

This case has been selected by the ANZASM Committee for your information.

Potentially survivable primary cerebral injury with several lessons to be learnt

Case Summary:

A male patient in his late thirties was injured in quad bike rollover accident while intoxicated.

The patient suffered a cranial crush injury, sustaining complex fronto-facial fractures, a severe injury to the left eye, and intracerebral (minor frontal, cerebellar) and subarachnoid (peri-mesencephalic and basal) haemorrhage.

The patient presented with a Glasgow Coma Scale (GCS) score of 14-15 some hours after the injury. The patient was intubated for airway protection and subsequently underwent Faciomaxillary and Ophthalmological surgery, closure of scalp lacerations and insertion of an external ventricular drain (EVD).

There was subsequent management in the Intensive Care Unit (ICU); with the patient sedated and ventilated. The intracranial pressure (ICP) was monitored intermittently, with the EVD being used primarily as free drainage.

A review computed tomography (CT) scan was performed on day one post injury, and signs of raised posterior fossa pressure (development of cerebellar tonsillar herniation reduced definition of cerebellar folia) were demonstrated. The patient subsequently underwent a posterior fossa procedure, with midline decompression to the foramen magnum, and partial evacuation of subarachnoid clot.

Ventricular drainage continued, with intermittent measurement of ICP. On day five post injury, the patient underwent craniocervical Magnetic Resonance Imaging (MRI). This demonstrated persistent tonsillar herniation, and signs of raised posterior fossa pressure (oedema in cerebellar hemispheres, obliteration of folia, compression of brainstem against clivus, loss of subarachnoid space around brainstem).

The patient's condition subsequently declined and caused brain death.

Case Comments:

Areas for consideration;

The primary cerebral injury in this case would probably have been survivable, judging from the patient's presenting GCS, the mechanism of injury (crush rather than kinetic) and the initial imaging

findings.

It appears that the treating team may have under appreciated the significance of the injury involving the posterior fossa, and the subsequent development of raised pressure in this compartment (as indicated by radiological changes).

Reliance on supratentorial ICP measurement, and favouring free drainage over continuous pressure measurement, has probably compounded the problem, and resulted in a false sense of security as to control of the situation.

There appears to have been no attempt to lighten the patient to allow a clinical assessment of the neurological state, a step which was probably indicated given the patient's high presenting GCS, and which may have alerted to a deterioration.

There is scant documentation from the surgical team in the patient's file, and so an understanding of the strategy of management cannot be arrived at. This patient appears to have had a potentially survivable primary cerebral injury, and to have died from secondary injury, which may have been preventable, if the evolving changes in the posterior fossa were appreciated in time.

I suspect that the images were reviewed in timely fashion, but probably by junior staff, who did not observe, or did not appreciate the significance of the changes.

The reporting radiologist did note the tonsillar descent, but exactly when the report was completed, and to whom the information was passed, cannot be discerned.

Lessons to be learnt:

1. Recognise the different significance of a crush versus kinetic cerebral injury

2. Recognise that supratentorial ICP measurement may not reflect conditions in the posterior fossa

3. Consider whether ICP measurement intermittently is appropriate, rather than continuous monitoring

4. Be aware that continuous drainage is a treatment for hydrocephalus, not raised ICP due to cerebral insult

5. Consider waking a patient to assess, particularly if presenting GCS has been high.



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