the area artery and dural venous sinuses leading to high-pressure malformation (AVM), bleeding within the cranial vault¹. The potential space aneurysms and use of anti-coagulants. between the dura and bone is developed by the expanding haematoma taking on the convex lens The main symptom is a severe headache that starts configuration inside the cranium (Figure 2). Cushing's suddenly and is typically occipital or unilateral. Patients triad describes the physiologic response to the rapidly often describe it as the "worst headache ever" or also increasing intracranial pressure and imminent brain known as 'thunderclap headache' which reaches herniation.

EDH is more likely to occur in younger age groups as the become comatosed. 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, Rapid clinical dizziness, nausea and vomiting. deterioration is a significant criterion. An extradural



Subarachnoid haemorrhage

blood between the dura matter and skull, typically between the brain and the thin tissues that cover the occurring after significant blunt head trauma. Fractures brain (subarachnoid space). Subarachnoid haemorrhage of the temporal bone can disrupt the middle meningeal can be caused by bleeding from an arteriovenous bleeding disorders, cerebral

> maximum intensity within seconds⁶. Patients may experience photophobia, agitation, drowsiness or

> 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 bleeding⁵. 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 months³.





Box 4

Prior to obtaining consent for a lumbar (LP), patients puncture should be warned of post-LP headache as it is common

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
- ☐ 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 $\ \Box$ To determine level of lesion - counted as the lowest injury is more than £500 million. Twenty-one percent of people discharged from Spinal Cord Injury Centres

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 syndrome⁸.

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 asses 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:

level at which neurological function is intact bilaterally



- □ To determine whether damage is complete or □ Then 5.4mg/kg/hour over 23 hours or 48 hours incomplete
- □ To determine prognosis

This may be difficult until period of spinal shock (flaccidity, areflexia) is over i.e., 24-48 hours after injury.

depending on time since injury i.e. given over 23 hours(if presentation is within 3 hours since injury) or given over 48 hours(if presentation is between 3 to 8 hours since injury)

Imaging

X-rays

Top

Tip

- 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 bulbocavernosus 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 sympathectomy)
- ☐ 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 ☐ Cervical spine: AP, lateral including C7/T1, open 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 Top Tip o f U s e high-dose methylprednisolone therapy for spinal cord injury patients remains controversial. Clinicians should carefully weigh the potential benefits versus the risks of this treatment⁸.



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.



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