Queensland Trauma Education

TRAUMATIC BRAIN INJURY

Elderly patient

Case discussion

Facilitator resource kit

Clinical Excellence Queensland
JAMIESON TRAUMA INSTITUTE
CSDS Clinical Skills Development Service
Queensland Government

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 – Elderly patient: Case discussion – Facilitator resource kit
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About this training resource kit

This resource kit provides a learning framework for the assessment and management of the elderly patient with traumatic brain injury.

National Safety and Quality Health Service (NSQHS) Standards

Target audience

Emergency department nursing clinicians.

Duration

20 minutes.

Group size

Suited to small group participation.

Learning objectives

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

1. Understand the injury profile leading to traumatic brain injury
2. Define important anatomical and physiological parameters in TBI
3. Discuss neuroprotective measures
4. Use guidelines to manage anticoagulation reversal

Facilitation guide

1. Use case discussion to highlight learning objectives
2. Incorporate local resources where available

Supporting documents (in Printable Resources)

The following supporting documents are provided for this case discussion:

1. Canadian CT rule infographic
2. Monroe-Kellie Doctrine infographic
3. Cerebral Blood Flow diagram

Case discussion

Case study

A 76-year-old man in brought to your ED after falling in the garden at home. His son has driven him to ED, and he has walked into triage.

He reports he was gardening when he slipped on wet grass and fell striking his head against the retaining wall. It was unwitnessed. He is unsure if there was any loss of consciousness, with amnesia to events. He has a 4 cm occipital laceration with has ongoing ooze despite pressure applied by his family.

His PMHx includes AF and a mechanical aortic valve replacement. He is on warfarin and atenolol. He has no allergies.

He denies any other injuries.

Question and answer guide

1. **What are the injury mechanisms for older patients who sustain head trauma? (1)**

Falls 57%

MVC 14%

Assaults 11%

2. **How common are head injuries in older adults' post fall? (2)**

Common! 28% of injury presentations to hospital had a head injury as the primary site injured.

3. **What features on history or examination suggest serious injury post head trauma in adults?**

Increasing age

Use of anticoagulant therapy

Loss of consciousness

Confused or altered behaviour

Neurological deficits

Prolonged seizures

Prolonged headache and repeated vomiting

Signs of base of skull or facial fractures

Cushing's reflex

4. **What is the Canadian CT head rule?**

It is a validated clinical decision rule to identify adult patients who need a CT brain with a mild head injury.

Importantly patients are excluded if they are taking anticoagulant medication.

Some centres may have an abridged version incorporating New Orleans criteria (headache, vomiting, age >60, ETOH/drugs, amnesia, trauma above clavicles and seizure).

5. How does the GCS (Glasgow Coma Scale) help define the severity of brain injury?

The GCS was first used in trauma as part of ATLS in 1980! (It was adopted for use in Subarachnoid Haemorrhage grading in 1988.)

Scores are given for best motor, eye and verbal response.

In trauma- the motor score is most predictive for neurological outcome and therefore highlighted in reporting (eg GCS 10 with motor score 5).

GCS 3-8 (severe TBI), 9-12 (moderate TBI) and >13 (mild TBI).

The inclusion of pupillary response can add to the prediction of outcome with bilateral absence of pupillary reaction indicating a more severe TBI.

6. What is neuroprotection and who needs it?

Neuroprotection is the term given to a collection of therapies aimed at reducing intracranial pressure and reduce the secondary insult. They are often implemented in a stepwise fashion but may be instituted concurrently if signs of raised intracranial pressure is present (as demonstrated by Cushing's reflex- bradycardia, hypertension and bradypnoea or focal neurological signs- pupillary inequality and dilatation, rapidly falling GCS or localising neurological deficits). The aim of instituting neuroprotection is to increase the time to access definitive surgical intervention.

7. What are the elements of neuroprotection?

The mainstay of neuroprotection is to avoid hypoxia and hypotension.

This is achieved by:

- Early intubation to titrate FiO2, and control CO2 (low normal 35-40mmHg)
- Head up 30 degrees to allow venous drainage (can tilt whole bed)
- Sedation and muscle relaxation (paralysis) to reduce brain metabolism
- Maintain blood pressure (SBP > 100 and MAP > 65)- Assumption of raised ICP and need to maintain CPP (cerebral perfusion pressure = MAP-ICP)
- Add osmotherapy if localising sign (3% hypertonic saline 5ml/kg or Mannitol 1g/kg)

Consideration will then be given to the need for surgical intervention- EVD or decompressive craniectomy depending on the type of intracranial injury.

8. Why do we only target low normal CO2?

When the CO2 is lowered significantly (below 30mmHg) the blood vessels in the brain will vasoconstrict. This can initially cause reduction in blood flow and to a lesser effect reduce blood brain volume (reduction of each mmHg CO2 will result in cerebral blood flow by 3%), temporarily reducing intracranial pressure. In time, reduction of cerebral blood flow will result in brain hypoxia from hypoperfusion. There are animal studies which suggest that hypocapnia can reduce the seizure threshold, which is detrimental in TBI.

Importantly the relationship between PaCO2 and ICP is not linear, and with prolonged hyperventilation the effect on ICP will be reduced.

Therefore a CO2 target of 30-35mmHg should only be used in a temporary fashion if the patient has signs of coning to allow time to definitive neurosurgical care.

9. What about management of anticoagulation in TBI?

Intracranial haemorrhage is a life-threatening condition. If the patient is taking anticoagulation this should be 'reversed' if able. Senior decision makers in conjunction with patient and family discussion should consider indication for anticoagulation, severity of intracranial haemorrhage and acute resuscitation plans to determine the reversal chosen. The anticoagulation medication should be withheld/ceased in addition to specific reversal agents.

Warfarin: life threatening bleeding plus INR >1.5 = Vit K 5-10mg IV, FFP 300mls and Prothrombinex 50units/Kg IV.

Apixiban/Rivaroxiban: life threatening bleeding = no specific antidote. Vit K 10mg IV (if nutritionally deplete), Prothrombinex 50units/kg IV, TXA 1g IV, possible role for Novoseven (DW haematology).

Dabigatran: life threatening bleeding = consider haemodialysis, consider Vit K 10mg IV (if nutritionally deplete), Idarucizumab (Praxbind) 5g IV.

Dalteparin/Enoxaprin: Protamine sulfate (approximately 60% effective)- dose will depend on time since administration.

Acronyms and abbreviations

Term	Definition
ICP	Intracranial Pressure
CPP	Cerebral Perfusion Pressure
TBI	Traumatic Brain Injury
MVC	Motor vehicle collision
TXA	Tranexamic Acid

References

1. Head injuries in Australia 2020–21, Causes of head injury - Australian Institute of Health and Welfare (aihw.gov.au)
2. Injury in Australia: Falls - Australian Institute of Health and Welfare (aihw.gov.au)

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