CASE REPORT

Traumatic brain injury and the evidence for its management

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SUMMARY

A 29-year-old man presented to a major trauma centre with traumatic brain injury. Following cranial decompression, the patient was admitted to the intensive care unit for medical management and monitoring. This case report reviews the evidence for the management of traumatic brain injury.

BACKGROUND

Traumatic brain injury (TBI) is a key cause of death and disability in young adults and is becoming more prevalent.¹ While advances in prehospital care have reduced all-cause mortality following major trauma, a reduction in mortality following TBI has not occurred since 1994.¹ Intracranial pressure (ICP) and cerebral perfusion pressure (CPP) monitoring represents 'gold-standard' care, but its use in guiding patient management is controversial.² The study presents a case of TBI and reviews the literature to understand the rationale for ICP monitoring and its role in patient management.

CASE PRESENTATION

A 29-year-old man presented to a major trauma centre by land ambulance having sustained a witnessed fall from the third floor. On arrival at the emergency department (ED), he was combative and vomiting. Owing to his agitated behaviour, the ambulance crew had been unable to immobilise his cervical spine (C-spine) and place the patient onto a hardboard.

His airway was initially managed with simple airway manoeuvres. He was placed in the lateral position and secretions suctioned. His Glasgow Coma Scale (GCS) was 12 (eyes 3; verbal 3; motor 6). His pupils were reactive bilaterally (2+/2+). The decision was made to perform a rapid sequence induction in order to protect his airway and safely perform investigations. The patient was preoxygenated and induced with 50 mg propofol, 150 µg fentanyl and 100 mg rocuronium. Cricoid pressure was applied and a size 8 endotracheal tube placed to 21 cm at the lips and secured. He was transferred directly from the ED resuscitation room to the CT scanner.

INVESTIGATIONS

► A CT of the head and neck (figure 1) was performed in order to assess for intracranial blood causing mass effect. This revealed an extensive fracture running through the right parietal and squamous temporal bone into the central skull base. There was an extensive

subdural haematoma overlying the right cerebral convexity with subarachnoid blood (figure 2). A moderate midline shift of approximately 10 mm was evident. Contrecoup haemorrhagic contusions were located within the left inferior temporal lobe. Right-sided uncal herniation in keeping with cerebral oedema and raised ICP was apparent. There was no evidence of fracture-dislocation to the C-spine.

- A CT intracranial angiogram and a CT of the aortic arch and carotid arteries were performed as deceleration injury to the cerebrovascular arteries required exclusion (figure 3). The calibre of the right internal carotid artery within the carotid canal and cavernous sinus was slightly reduced and the vessel irregular. The appearances were suspicious of a dissection flap. However, flow was not limited and the intracranial vessels opacified normally (figure 3).
- A CT of the chest, abdomen and pelvis was performed to exclude the presence of either solid or hollow organ injury. This excluded a haemothorax/pneumothorax and the heart and mediastinal vessels opacified normally. No intra-abdominal injury was identified but multiple thoracic spine fractures with mild-to-moderate wedge compression were evident in T3, T5 and T9.

Figure 1 This plane CT scan of the head reveals an acute traumatic subdural haematoma within the right frontoparietal convexity. Subdural collections are typically crescent shaped and are limited by dural reflections such as the falx cerebri. The brain is oedematous with loss of sulci and gyri.

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Figure 2 This plane CT reveals a fracture within the right temporal bone with overlying cutaneous haematoma. Subarachnoid blood is demonstrated by the hyperattenuating material within the lateral ventricles.

DIFFERENTIAL

Traumatic brain injury

This patient has presented to the ED with a reduced GCS, vomiting and combative behaviour. These clinical features are highly suggestive of an intracranial injury. The principle diagnoses are an extradural haematoma, subdural haematoma or a subarachnoid haemorrhage. Extradural haematomas are commonly

Figure 3 This CT intracranial angiogram demonstrates the intracranial vascular anatomy. The middle cerebral arteries course laterally and are united by the anterior communicating artery. Posterior cerebral arteries opacify normally. The Circle of Willis is completed by the posterior communicating arteries.

associated with skull fractures and are caused by the tearing of a meningeal artery or dural venous sinus. Blood collects between the dura and skull causing compression of brain tissue and transtentorial herniation. Subdural haematomas are caused by bleeding from bridging veins that cross the subdural space. Subdural collections occur commonly following an assault or fall. Subarachnoid blood collects within the subarachnoid space between the arachnoid and pia mater. Hypoxic brain injury resulting in permanent disability and hydrocephalus caused by occlusion of cerebrospinal fluid (CSF) circulation are common sequelae.

Blunt visceral injury

A fall from a height can result in deceleration injury to the mediastinal vasculature, particularly at the junction of the mobile aortic arch and fixed descending aorta. This can lead to dissection or transection of the vessel. Haemothoraces/pneumothoraces are potentially fatal conditions which require rapid evaluation and treatment. Solid intra-abdominal organs are particularly vulnerable following falls. The liver and spleen are susceptible to rupture leading to blood loss.

Pelvic and spinal fractures

The rigid bony pelvis is vulnerable to anteroposterior, lateral compression and vertical shear fracture. Owing to the close proximity of the iliac vessels and presacral venous plexus, haemorrhage is a major cause of death from displacement of the pelvic ring. Confirmation of pelvic stability is mandatory in those who have fallen from a height. Long bone fractures and spinal injury should also be excluded.

TREATMENT

Surgery—right decompressive craniectomy

The patient underwent an emergency decompressive craniectomy, minimal right parietal lobectomy and ICP bolt insertion. The surgical site was prepared and the trauma flap raised. On inspection, the brain appeared swollen and tight. The dura was incised and the clot evacuated. Following evacuation of the clot, the brain continued to swell mandating extension of the craniectomy anteriorly by 2–3 cm. Extension of the craniectomy permitted brain swelling and the reduction of ICP via the Monro-Kellie hypothesis. The wound was closed and an ICP bolt placed over the left frontal lobe. The pressure on insertion was 17 mm Hg.

Neuroprotective measures

The patient was transferred directly to the intensive care unit (ICU) for medical management and monitoring. His ICP and CPP were controlled by ventilatory and pharmacological strategies. Over 5 days, his sedation was weaned and he was extubated in order to assess his neurological function.

OUTCOME AND FOLLOW-UP

On waking from sedation, the patient had a dense left-sided hemiplegia and aphasia. His GCS off all sedation was 11 (eyes 4; verbal 1; motor 6). He remained in the ICU for several weeks and required a tracheostomy to facilitate suctioning of secretions and prevent aspiration pneumonia. Following removal of the ICP bolt, he had a further CT scan of the head in order to plan a cranioplasty. After a prolonged stay, the patient was stepped down to the neuromonitored ward for ongoing physiotherapy and rehabilitation.

DISCUSSION

TBI can be subdivided into primary and secondary injury. Primary injury results from an external force disrupting intracranial structures. The intensity and duration of these forces determine the extent of the damage, which may include shearing of white matter tracts, contusions, haematomas and swelling. Secondary brain injury evolves following the primary insult. Cerebral metabolism is impaired causing anaerobic glycolysis and the accumulation of lactic acid.³ Membrane permeability and cerebral oedema results. Deregulation of cerebral blood flow (CBF) and neurotransmitter release causing excitotoxicity, free-radical generation and mitochondrial dysfunction.

The principle of ICP monitoring is to maintain adequate cerebral perfusion and oxygenation to meet metabolic demands.⁴ Raised ICP reduces CPP and CBF, which exacerbates secondary injury.⁴ Several studies have shown that a high ICP is strongly associated with poor outcome and may lead to brainstem pressure, herniation, respiratory depression and death.⁴ Level I and level II evidence for ICP monitoring influencing outcome is lacking.² In one randomised trial of ICP versus clinical examination plus imaging, there was no significant improvement in survival between the study groups.² Similar findings have been confirmed by other studies that have concluded that ICP-targeted/CPP-targeted intensive care prolongs mechanical ventilation and increases therapy intensity without evidence of improved outcome.⁵ However, management guidelines endorsed by the American Association of Neurological Surgeons (AANS) advocate the use of ICP monitoring for those at risk of intracranial hypertension.⁴ Contradictory studies purport reduced mortality and improved outcomes in those with ICP monitoring.⁴ The IMPACT meta-analysis reviewed reversible insults present on admission and their potential to influence outcome by exacerbating secondary injury. Hypoxia (arterial $pO_2 < 60$ mm Hg) (OR=2.1; 95% CI 1.7 to 2.6), hypotension (systolic blood pressure (SBP) < 90 mm Hg) (OR=2.7; 95% CI 2.1 to 3.4) and hypothermia (temperature $< 35^\circ\text{C}$) (OR=2.2; 95% CI 1.6 to 3.2) were strongly associated with poor outcomes, with hypoxia and hypotension having synergistic effects.⁶ The reversibility of these physiological insults reinforces the need for close monitoring in a critical care environment, where the early instigation of corrective therapy may restore normal physiology and improve outcome.⁶

Intracranial pressure

Several studies have shown that patients with an ICP < 20 mm Hg have a reduced risk of neurological deterioration, (relative risk (RR) 0.341 (95% CI 0.252 to 0.461; $p < 0.00001$), compared with higher ICPs (ICP ≥ 25 mm Hg; RR=3.042 (95% CI 2.288 to 4.045; $p < 0.00001$)).⁷ Mortality also increases dramatically from 17% to 47% when an average ICP > 20 mm Hg ($p < 0.0001$).⁸ However, ICP monitoring is complicated by infection in up to 22% of cases, the risk dramatically increasing > 5 days following insertion (RR=4.0; 95% CI 1.3 to 11.9).⁹ Regular CSF analysis to diagnose ventriculitis is recommended, which increases cost and consumes resources.⁹

Cerebral perfusion pressure

TBI has been managed by increasing mean arterial pressure and CPP with pharmacological agents such as dopamine and nor-epinephrine.¹⁰ Some have suggested that targeted therapy aimed at improving CPP is of greater significance than lowering ICP.¹⁰ Studies have revealed that mortality rates for CPP < 55 mm Hg are much greater than those with a CPP > 55 mm Hg (81% vs

23% respectively; $p < 0.0001$).¹⁰ Meanwhile, a CPP > 95 mm Hg has a detrimental impact on a positive neurological outcome (CPP < 95 mm Hg=50% vs CPP > 95 mm Hg=28%; $p < 0.033$).¹⁰ Others have failed to illustrate a statistically significant influence from CPP measurement alone.

Cerebral blood flow

Cerebral vasoreactivity ensures constant CBF. One treatment strategy for raised ICP focuses on hyperventilation causing cerebral vasoconstriction and reduced CBF.⁴ One randomised control trial assigned patients to receive normal ventilation ($PaCO_2 = 35 \pm 2$ mm Hg) or hyperventilation ($PaCO_2 = 25 \pm 2$ mm Hg) and found favourable neurological outcome to be significantly lower in the hyperventilated group ($p < 0.05$).¹¹ AANS therefore recommends avoiding hyperventilation and maintenance of normocarbida, other than as a temporising measure to reduce elevated ICP.⁴

Does ICP monitoring affect management?

Increasing ICP measurements are often the first indicator of an evolving mass lesion, 80% of which require surgery.⁴ Decompressive craniectomy removes part of the skull permitting cerebral swelling and reducing ICP. However, the procedure has not gained complete acceptance due to the poor functional outcome of surviving patients.¹² A multicentre randomised controlled trial of decompressive craniectomy versus standard therapy (DECRA trial) found that those receiving a craniectomy spent less time with raised ICPs ($p < 0.001$), received fewer interventions ($p < 0.02$) and spent fewer days in critical care ($p < 0.001$).¹² However, they had worse extended Glasgow Outcome Scale scores (OR=1.84; 95% CI 1.05 to 3.24; $p = 0.03$) and a greater risk of an unfavourable outcome (OR=2.21; 95% CI 1.14 to 4.26; $p = 0.02$).¹² Others have contradicted this, suggesting good neurological outcomes in those surviving > 30 days following craniectomy.¹³ Those with admission GCS > 6 were particularly good candidates for the procedure.¹³ Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of intra-cranial pressure (RESCUE-icp) study aims to determine the effectiveness of decompressive craniectomy and better define the indications for surgery. Hypertonic solutes such as 7.5% saline and 20% mannitol have been used to treat refractory intracranial hypertension, with

Learning points

- ▶ Primary injury refers to the initial insult caused by traumatic force, while secondary injury refers to delayed injury caused by tissue hypoxia and raised intracranial pressure (ICP).
- ▶ The Monro-Kellie hypothesis is key to understanding ICP and cerebral perfusion pressure (CPP) following traumatic brain injury (TBI). The rigid skull results in a fixed intracranial volume. Normal ICP is comprised of three components: brain tissue, cerebrospinal fluid and blood. If one component is elevated, ICP will increase unless the volume of the other two components decreases proportionately. Brain shift and herniation occur following a critical increase in ICP. This may manifest clinically as a drop in Glasgow Coma Scale.
- ▶ CPP=mean arterial pressure–ICP. The effects of raised ICP are exerted on the cerebral vasculature causing a progressive reduction in CPP.
- ▶ A review of the literature reveals a lack of evidence and contradictory evidence for many strategies used in the monitoring and treatment of TBI.

some studies suggesting the superiority of saline over mannitol.^{4 14} Others have treated patients with hypotension with 7.5% saline or Ringer's lactate solution and found little difference in favourable neurological outcome (risk ratio 0.99; 95% CI 0.76 to 1.30; p=0.96).¹⁵ AANS advocates the use of hyperosmolar therapy for the control of raised ICP, though hypotension (SBP<90 mm Hg) should be avoided.⁴

Competing interests None declared.

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