Traumatic Brain and Spinal Injury

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A 25 year old adult had alleged history of bike skid. On arrival to the emergency department, he was found to be unconscious with bleeding from the scalp. Patient had history of vomiting twice. His pulse rate was 60/min and blood pressure was 140/80 mmHg. The pupillary size showed asymmetry and his breathing was laboured.

Traumatic brain injury (TBI) is the leading cause of mortality and morbidity in children and young adults in both developed and developing nations worldwide. The aims and objectives of its management are prompt management of intracranial hypertension and secondary brain injury, maintenance of cerebral perfusion pressure, and ensuring adequate oxygen delivery to injured brain tissue.

Step 1: Initial Assessment/Components of Primary Survey

Airway Control and Ventilation

Airway, breathing and circulation takes precedence in spite of obvious head injury.

- Secure cervical spine with a cervical collar: The unstable cervical spine injury can occur in 5–6% cases of the TBI. Risk factors include a motor vehicle collisions, assaults, falls and a GCS less than 8.
- In suspected cervical spine injury, orotracheal intubation and ventilation with 100% oxygen along with manual in—line cervical immobilization with cervical

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collar off to reduce the chance of worsening a neurological injury until radiological clearance is obtained.
- Prevent hypoxemia: Avoid PaO_2 less than 60 mmHg or O_2 saturation below 90%.
- Consider Rapid Sequence Intubation. Succinylcholine or rocuronium may be used as muscle relaxant. Although succinylcholine may produce a small increase in ICP, this has not proven to be clinically significant. To facilitate intubation an opiate such as fentanyl (1 μg/kg) may be used, there is no evidence to support the use of lidocaine during intubation.
- Adequate sedation and muscle relaxation tends to reduce the cerebral metabolic oxygen requirement ($CMRO_2$), optimize ventilation and prevent coughing or straining.
- Choice of sedative agent: Anaesthetic drugs that allow for rapid control of the airway while avoiding an increase in intracranial pressure (ICP) and providing hemodynamic stability are preferred. Propofol and thiopental are the most commonly used drugs, but they may cause hypotension. Etomidate has advantages in terms of cardiovascular stability, but the possibility of adrenal suppression exists. Ketamine is popular in trauma patients and recent evidence suggests that its effect on ICP may be limited
- Ventilator strategy:
  - Hypoventilation should be avoided, as increased PCO_2 levels may lead to cerebral hyperemia with an increase in blood volume and ICP.
  - Hyperventilation, on the other hand, results in an increased risk of vasoconstriction and increased tissue hypoxia, especially in the penumbra zone, so it is best avoided. CO_2 level should be kept in low normal zone by the use of End Tidal CO_2 monitor in most intubated patient. Ventilator should be adjusted to achieve a PaO_2 of ~60 mmHg, which can oxygenate the penumbra zone. High PaO_2 should be avoided considering the risk of hyperoxic cerebral vasoconstriction. PEEP of 5–10 cmH_2O may be administered to prevent atelectasis and has been proven to be safe in these patients
  - Hyperventilation up to a $PaCO_2$ between 32 and 36 mmHg for the purpose of reducing ICP is recommended for a brief period of time to avoid brain herniation.

Blood Pressure and Cerebral Perfusion

Pressure (CPP)

- Maintain systolic blood pressure >100 mmHg in patients with 50–69 years old, and >110 mm Hg in patients 15–49 years or >70 years old.
- In a hypotensive TBI patient, hypovolemia resulting from non cranial hemorrhage should be ruled out.
- Choice of fluid: Hypotonic solutions like 5% dextrose should be avoided. Isotonic normal saline is the most common crystalloid used in TBI patients, but Ringer's

lactate or other balanced crystalloids are an alternative and result in less acute kidney injury. Infusion of large volumes of normal saline results in adverse hyperchloremic metabolic acidosis that is detrimental in TBI. On the other hand balanced crystalloids are relatively hypotonic and may exacerbate cerebral edema.

- Colloids appear to provide no further benefit.
- Saline is preferable to albumin resuscitation as the later has been found to increase mortality in TBI patients
- The recommended target CPP (MAP-ICP) value for survival and favourable outcomes is between 60 and 70 mmHg.
- Vasopressors are commonly used to augment CPP in the setting of TBI but avoid aggressive attempts to maintain CPP above 70 mmHg with fluids and pressors.
- Labetolol is the drug of choice for control of hypertensive emergency in TBI.

Step 2: Secondary Survey/Neurological Assessment

Neurological Assessment

- Glasgow coma scale (GCS) has been the most widely used method of recording the level of consciousness in patients at presentation and at subsequent assessment. A score of ≥13 correlates with a mild brain injury, 9–12 is a moderate injury, and ≤8 a severe brain injury.
- Patients with a Glasgow Coma Scale ≤13 and moderate to severe extracranial anatomical injuries should be rapidly transferred to the higher level of care.
- TBI can be classified based on severity and morphology (Table 11.1)

Table 11.1 Classification of TBI

Based on severity (GCS)	Mild	13–15	
	Moderate	9–12	
	Severe	<9	
Based on morphology	Skull fractures	Vault	Linear vs. stellate/compound
			Depressed/nondepressed
		Basilar	With/without CSF leak
			With/without cranial nerve palsy
	Intracranial lesions	Focal	EDH, SDH Intracerebral
		Diffuse	Hypoxic/ischemic injury Diffuse axonal injury Multiple contusion Concussion

Step 3: Do Imaging—CT Scan

Indications of CT in TBI

- CT scan brain should be carried out in all moderate to severe TBI.
- For mild TBI CT scan indications are:
  - Open or depressed skull fracture
  - Sign of basilar skull fracture
  - Vomiting more than 2 episodes
  - Age >65 years
  - Anticoagulant use
  - Seizure

Step 4: Monitoring of ICP and Measures to Reduce ICP

ICP Monitoring and Management

- ICP monitoring is important, but it does not replace careful neurological and radiological examination. ICP should be monitored in patient with GCS of 3–8.
- ICP should also be monitored in patient with severe traumatic head injury with normal CT scan if patient is above 40 years, unilateral or bilateral motor posturing, or systolic pressure less than 90 mmHg
- Patients with elevated ICP have been shown to have worse outcomes and are at a higher risk of mortality.
- Management of severe TBI patients based on ICP monitoring may reduce in hospital and 2-week post-injury mortality;
- Clinical judgement should be used to initiate intracranial monitoring in patients who are at a high risk of clinical deterioration.
- It is recommended to treat ICP > 22 mmHg to reduce mortality.

Initial measures of raised ICP include head of bed elevation, keeping neck in neutral position appropriate sedation and analgesia, osmotherapy and removal of CSF.

Sedation and Analgesia

- Sedatives and analgesics can affect outcomes in head-injured patients.
  - Adequate pain control and sedation can be used as initial measures to control raised ICP.
  - Short-acting agents such as fentanyl, midazolam, or propofol are preferred for frequent neurological assessments. (Table 11.2). High dose barbiturates are recommended to control ICP refractory to maximum standard surgical and medical treatments while ensuring hemodynamic stability.

Table 11.2 Drugs and doses of sedatives and analgesics

Commonly used sedatives	
Fentanyl	2 mcg/kg test dose, 2–5 mcg/kg/h continuous infusion
Midazolam	2 mg test dose, 2–4 mg/h continuous infusion
Sufentanil	10–30 mcg test bolus, 0.05–2 mcg/kg continuous infusion
Propofol	0.5 mg/kg test bolus, 20–75 mcg/kg/min continuous infusion (not to exceed 5 mg/kg/h)

Hemodynamic stability is essential before and during barbiturate therapy. Although propofol may be used for ICP control, it is not recommended for improvements in mortality or 6-month outcomes

- Care should be taken to maintain an adequate mean arterial pressure throughout the duration of sedation. Minimising sedation duration helps in decreasing Incidence of delirium and helps in early mobilization

Start Osmotherapy

- Osmotherapy with mannitol or hypertonic saline has been used since many years but controversy remains regarding which solution is the best agent and regarding the best method of administration.
- Mannitol is used more often as intermittent boluses (0.25–1 g/kg). It should be stopped if serum osmolality exceed 320 mOsm/L. It should be avoided in hypovolemia and renal failure patients. Hypertonic saline as intermittent boluses of 3% 250 mL over half an hour or 30 mL of 23.4% can also be used. It should be witheld if sodium exceeds 160 mEq/L. Serum sodium and osmolality must be assessed every 6 h.

Decompressive Craniectomy

- Decompressive craniectomy is a surgical procedure that involves removal of a large section of the skull. Craniectomy reduces ICP by giving extra space to the swollen brain, and it may quickly prevent brainstem herniation.
- Decompressive craniectomy may be a life-saving surgery, but it comes at the expense of higher chances of severe disability among survivors.
- Guidelines recommend a large frontotemporoparietal decompressive craniectomy, as opposed to a smaller one, to target reduced mortality and better neurological outcomes.

Step 5: Advance Multimodal Neuromonitoring (Tool to Monitor CPP if Resource Available)

- Identification of the range of autoregulation following TBI to provide individualized CPP therapy may be a means to improve outcome and is made possible by newer monitoring devices.

- Jugular venous oxygen saturation ($SjvO_2$): Used to estimate the balance between global cerebral oxygen delivery and uptake. Both reduction in $SjvO_2 < 50\%$ and $SjvO_2 > 75\%$ after TBI are associated with poor outcomes. Monitoring of $SjvO_2$ following TBI may lead to improved outcomes.
- Brain tissue oxygen tension: $PbrO_2$ represents the balance between oxygen delivery and cellular oxygen consumption. $PbrO_2$ provides a highly focal analysis of brain milieu and may be used to monitor the potentially salvageable penumbra following TBI. Normal values are between 35 and 50 mmHg. Following TBI, reduced levels of $PbrO_2$ (<5–10 mmHg) have been seen to be associated with poorer outcomes.
- Cerebral microdialysis: Increasingly used as a bed side tool to provide analysis of brain homeostasis in the intensive care setting. Severe ischemia is usually associated with significant increases in the lactate/pyruvate ratio (>20–25) and is associated with poor outcomes following TBI.

Step 6: Pharmacotherapy

Anticonvulsant Therapy

- Posttraumatic seizures are a major cause of secondary brain injury following TBI, and are associated with higher injury severity and worse outcomes. Seizures occur in up to 20% of patients with TBI. These seizures are usually nonconvulsive in nature and cannot be detected clinically and EEG monitoring (preferably continuous) is needed for this.
- This should be clinically suspected if consciousness impairment is disproportionate to the severity of injury
- Phenytoin or levetiracetam (500–1000 mg every 12 h) is effective in decreasing the rate of early posttraumatic seizures in the first 7 days of injury, but has no significant role in prevention of posttraumatic seizures after the first week of injury.
- Patients with TBI who develop any seizures will require prolonged antiseizure medications.

Role of Steroids

- No benefit in lowering ICP or improvement in patient outcome has been shown through the use of high-dose corticosteroids in acute TBI.
- The use of methylprednisolone in patients with moderate to severe TBI has been demonstrated to increase mortality and is contraindicated.

Antibiotic Therapy

- Since TBI patients are more likely to receive invasive monitoring and therapeutic treatments, including mechanical ventilation, they are also more likely to be at increased risk for the development of infections.
- Sources of potential infections need to be identified and appropriate therapy should be instituted. A common source of infection is invasive monitoring of ICP. The incidence of ICP device infection has been reported to range from 1% to 27%.
- The current guidelines suggest the use of antibiotic-impregnated catheters to reduce infection rates.
- Prophylactic antibiotic should be avoided

Role of Tracheostomy

- Early tracheostomy (preferably percutaneous) should be performed to reduce ventilation days in patients with anticipated prolonged ventilation and/or need for airway protection
- The goals of treatment including clinical, laboratory and monitoring parameters are summarized in Table 11.3

Table 11.3 Goals of treatment

Clinical	Systolic BP	≥100 mmHg, avoid hypotension
Laboratory	Temperature	36–38°, avoid hyperthermia
	Hb	≥7 g/dL
	Glucose	140–180 mg/dL
	INR	≤1.4
	pH	7.35–7.45
	$PaCO_2$	35–45, never less than 25 mmHg
	PaO_2	≥100 mmHg, avoid hypoxemia
	Na	135–145
	Platelet	≥75,000
Monitoring	CPP	≥60 mmHg, 60–70 mmHg
	ICP	<22, 5–15 mmHg
	$PbtO_2$	≥15
	SPO_2	≥95%

No role of prophylactic antibiotic and anticonvulsant. No role of steroid

Step 7: Supportive Care and ICU Bundle

Glycemic Control

- Prevention of hyper- and hypoglycemia Glucose-containing fluids should be avoided and blood sugar monitored to maintain levels between 140 and 180 mg/dL

Nutrition

- Early nutritional support is associated with better outcomes and early enteral feeding has been found to be beneficial.
- Calculated or measured caloric replacement (100–140% of basal expenditure) should be started early and full goal should be reached by 5–7 days
- Post pyloric feeding may also be used reduce the risk of ventilator associated pneumonia.
- Patients with severe TBI have gastric feeding intolerance, Prokinetic agents, such as metoclopramide, may improve feeding tolerance

Temperature Management

- Avoidance and aggressive treatment for fever should be instituted and normothermia should be maintained.
- Prevention of hyperthermia: In clinical practice, even mild hyperthermia has been associated with poorer outcomes and longer ICU stays, as it is may lead to increased brain edema and inflammation.
- Use of Therapeutic hypothermia is controversial.

Thromboprophylaxis

Intermittent pneumatic compression stockings should be used (except in lower limb injuries) and continued till the patient is ambulatory

- Low-molecular-weight heparin (e.g. enoxaparin 40 mg s.c) or low-dose unfractionated heparin (5000 s.c. 3 times a day) should be used in combination with mechanical prophylaxis when it is safe, preferably after 48–72 h of intracranial hemorrhage/craniotomy with close monitoring and repeat NCCT head to detect expansion of hematoma.

Coagulopathy Management

- Coagulation parameters and platelet count should be routinely monitored and if deranged should be corrected in patients with bleeding manifestation or requiring neurosurgery
- Rapid reversal is best attained with Activated prothrombin complex concentrate

- Antiplatelets and anticoagulants should be stopped and there effects reversed if clinically indicated
- **Stress ulcer prophylaxis**: early enteral nutrition, H_2 Blockers or PPI, physiotherapy, and Skin/Eye care.
- **Proper postdischarge care**

Step 8: Identify Complications of TBI

Complications of TBI

1. Trauma induced coagulopathy
2. ARDS/Negative pressure pulmonary oedema
3. Paroxysmal sympathetic hyperactivity/Stress cardiomyopathy
4. Hypothalamic-pituitary-adrenal dysfunction/SIADH/Cerebral salt wasting/DI
5. Hydrocephalus
6. Heterotopic ossification
7. Spasticity
8. Chronic traumatic encephalopathy/Post traumatic headache and depression/Cognitive impairment
9. GI and GU complications
10. Gastric ulceration and DVT

Step 9: Outcome Measures

- Three tools commonly used to measure outcome after TBI are Functional Independence Measure (FIM), Glasgow Outcome Scale (GOS) and Disability Rating Scale (DRS).

Step 10: Prognosis

- Very difficult and complex
- As a general rule patients with GCS < 8 have a 30% risk of mortality
- Patient who remain in vegetative state or minimally conscious state have a poor chance of meaningful recovery
- Recovery with functional independence or partial dependence may occur in >50% of severe TBI over a period of years, if initial aggressive management is pursued
- Individual risk factors for poor outcome are Low GCS score (specially GCS motor score), increasing age, bilaterally absent pupillary light reflex, associated injuries, abnormal CT scan, hypotension, hypoxemia, elevated ICP, reduced CPP, bleeding diathesis, pyrexia.
- CT findings associated with poor prognosis: Absence or compressed basal cisterns, tSAH, presence and degree of midline shift (CT severity) and presence of abnormalities in initial CT.

Traumatic Spine Injury

- Spine injury, with or without neurological deficits, must always be considered in patients with multiple injuries. Approximately 5% of patients with brain injury have an associated spinal injury, whereas 25% of patients with spinal injury have at least a mild brain injury and injury to limbs and viscera
- Approximately 55% of spinal injuries occur in the cervical region producing quadriparesis, 15% in the thoracic region, 15% at the thoracolumbar junction, and 15% in the lumbosacral area. Up to 10% of patients with a cervical spine fracture have a second, non-contiguous vertebral column fracture.
- Early management should incorporate a full Advanced Trauma Life Support (ATLS) assessment with the intent to avoid hypotension, bradycardia, and hypoxia

Manage Traumatic Spinal Injury Patient

1. Early intubation and mechanical ventilation is recommended for patients with high cervical injuries (C1–C5).
2. All trauma victims with suspected cervical spine injury should have cervical spine immobilised until an unstable fracture has been ruled out.
3. All patients with suspected cervical spine injury should have complete spinal imaging by X-ray or CT scan
4. Urgent neurosurgical consultation
5. Mean arterial pressure (MAP) augmentation with norepinephrine (if needed) is recommended for at least the first 72 h following injury to a maximum of 7 days. Goal MAP ≥85 mmHg for blunt/incomplete penetrating injury. Goal MAP ≥65 mmHg for complete penetrating injury
6. Use of high-dose methylprednisolone is not recommended routinely (not after 8 h of onset of injury). Even if it used, it should be within 8 h of onset of injury and in isolated non penetrating spinal cord injury as a 30 mg/kg IV bolus followed by an infusion of 5.4 mg/kg/h for 23 h
7. Early (definition of early not standardized ranging from <8 h to <72 h) neurosurgical decompression of acute spinal cord compression is recommended.
8. Venous thromboembolism prophylaxis should be initiated within first 72 h of injury.
9. Consider early (definition of early not standardized) tracheostomy in high cervical injury (C1–C5) patients.

Rehabilitation should be offered to all patients.

ICU Bundle Care

Nutrition, bowel care (as patient may develop neurogenic bladder and require urinary catheterization), skin care and bed sore prevention, psychological support, thromboprophylaxis, ulcer prophylaxis, treatment of spasticity and neuropathic pain are the supportive care required in spine injury patient.

Suggested Reading

Advanced Trauma Life Support®. Student course manual. 10th edn. Library of Congress Control Number: 2017907997. ISBN 78-0-9968262-3-5. 2018. *A reference manual on trauma life support for beginners*

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