



Queensland Trauma Education

TRAUMATIC BRAIN INJURY

Management of closed head injury

Case discussion

Facilitator resource kit

Queensland Trauma Education

The resources developed for Queensland Trauma Education are designed for use in any Queensland Health facility that cares for patients who have been injured as a result of trauma. Each resource can be modified by the facilitator and scaled to the learners needs as well as the environment in which the education is being delivered, from tertiary to rural and remote facilities.

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Queensland Trauma Education

Traumatic Brain Injury – Management of closed head injury: Case discussion – Facilitator resource kit

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About this training resource kit

This resource kit provides healthcare workers with the knowledge on how to effectively assess and manage a closed head injury following a traumatic incident.

National Safety and Quality Health Service (NSQHS) Standards



Target audience

Emergency department medical and nursing clinicians.

Duration

30 minutes.

Group size

Small or large group participation.

Learning objectives

By the end of this session the participant will be able to:

- Demonstrate an understanding of the medical and surgical management of mild/moderate/severe traumatic brain injury (TBI).
- Describe the differing patient disposition and features of patients with mild/moderate/severe TBI.
- Describe the appropriate follow up of mild/moderate/severe TBI.

Facilitation guide

1. Facilitator to divide group into 3 and provide vignette to each group – mild/moderate/severe.
2. Facilitator to use cases and questions to lead discussion around assessment and management.
3. Facilitator to refer to supporting documents and incorporate into case discussion to support clinical decisions around management.

Supporting resources (in Printable Resources)

1. Abbreviated Westmead PTA Scale (A-WPTAS) incorporating Glasgow Coma Scale (GCS).
2. How to use the Abbreviated Westmead Post Traumatic Amnesia Scale (A-WPTAS).

Case discussion

Case study 1

17 yo M brought into the emergency department following a head knock at rugby training. He was witnessed to be briefly unconscious, with no seizure activity and orientated on review by the Rugby team medical officer within 5 minutes of injury. On review in the emergency department he is complaining of a mild headache, no visual changes and has no neurological deficits.

Case study 2

32 yo F is brought into the emergency department after being involved in an alleged assault with another person. She was hit once to the back of the head and pushed to the ground. On arrival of the ambulance she was confused, agitated, moving limbs on command, pupils 3mm and reactive bilaterally. In the emergency department 1 hour post injury she remains confused to date, year and place. She opens her eyes to pain and is obeying commands.

Case study 3

72 yo M is brought into the emergency department from his home after a trip and fall on the wet footpath whilst taking the rubbish bins out. He was found by neighbours after an unknown time on the ground. On arrival to the emergency department he is withdrawing to pain in the R UL and extending to painful stimuli on the L UL. There is no verbal response and no eye response to painful stimuli. Both pupils are 5mm and sluggishly reactive.

Question and answer guide

1. What constitutes mild/moderate/severe TBI?

GCS score and localising neurological signs including: pupillary abnormalities, focal neurological deficits, lateralising weakness.

Mild	Moderate	Severe
<ul style="list-style-type: none"> • GCS: 13-15 • Loss/alteration of consciousness: < 30min • Amnesia: < 24hrs • Imaging: negative 	<ul style="list-style-type: none"> • GCS: 9-12 • Loss/alteration of consciousness: > 30min, < 24hrs • Amnesia: > 24hrs, < 7days • Imaging: transient changes 	<ul style="list-style-type: none"> • GCS: < 8 • Loss/alteration of consciousness: > 24hrs • Amnesia: 7days • Imaging: positive, lasting abnormalities

2. How would you initially categorise the severity of head injury in the above cases and what is your rationale?

1. GCS 15 - Mild head injury. Will require ongoing GCS assessment to accurately classify head injury.
2. GCS 13 - Mild head injury. Serial GCS assessment to monitor for deterioration.
3. GCS 5 - Severe head injury. Will require early treatment and neuroprotective management.

3. In mild TBI what additional assessment can aid severity classification?

- a. An Abbreviated Westmead Post Traumatic Amnesia Scale (A-WPTAS) - see supporting document.
- b. How to use A-WPTAS - see supporting document.
- c. A-WPTAS is an outpatient assessment tool and differs from the inpatient tool BRISC and 3 day PTA assessment.

4. What is a neuroprotective medical management strategy?

Strategy	Rationale	Management
Intubation and oxygenation	Avoid: <ul style="list-style-type: none"> Aspiration. Secondary brain injury from hypoxaemia. 	<ul style="list-style-type: none"> Early RSI performed by most experienced operator. Ketamine to avoid hypotension and cerebral hypoperfusion. Initial FiO₂ 1.0 and titrate to maintain Sats > 95% (avoid hyperoxia for prolonged periods).
Ventilation	Avoid: <ul style="list-style-type: none"> Hypo and hypercarbia to reduce risk of cerebral vasoconstriction (ischaemia) and vasodilation (hyperaemia of damaged brain tissue). 	<ul style="list-style-type: none"> Early use of ETCO₂ until invasive arterial access obtained for PaCO₂ – target 35-40mmHg Aggressive hyperventilation with hypocapnia is only used as a rescue strategy i.e. patient coning, needing definitive surgical care.
Haemodynamic management	<ul style="list-style-type: none"> Maintain adequate cerebral perfusion pressure (CPP). CPP=MAP-ICP. 	<ul style="list-style-type: none"> Fluid bolus (may opt for hypertonic saline if haemodynamic instability), blood/blood products, vasopressors for MAP > 80mmHg (avoid if non-compressible bleeding likely) until ICP monitoring insitu then aim for CPP 50- 60mmHg.
Sedation and paralysis	<ul style="list-style-type: none"> Optimise ventilation and oxygenation. Optimise cerebral metabolic rate (CRMO₂), cerebral blood flow (CBF). Reduce ICP by reducing patient movement. 	<ul style="list-style-type: none"> Sedation and analgesia titrated to desired effect via continuous infusion (morphine/ midazolam). Paralysis agent via initial bolus, may need continuous infusion (vecuronium/rocuronium) to follow.
Osmotherapy	<ul style="list-style-type: none"> Osmotic agent: gradient created to remove water from normal brain tissue therefore reducing ICP. 	<ul style="list-style-type: none"> Intravenous Mannitol 1g/kg or 5ml/kg of 3% hypertonic saline. See MNHHS RBWH Medication protocol: 000263 Hypertonic Saline (sodium chloride 3%), Traumatic Brain Injury http://hi.bns.-health.qld.gov.au/RBH/policies/000263.pdf NB. Mannitol may cause hypotension as it acts as osmotic diuretic.
Patient positioning	<ul style="list-style-type: none"> Head up 30 degrees to reduce ICP by augmenting venous outflow. 	<ul style="list-style-type: none"> Ensure patient alignment (neutral head position) and spinal immobilisation is maintained and tilt whole bed 30 degrees. Loose ETT ties or opt for other methods of securing ETT. Ensure soft cervical collars are loosened to avoid compressing jugular veins.

5. What additional TBI management should be considered?

- **Glucose:** Strict glucose monitoring to maintain normal range to avoid hypoglycaemia as may worsen brain injury.
- **Corticosteroids:** Not recommended in TBI as associated with increased mortality rate.
- **Anticonvulsants:** Optimise seizure prevention to reduce risk of further brain injury. Use of phenytoin or levetiracetam for 7 days – no evidence of benefit (severe TBI at risk of seizures are typically on midazolam/propofol infusions).
- **Temperature control:**
 - Hypothermia - patients > 45years do worse with hypothermia.
 - > 45years aim for normothermia.
 - Hyperthermia - increases neuronal cell death when temp > 39 degrees.
 - If temp > 39 degrees, cool the patient until normothermic.
- **Erythropoietin (EPO):** No effect from Hb > 100mg/dL in neurological outcomes, ongoing trials (EPO-TBI) for prevention of secondary injury.
- **Barbiturate coma:** Decreases cerebral metabolic rate. However, can cause hypotension and has long half-life.
- **Deep vein thrombosis (DVT) prophylaxis:** Chemical can usually be instituted day 2-3 post injury (discuss with neurosurgeons).
- **Nutrition:** Feed early as high metabolic demand.

Surgical - Moderate/Severe TBI

a. **Burr hole** - 'Management of the rapidly deteriorating TBI patient' outlines the burr hole procedure including the flowchart for management and escalation. (<https://trauma.reach.vic.gov.au/guidelines/traumatic-brain-injury/the-rapidly-deteriorating-tbi-patient>)

b. **Craniotomy.**

c. **Decompressive Craniotomy (DECRA)** - In adults with severe diffuse traumatic brain injury and refractory intracranial hypertension, early bifrontotemporoparietal decompressive craniectomy decreased intracranial pressure and the length of stay in the ICU but was associated with more unfavourable outcomes. (Decompressive Craniectomy in Diffuse Traumatic Brain Injury April 21, 2011, N Engl J Med 2011; 364:1493-1502 <https://www.nejm.org/doi/full/10.1056/NEJMoa1102077>)

Medical - ongoing assessment

- a. Appropriate discharge advice and information.
- b. Outpatient followup if required.

Acronyms and abbreviations

Term	Definition
A-WPTAS	Abbreviated Westmead Post Traumatic Amnesia Scale
GCS	Glasgow coma scale
CPP	Cerebral perfusion pressure
ICP	Intracranial pressure
MAP	Mean arterial pressure
FiO2	Fraction of inspired oxygen
ETCO2	End tidal carbon dioxide
PaCO2	Partial pressure of carbon dioxide
RSI	Rapid sequence induction
CRMO2	Cerebral metabolic rate
CBF	Cerebral blood flow

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