Intracranial pressure monitoring for traumatic brain injury

Clinical Problem

This expanded case summary has been chosen to explore the evidence in support of intracranial pressure (ICP) monitoring in patients with traumatic brain injury (TBI) who do not need primary surgical intervention.

A 33 year old male polytrauma patient was admitted to the neurosurgical intensive care unit (NICU) from the Accident and Emergency (A&E) department.

He had been involved in a road traffic collision with a truck whilst riding a bike. He suffered a cardiac arrest on scene and required cardiopulmonary resuscitation (pulseless electrical activity) and bilateral needle decompression from the paramedics. He had a return of spontaneous circulation after five minutes.

Management

On arrival in A&E he had a guedel airway in situ to maintain his airway, he had a respiratory rate of 36, a heart rate of 150 beats per minute and a systolic blood pressure of 60 mmHg. His Glasgow Coma Score (GCS) was 6 (E1,V2,M3). He was moving all four limbs.

He had bilateral chest drain insertions and underwent intubation and ventilation via a rapid sequence induction. He was transfused four units of blood and given tranexamic acid (1g). A trauma computed tomography scan (CT) was completed. He had the following injuries: basal skull fracture, C5-C7 lateral process fractures, small traumatic sub arachnoid haemorrhage, left scapula fracture and 1st to 4th and 6th to 7th left rib fractures.

The management plan was for 24 hours sedation and invasive ventilation with ICP guided medical treatment. The target cerebral perfusion pressure was 60mmHg.

On arrival to ICU an intracranial pressure “bolt” was inserted. Initial pressure was 11 mmHg. He was cardiovascularly stable at this time, with equal and reactive pupils.

Following a sedation hold the next day he was found to have profound upper limb weakness. He was re-sedated and taken for a Magnetic Resonance Imaging (MRI) scan on brain and spinal cord. Although the ICP bolt is listed as an MRI compatible device the neurosurgical team were concerned with the risk of heat injury and it was therefore removed prior to MRI. It had been in situ for approximately 36 hours and had not recorded a value greater than 15mmHg.

His MRI was normal, he was then extubated following a sedation hold but rapidly required reintubation for drowsiness and poor cough. The following day he was successfully extubated, he had persistent upper limb weakness and a degree of agitation. At the time of writing he is awaiting a High Dependency Unit bed.

At no point was medical management of raised ICP needed, nor was there evidence of raised ICP from either clinical examination or the ICP device. This case raises the
question as to whether it is appropriate to inset ICP devices in the initial phase of care in patients with only minor abnormalities on scan. Is it more appropriate to use clinical examination and repeat CT scanning if indicated?

**Discussion**

North American traumatic brain injury guidelines\(^1\) state that ICP monitoring is advisable in patients with a severe TBI (GCS 3-8 after resuscitation) who have an abnormal scan. ICP monitoring is also advised in patients with a normal scan and two of the following: age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure less than 90 mm Hg. National Institute for Health and Care Excellence guidelines\(^2\) state that severe TBI patients should be managed in a neurosurgical centre but do not give further recommendations on their management.

Until recently there has been a lack of high quality randomised controlled trials (RCT) addressing this issue. There have been a number of non-randomised trials which questioned the appropriateness of the guidelines quoted above. Ethical concerns regarding a possible lack of equipoise hampered progress.

In 2012 Mendelson et al\(^3\) published a systematic review of observational studies after a Cochrane review\(^4\) was published with no RCT's identified. Mendelson identified six studies (11371 patients in total) which were deemed too heterogeneous to allow valid pooled analysis. Of these studies four found no impact on survival with one showing improved survival and one showing increased mortality. A relatively consistent factor across these studies was patients with a GCS greater than 6 showing worse outcomes with ICP monitoring. This in part validates the guidelines from the Brain Trauma Foundation (BTF).

A retrospective cohort study of 333 patients with severe TBI (GCS <8) by Cremer et al\(^5\) found ICP monitors led to increased treatment intensity and ventilator days without improved outcome, consistent with Mendelson. In contrast Farahvar et al\(^6\) found improved two week mortality in a prospective cohort study of 1446 patients. This study, however, assessed only patients who were treated for raised ICP. Treatment consisted of medical (mannitol, hypertonic saline or barbiturates) or surgical (drain or craniectomy). They did not include patients with an ICP monitor who were not treated and as such the clinical applicability of the trial was impaired.

Chesnut et al\(^7\) succeeded where others had failed by identifying a group of ICU doctors in South America who routinely treated severe TBI without ICP monitoring. This allowed ethical approval for a RCT. 324 patients, age 13 and above, with severe TBI (GCS 3-8 within 48 hours of admission) that was deemed survivable were included. Six centres with neurosurgical services and high volumes of trauma patients were included. Randomisation was computer generated. The pressure monitoring arm had intraparenchymal monitors placed and ICP was aimed to be kept below 20mmHg in accordance with the BTF guidelines. The imaging-clinical examination arm had serial clinical examination and imaging, treatment was by pre-existing protocol with fixed regime hyperosmolar therapy escalating to hyperventilation and ventricular drainage and then barbiturate coma. Blinding of the treating team was not possible. Both trial arms were similar at baseline.
Primary outcome was a composite result of assessments undertaken at three and six months, these included survival, neuropsychiatric tests and functional status. Assessors were blinded to trial arm. There was a good successful follow up rate of 92%. Six month mortality was the same in both groups, as was the authors primary outcome and the extended Glasgow Outcome Scale (GOS-E). The imaging-clinical examination group had greater frequency and overall intensity of treatment, but barbiturates were used more frequently in the pressure monitored group. In summary this trial has failed to demonstrate a benefit to routine measuring and manipulation of ICP when compared to a clinical and radiological protocol driven strategy.

The catheter related complication rate in this paper was 6%, four devices malfunctioned, four were accidentally dislodged and two led to haemorrhage (severity not reported). Overall complication rates were similar between groups.

This study was well executed and analysed, however, there are weaknesses. Firstly there are marked differences in pre-hospital and rehabilitation care between the developed and developing world, this impacts on external validity. Furthermore the primary outcome measure is a composite score developed for this trial and not a more conventional and widely recognised system such as the GOS-E. Powering to detect a difference in GOS-E was inadequate. The pressure monitored group were also rigidly treated to keep ICP below 20mmHg which does not perhaps reflect current practice for all patients.

**Lessons learnt**

The use of ICP monitoring in isolation and without interpretation of context is of questionable merit. I believe that the use of ICP monitoring in conjunction with clinical examination, imaging and assessment of overall patient condition is still the most appropriate strategy in countries with access to such equipment. The Chesnut study does give evidence that when access to such equipment is not available then a protocol driven regime may be able to produce similar outcomes. Serious complications related to the ICP device are uncommon.

In this case the monitor did not prove useful but that is only known with hindsight. No harm was done to the patient and the device may have aided in the early detection and management of oedema, obstructive hydrocephalus or expanding haemorrhage.

A large RCT undertaken in a country whose infrastructure is similar to that of the UK would be helpful to further assess the topic, particularly if the treatment arm reflected current integrated use of ICP monitoring. I think ethical approval of such a study may remain elusive despite the Chesnut study.

Current evidence does not suggest the abandonment of ICP monitors but rather the intelligent use of them as part of an overall package of care.

**References**


