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Decompressive craniectomy for severe traumatic brain injury: The relationship between surgical complications and the prediction of an unfavourable outcome

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ABSTRACT

Object: To assess the impact that injury severity has on complications in patients who have had a decompressive craniectomy for severe traumatic brain injury (TBI).
Methods: This prospective observational cohort study included all patients who underwent a decompressive craniectomy following severe TBI at the two major trauma hospitals in Western Australia from 2004 to 2012. All complications were recorded during this period. The clinical and radiological data of the patients on initial presentation were entered into a web-based model prognostic model, the CRASH (Corticosteroid Randomization After Significant Head injury) collaborators prediction model, to obtain the predicted risk of an unfavourable outcome which was used as a measure of injury severity.
Results: Complications after decompressive craniectomy for severe TBI were common. The predicted risk of unfavourable outcome was strongly associated with the development of neurological complications such as herniation of the brain outside the skull bone defects (median predicted risk of unfavourable outcome for herniation 72% *vs.* 57% without herniation, $p = 0.001$), subdural effusion (median predicted risk of unfavourable outcome 67% with an effusion *vs.* 57% for those without an effusion, $p = 0.03$), hydrocephalus requiring ventriculo-peritoneal shunt (median predicted risk of unfavourable outcome 86% for those with hydrocephalus *vs.* 59% for those without hydrocephalus, $p = 0.001$), but not infection ($p = 0.251$) or resorption of bone flap ($p = 0.697$) and seizures (0.987). We did not observe any associations between timing of cranioplasty and risk of infection or resorption of bone flap after cranioplasty.
Conclusions: Mechanical complications after decompressive craniectomy including herniation of the brain outside the skull bone defects, subdural effusion, and hydrocephalus requiring ventriculo-peritoneal shunt were more common in patients with a more severe form of TBI when quantified by the CRASH predicted risk of unfavourable outcome. The CRASH predicted risk of unfavourable outcome represents a useful baseline characteristic of patients in observational and interventional trials involving patients with severe TBI requiring decompressive craniectomy.

Introduction

Surgical intervention in the context of trauma can take many forms whether it is to arrest catastrophic haemorrhage, repair a ruptured viscus or to fixate a fractured limb. In most instances the decision to surgically intervene is based on the premise that any benefit provided in terms of outcome is not offset by the morbidity of the surgical procedure. A contemporary illustration of this problem is seen when considering decompressive craniectomy in the management of severe traumatic brain injury. The procedure is technically straightforward and can be performed either unilaterally or bilaterally (or bifrontally). A unilateral decompression is usually performed following evacuation of a mass lesion such as a subdural haematoma or when the cerebral swelling is localized to one hemisphere. A bilateral or bifrontal craniectomy is usually performed when there is diffuse cerebral swelling.

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Use of the procedure initially gained popularity in the early 1970s [1] only to fall into disrepute due to a combination of poor clinical outcomes [2] and experimental studies that suggested that decompression may actually worsen cerebral oedema [3] and this led to use of the procedure being almost abandoned. However, throughout the 1980s its popularity returned as an increasing number of studies demonstrated that surgical decompression could reliably lower the intracranial pressure and there would appear to be little doubt that in the context of intractable intracranial hypertension, surgery can represent a lifesaving intervention [4–6]. However despite many assertions to the contrary [7] evidence that the well documented reduction in ICP that occurs following surgical decompression is translated into an improvement in outcome is far less forthcoming.

The DECRA (Decompressive Craniectomy in Patients with Severe Traumatic Brain Injury) compared early decompressive craniectomy for diffuse traumatic brain injury with standard medical therapy and found that patients in the surgical arm of the trial had worse outcomes than those treated medically [8]. Notwithstanding a number of criticisms [9,10] the trial unequivocally demonstrated that at the particular ICP threshold at which these patients were enrolled there was insufficient ongoing secondary brain injury and therefore any benefit obtained by lowering the ICP was offset by surgical morbidity [11]. Indeed it is becoming increasingly apparent that use of the procedure exposes patients to significant morbidity not only from the initial decompression but also from the subsequent cranioplasty [12–15].

The aim of this study was to determine what features predispose patients to the development of complications and most notably whether injury severity was a contributing factor.

Methods

This is an ongoing observational cohort study for which approval has been given by the Royal Perth Hospital ethics committee. The data has been collected prospectively since 2009 and this has been combined with data from previous retrospective studies [16,17]. The time period covered is from 2004 to 2012 and includes all patients who had had a decompressive craniectomy following severe TBI at the two major trauma hospitals in Western Australia during this time. These two major trauma hospitals are the only neurosurgical centres that provide adult neurotrauma services in Western Australia serving a population of approximately 2.2 million. The indications for decompressive surgery were based on the Brain Trauma Foundation guidelines for management of intracranial pressure (ICP) following traumatic brain injury [18]. This involves protocol driven step wise administration of sedation, ventilation, neuromuscular paralysis, super salt therapy and CSF drainage where possible. Judicious use of mannitol and hyperventilation are used to treat transient rises in intracranial pressure. All patients were managed in the intensive care unit and had a parenchymal ICP monitor inserted. Eighteen (24%) of the seventy four patients, who went on to have a bifrontal decompression, had an external ventricular drain inserted prior to surgery.

The aim was to maintain the ICP below 20 mmHg and the cerebral perfusion pressure above 60 mmHg. A bifrontal decompressive craniectomy was considered if the intracranial pressure could not be maintained below 20 mmHg despite maximal medical management. In the majority of cases the intracranial pressure was consistently above 30 mmHg prior to surgery.

A unilateral decompressive craniectomy was performed following evacuation of a mass lesion when is was not possible to replace the bone flap because the intracranial pressure was greater than 20 mmHg (All patients have a parenchymal ICP monitor placed for post operative monitoring.) following evacuation of the haematoma and attempted replacement of the bone flap.

All complications were recorded. They were subdivided into those attributable to the initial decompressive procedure and those attributable to the subsequent cranioplasty.

Complications attributable to the initial decompressive procedure

Specific definitions of those complications included:

1. Cortical herniation and injury to the cortical surface. This was defined as more than 1.5 cm's herniation of the cortical surface outside of the line of the outer table of the (craniectomised) skull [15].
2. Injury to herniated cortex. Radiological evidence of new areas of haemorrhagic contusion relating to the edge of the craniectomy (as opposed to maturation of a previous cortical contusion).
3. CSF hydrodynamic disturbances
   (a) Subdural/subgaleal effusion; defined a low density collection greater than 1 cm maximal depth measured from the cortical surface to the inner aspect of the scalp [15]. The incidence of subdural/subgaleal effusion was only assessed amongst the patients that survived long enough to develop this complication (approximately six weeks).
   (b) Hydrocephalus; defined as dilatation of the ventricular system with accompanying clinical features that required placement of a shunt with subsequent clinical improvement [19]. The incidence of hydrocephalus was only assessed amongst the patients that survived long enough to develop this complication (approximately two months).
4. Seizures; all types of seizures were recorded.

Complications attributable to the cranioplasty

Specific definitions of those complications included:

1. Cranioplasty infection; defined as infection requiring removal of either an autologous or titanium cranioplasty in those survivors who had had a cranioplasty performed (superficial wound infections were excluded).
2. Bone flap resorption; This was defined as;
   a. Clinically significant (*i.e.* symptoms or signs noticed by the patient or the carers) requiring augmentation.
   b. Clinically significant (*i.e.* symptoms or signs noticed by the patient or the carers) either not warranting augmentation or augmentation offered but declined.
   c. Radiologically significant; for the purposes of this study it was defined as loss of both the inner and outer table of the cranioplasty such that protection to the underlying cranial contents was not fully restored [20].

Assessment of injury severity

The clinical and radiological data on initial presentation was entered into the CRASH (Corticosteroid Randomization After Significant Head injury) collaborators web based outcome prediction model [21] in order to obtain the percentage of predicted risk of an unfavourable outcome at six months (defined by the Glasgow Outcome Scale [22] as; dead, persistent vegetative state or severely disabled). This was used to stratify patients according to their severity of injury.

The clinical features required for the model are: Patient age, post resuscitation Glasgow coma score (GCS), pupillary response and presence of extra-cranial injuries. The radiological features required are the presence of; petechial haemorrhages, subarachnoid blood,