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Clinical Study

Complications of decompressive craniectomy for head injury

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ABSTRACT

There is much interest in the use of decompressive craniectomy for intracranial hypertension. Whilst technically straightforward, the procedure is not without significant complications. A retrospective analysis was undertaken of 41 patients who had had a decompressive craniectomy for severe head injury in the years 2006 and 2007 at the two major hospitals in Western Australia, Sir Charles Gairdner Hospital and Royal Perth Hospital. Complications attributable to the decompressive surgery were: herniation of the cortex through the bone defect, 18 patients (51%); subdural effusion, 22 patients (62%); seizures, five patients (14%) and hydrocephalus, four patients (11%). Complications attributable to the subsequent cranioplasty were: infection, four patients (11%) and bone flap resorption, six patients (17%). Syndrome of the trephined occurred in three (7%) of those patients whose bone flap had significantly resorbed. Two deaths (5.5%) occurred as a direct complication of the craniectomy or cranioplasty procedure. I attempted to define what may be regarded as a complication of the decompressive procedure rather than what may be a consequence of the primary pathological process of traumatic brain injury.

1. Introduction

Since the 1990s there has been a resurgence of interest in the use of decompressive craniectomy. Some studies have demonstrated that the procedure is effective in reducing intracranial pressure (ICP) in severe head injury,[1–7] stroke,[8] subarachnoid haemorrhage[9] and infection.[10] Whilst technically straightforward, the procedure is not without significant complications.[1,6,7,11–14] This report details the complications recorded in a cohort of 41 patients who had a decompressive craniectomy for trauma with an 18-month follow-up.

2. Method

A retrospective analysis was undertaken of all patients who had had a decompressive craniectomy for severe head injury in the years 2006 and 2007 at the two major trauma hospitals in Western Australia. These two major hospitals, serving a population of over two million people, are the only neurosurgical centres that offer neurological trauma services in Western Australia. All case notes and radiological investigations were reviewed to determine the clinical details, treatment and complications. The literature was reviewed to determine previously reported complications.[1,6,7,11–14]

The complications were divided into two main groups.

1. Complications attributable to the initial decompressive craniectomy. These were:
   (i) herniation of the cortex through the bone defect (defined as >1.5 cm outside the craniotomy defect)[14]
   (ii) subdural/subgaleal effusion (defined as a low density collection >1 cm maximal depth)
   (iii) syndrome of the trephined
   (iv) seizures
   (v) hydrocephalus (defined as dilatation of the ventricular system with accompanying clinical features that required placement of a shunt).

2. Complications attributable to the subsequent cranioplasty. These were:
   (i) infection (excluding superficial wound infections)
   (ii) bone flap resorption (defined as clinically significant, noticed by the patient or the carers).

In addition, secondary procedures, deaths and incidents that affected the patients overall recovery were recorded.

3. Results

During 2006 and 2007, there were 45 patients who had a decompressive craniectomy for traumatic brain injury. During this time six patients were enrolled in the DEcompressive CRAniectomy (DECRA) study, of whom four were randomised to the surgical arm of that trial. These patients were excluded from this study. For the remaining 41 patients, 100% follow up was achieved. The clinical outcome of these patients has been discussed elsewhere.[15] The

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patients were evenly distributed between the two hospitals and among the eight neurosurgeons who cover the on-call roster. The patient characteristics are shown in Table 1.

The indications for decompressive craniectomy were: (i) following evacuation of a mass lesion when the brain was felt to be excessively swollen; and (ii) persistently high ICP measurements despite protocol-driven, maximal medical management.

There were eight patients in the first group, who had a unilateral craniectomy for evacuation of a mass lesion. The remaining 33 patients had a bifrontal decompressive craniectomy for persistently raised ICP measurements. The timing of the decompressive procedure ranged from a few hours to 9 days following the initial trauma (Table 2).

The complications were subdivided into those attributable to the initial decompressive procedure and those attributable to the subsequent cranioplasty.

3.1. Complications of the decompressive surgery

Six patients died after the decompressive procedure (Table 2). In four patients the procedure failed to control intracranial hypertension. One patient died following a thoracic aorta rupture 36 hours following the initial trauma. One patient died following a fall onto the unprotected cranium.[16]

Of those 35 patients who survived, subgaleal/subdural effusions >1 cm in maximal thickness were observed in 22 patients (63%) (Fig. 1). In all but one patient, these were self-limiting. One patient developed a contralateral effusion with significant mass effect and this was successfully drained through a single burr hole (Fig. 2).

Herniation of the brain through the skull defect was noted in 18 patients (51%). In none of these patients was there any radiological or definite clinical evidence that the herniation had caused injury to the underlying cortex. The herniation usually resolved over 4 to 6 weeks as the cerebral oedema subsided (Fig. 3).

Four patients (11%) developed clinical and radiological evidence of hydrocephalus and required insertion of a ventriculoperitoneal (VP) shunt with associated clinical improvement. One patient who was in a prolonged vegetative state had a VP shunt inserted; however, there was no clinical improvement and there was severe shrinkage of the scalp flap. The diagnosis was amended from hydrocephalus to ventriculomegaly.

Five patients (14%) developed post-traumatic seizures.

3.2. Complications of the cranioplasty procedure

Thirty-five patients underwent a cranioplasty procedure (Table 3). In 33 patients an autologous bone flap was used. The bone flaps were stored in a refrigerator at −35 °C in sterile plastic containers. A preformed titanium cranioplasty was used in two patients: in one patient the initial bone flap had been badly contaminated and comminuted and was not suitable for reimplantation; in the second patient, the original bone flap had been inadvertently discarded.

There was one death following cranioplasty and ligation of a VP shunt. This was a young male with a very severe injury who had failed to make any significant neurological recovery after his decompressive procedure and subsequent insertion of VP shunt for ventriculomegaly. Following extubation after an uneventful procedure, he had a cardiorespiratory arrest and both pupils became fixed and dilated. CT scan confirmed diffuse cerebral oedema. He failed to recover despite removal of the bone flap and reopening of the shunt.

In four patients (11%) the cranioplasty became infected and required removal. In three of these patients the cranioplasty was autologous and the infections presented at post-operative day 5, 13 and 18. In one patient who had had a titanium cranioplasty (because of a badly comminuted skull fracture) the infection did not present until 9 months after cranioplasty. In all four patients the infecting organism was a staphylococcus and the patients responded to a prolonged course of antibiotic therapy. Whilst all four patients presented with headache, fever and soft-tissue swelling, none of the four patients sustained any new permanent neurological deficits.

Six patients (17%) developed clinical symptoms or signs relating to radiologically confirmed resorption of the bone flap. Three of these patients developed postural headaches and vertigo, two of whom heard noises similar to water movement when they changed posture. In one patient the bone flap sunk inwards as the titanium plates pulled out of the resorbing bone; an attempt at repositioning of the bone flap failed. These four patients required a preformed titanium cranioplasty to be manufactured (Fig. 4). The remaining two patients noted that the margins of their bone flap had become more noticeable and although CT scan confirmed some resorption of the bone flap they were relatively asymptomatic and unconcerned with the cosmetic appearance. In one of these relatively asymptomatic patients the bone was thought to be extremely thin and he was offered surgical augmentation; however, he declined (Fig. 5).

Table 1
Characteristics of 41 patients who underwent decompressive craniectomy for traumatic brain injury during 2006 and 2007

Mean age, years (median, SD, IQR)	32.1 (27.0, 14.2, 20–46)
Sex: male/female, no. (%)	34 (83%)/7 (17%)
Mechanism of injury, no. (%)	
Motor vehicle accident (excluding motorcycles)	13 (32%)
Fall	10 (24%)
Assault	8 (19%)
Motorcycle accident	8 (19%)
Other	2 (5%)
Mortality at 18 months, no. (%)	7 (17%)

SD = standard deviation, IQR = interquartile range.

Table 2
Decompressive craniectomy performed on 41 patients during 2006 and 2007 for traumatic brain injury

	Incidence (%)
Primary decompressive craniectomy procedures	41
Unilateral	12 (29%)
Bilateral	29 (71%)
Timing of procedure	
Following evacuation of a mass lesion	8
Following intractable intracranial hypertension	33
<24 hours	14
24–48 hours	7
48–72 hours	3
72–96 hours	1
96–120 hours	4
144–168 hours	2
>168 hours	2
Postoperative death following craniectomy	6 (12%)
Attributable to initial injury	5 (9.5%)
Complication of procedure	1 (2.5%)
Complications among the 35 survivors	
Subdural/subgaleal effusion (>1 cm max depth)	
Radiologically confirmed	22 (62%)
Clinically significant requiring drainage	1 (2.8%)
Cortical herniation (>1.5 cm outside the craniotomy edge)	18 (51%)
Hydrocephalus (requiring VP shunt insertion)	4 (11%)
Post-traumatic seizures	5 (14%)