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Early decompressive craniectomy and duraplasty for refractory intracranial hypertension in children: results of a pilot study

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Abstract

Introduction Severe traumatic brain injury (TBI) in childhood is associated with a high mortality and morbidity. Decompressive craniectomy has regained therapeutic interest during past years; however, treatment guidelines consider it a last resort treatment strategy for use only after failure of conservative therapy.

Patients We report on the clinical course of six children treated with decompressive craniectomy after TBI at a pediatric intensive care unit. The standard protocol of intensive care treatment included continuous intracranial pressure (ICP) monitoring, sedation and muscle relaxation, normothermia, mild hyperventilation and catecholamines to maintain an adequate cerebral perfusion pressure. Decompressive craniectomy including dura opening was initiated in cases of a sustained increase in ICP >20 mmHg for >30 min despite maximally intensified conservative therapy (optimized sedation and ventilation, barbiturates or mannitol).

Results In all cases, the ICP normalized immediately after craniectomy. At discharge, three children were without disability, two children had a mild arm-focused hemiparesis (one with a verbal impairment), and one child had a spastic hemiparesis and verbal impairment. This spastic hemiparesis improved within 6 months follow-up (no motor deficit, increased muscle tone), and all others remained unchanged.

Conclusion These observational pilot data indicate feasibility and efficacy of decompressive craniectomy in malignant ICP rise secondary to TBI. Further controlled trials are necessary to evaluate the indication and standardization of early decompressive craniectomy as a 'second tier' standard therapy in pediatric severe head injury.

Keywords craniectomy, intensive care, pediatric, severe head injury

Introduction

Severe traumatic brain injury (TBI) (Glasgow Coma Scale <8) occurs in 60% of polytraumatized children after car accidents or child abuse, and it is associated with a high mortality and morbidity [1,2]. The primary therapeutic aim is to maintain an adequate cerebral blood flow (estimated from cerebral perfusion pressure) and brain oxygenation. Intensive care management of severe head injury in cases of refractory

CBF = cerebral blood flow; CEO_2 = cerebral extraction rate for oxygen; CT = computed tomography; ICP = intracranial pressure; SEP = somatosensory evoked potentials; TBI = traumatic brain injury.

intracranial pressure (ICP) is not based on controlled, randomized studies. Studies in adults report more side effects than positive benefits [3].

Decompressive craniectomy has regained some therapeutic interest during the past decade. However, treatment guidelines for traumatic brain injury from German, European (European Brain Injury Consortium [4]) North-American (Brain Trauma Foundation [5]) and international (pediatric neurosurgery) [6] medical societies consider decompressive craniectomy only as last resort treatment strategy after failure of conservative therapy. In the pediatric population, a mere handful of case reports, cohort studies and pilot studies discuss the indication for decompressive craniectomy [7,8]. We report on the clinical course of six pediatric patients enrolled in a pilot study secondary to decompressive craniectomy after TBI.

Patients

All patients were immediately treated by the medical emergency team at the accident site and transferred to the Pediatric Intensive Care Unit (see Table 1 for details on diagnosis, treatment and follow-up). Early parameters of treatment at admission were transcutaneous oxygen saturation >92% and an estimated cerebral perfusion pressure of at least 50 mmHg.

Standard protocol of treatment

After emergent clinical evaluation with stabilization of ventilation and hemodynamics, a computed tomography (CT) scan was initiated. Significant traumatic masses were treated surgically on an emergency basis. In all other cases, an external ventriculostomy was performed and/or an ICP monitor was inserted. Insertion of an external ventriculostomy was performed in cases with accessible ventricles on admission for cerebrospinal fluid drainage as required by ICP monitoring. The ICP was monitored continuously by an intraparenchymal probe (MicroSensor; Codman, Johnson & Johnson, Raynham, MA, USA) in all cases. The treatment protocol generally applied was sedation and continuous muscle relaxation, 15–30° elevation of the upper part of the body, normothermia (36–37°C), and mild hyperventilation (pCO_2 = 30–35 mmHg). To maintain a sufficient cerebral perfusion pressure (50–60 mmHg; see [9]), all patients received catecholamines (epinephrine and norepinephrine) as needed.

Intensified treatment protocol

Standard therapy was intensified in cases of an ICP increase >20 mmHg for at least 30 min. Body position, body temperature, blood pressure, fluid management and ventilation as well as analgosedation were evaluated and optimized in order to lower the ICP. In each case of an unexpected and sustained elevation of ICP, a current CT scan was evaluated to rule out new space occupying intracranial lesions [10]. Continuing and sustained deviation of the ICP >20 mmHg for longer than 30 min was treated by single doses of barbiturate (2–5 mg/kg) and by infusion of mannitol (0.5 g/kg in 15 min). No treatment response within 30 min or even a further increase of ICP lead to immediate surgical treatment (decompressive craniectomy).

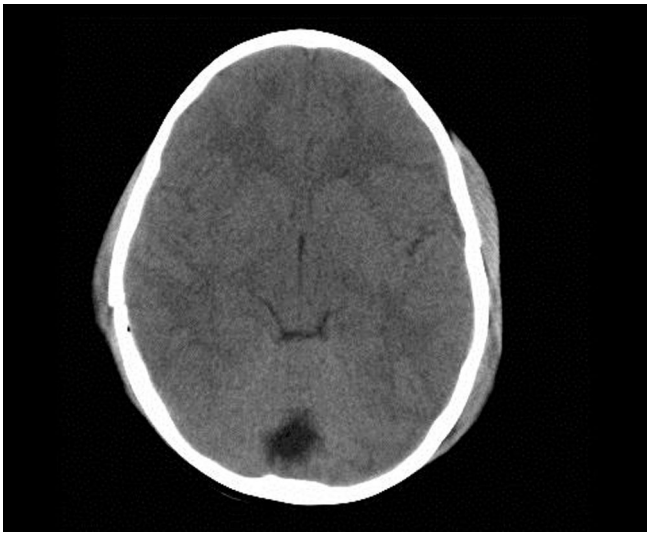
Table 1

Basic clinical data and course in study infants

Patient	Age (years)	Sex	Type of trauma	Glasgow Coma Scale on admission	Peak ICP (mmHg)	Initial cranial CT	Extent of craniectomy	Timepoint of craniectomy (days post-trauma)	Extubation (days post-trauma)
1	5	Female	Fall (3 m)	4	43	Bilateral skull fracture, infratentorial tSAH, DBS	Bilateral	1 and 2	7
2	5	Female	Fall (5 m)	5	30	Right-sided frontal brain contusion and tSAH, secondary DBS	Bilateral	3 and 5	8
3	11	Female	Child abuse	3	30	Right-sided acute subdural hematoma, extensive DBS	Right	1	6
4	6	Male	Car accident	4	70	Unilateral skull fracture; brain contusion in frontal lobe, basal ganglia and corpus callosum (DAI)	Bilateral	6	11
5	11	Male	Car accident	3	41	Left-sided calvarial and skull base fracture, tSAH, DBS	Left	2	9
6	9	Female	Kick by a horse	7	20	Left-sided temporal brain contusions, traumatic ventricular bleeding, infratentorial tSAH	Suboccipital	2	7

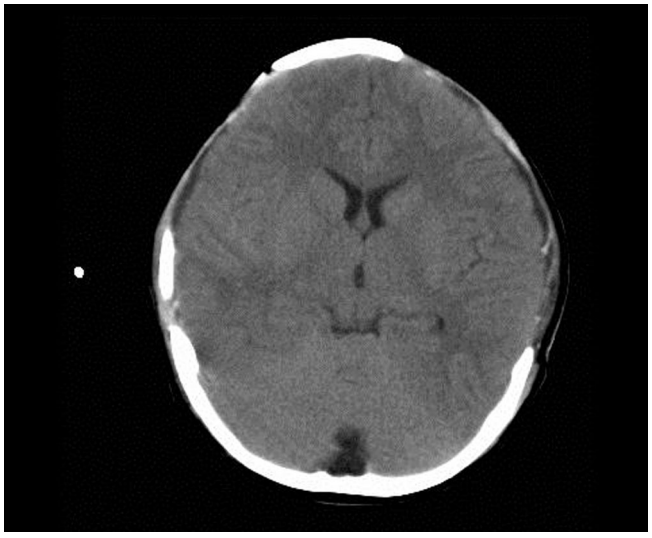
CT, computed tomography; DAI, diffuse axonal injury; DBS, diffuse brain swelling; ICP, intracranial pressure; tSAH, traumatic subarachnoid hemorrhage.

Figure 1



CT scan of patient 2 before craniectomy.

Figure 2



CT scan of patient 2 after bilateral craniectomy.

Surgical procedure

A unilateral or bilateral fronto-temporo-parietal craniectomy was performed depending on the extent and location of the brain swelling. The removed bone flap was stored by kryopreservation until secondary cranioplasty. The dura was opened and enlarged by an autologous galeal flap or by a Goretex patch. In patient 1, dura enlargement was restricted to one side despite bilateral decompression. In patient 6 (cerebellar contusion), a suboccipital craniectomy and duraplasty was performed because of a cerebellar swelling and altered somatosensory evoked potentials (SEP), in addition to a severe head trauma after blunt injury to the craniocervical junction.

Results

Immediate postoperative course

In five out of six patients, the ICP normalized (<12 mmHg) immediately after craniectomy and no secondary elevation in ICP was noticed. The continuous sedation and muscle relaxation could be tapered and stopped on day 5 or day 6 after surgical decompression.

Special clinical courses

Patient 2 showed a secondary brain swelling with an increase of ICP level intractable to intensified medical treatment on day 4 after unilateral decompression. A craniectomy of the contralateral side was therefore performed, with subsequent normalization of the ICP. Figure 1 presents the CT scan before and Figure 2 after bilateral craniectomy of patient 2.

Complications

There was neither infection nor disturbance of wound healing, nor mortality.

One patient (patient 3) developed a late aseptic necrosis of the replaced bone flap. In this case, a post-traumatic hydrocephalus led to subgaleal cerebrospinal fluid collection with surgical revision and transient insertion of a ventriculo-peritoneal shunt. This might have caused insufficient fixation of the bone flap and a lack of revascularization with subsequent partial necrosis. The shunt was removed 3 months after trauma and the bony defect was covered by an autologous calvarial split graft.

Neurological outcome

The neurological outcomes at discharge and at 6 months follow-up are presented in Table 2. Furthermore, SEP of the median nerve before and after decompressive craniectomy are described in Table 2.

Patient 1 suffered from a severe transitional syndrome after discontinuation of sedation. The neurological status was normal after recovery, in spite of pathological SEP of the median nerve. At discharge from the intensive care unit, patient 2 showed a hemiparesis, predominantly of the left arm, which resolved to normal strength in the following weeks.

None of the patients with severe head injury suffered from post-traumatic seizures or received anticonvulsive medication. Based on findings for SEP of the median nerve, a favorable and stable long-term outcome could be predicted for all of our patients suffering from TBI, confirming previously published data [11]. The SEP 1 week after trauma correlated with the neurological outcome 6 months after trauma, except for patient 1. Mild disturbances of SEP were seen in patient 1, but revealed no deterioration during follow-up.

Table 2

Neurological outcome of patients with decompressive craniectomy at discharge and after 6 months compared with somatosensory evoked potentials of the median nerve (M-SEP) before and after craniectomy

Patient	Neurological status (on demission)	Neurological status (6 months post-trauma)	M-SEP (prior to craniectomy)	M-SEP (first week after craniectomy)
1	Normal	Normal	Not tested	Moderate impairment (right)
2	Normal	Normal	Severe impairment (right)	Normal
3	Left-sided hemiparesis, VP shunt (PTH)	Distinct improvement of hemiparesis predominantly of the left arm, VP shunt removed	Not tested	Severe impairment (right)
4	Central impairment of coordination with tremor and ataxia; predominantly right-sided spasticity; speech retardation	Residual spasticity but not impaired in motor skills; visits a normal school	Moderate impairment (right), severe impairment (left)	Mild impairment (left)
5	Normal	Normal	Not tested	Normal
6	Hemiparesis predominatly of the right arm; left-sided abducent nerve paresis; impairment of swallowing and speech	Rehabilitation	Mild impairment (right), moderate impairment (left)	Severe impairment (left)

PTH, post-traumatic hydrocephalus; VP shunt, ventriculo–peritoneal shunt.

Discussion

After exclusion or surgical removal of traumatic hematomas and other space occupying lesions, prevention of secondary brain injury is the mainstay of intensive care treatment in pediatric severe head injury. Diffuse brain swelling and multiple cerebral contusions are the most common cause of morbidity and death after severe head injury in pediatric patients [12].

Standardized treatment protocols have been suggested for the management of severe head injury in children [13], including drainage of cerebrospinal fluid, mild hyperventilation (pCO_2 lower threshold of 30 mmHg) and mannitol bolus (unless serum osmolality exceeds 320 mosmol/l) as generally accepted baseline therapies for the pediatric population [6]. In cases of sustained elevated ICP (>20 mmHg) and reduced critically cerebral perfusion pressure (<50 mmHg), despite optimal medical therapy including controlled hyperventilation, further management using 'second tier' therapy is a matter of controversy [6] and has to follow the different stages of postinjury cerebral insults.

Brain swelling and intracranial hypertension in the early post-traumatic period has been proposed to induced by cerebral hyperemia (i.e. increased cerebral blood flow [CBF]), especially in children [14,15]. However, the impact of hyperemia on outcome has been rated controversially. Beneficial [16,17] as well as detrimental effects have been discussed [18].

'Second tier' intensified conservative treatment will have to rely on specific prognostic monitoring parameters. Therefore, CBF-dependent therapy has been studied [19]. But, as cerebral blood flow is age dependent in the unaffected child (normal range from 40 to >100 ml/100 g/min [20)], absolute cerebral hyperemia may only be defined within narrow age ranges [21]. CBF thresholds cannot be taken from adult studies for the initiation of therapeutic interventions in the pediatric population.

Monitoring of cerebral metabolic parameters has been reported for treatment in adult patients. In children, an early decrease in the cerebral metabolic rate of oxygen and the arterio-venous difference for oxygen has been reported to occur 1–3 days after trauma [14]. Recently, Cruz and colleagues [15] predicted clinical outcome based on monitoring of the ICP and the cerebral extraction rate for oxygen (CEO_2) in children. In their observational study of 45 children, an increased ICP and a decreased CEO_2 indicated cerebral hyperemia during the first 5 days after head injury. An unfavorable outcome occurred in children with higher ICP and lower CEO_2 (<17%). Monitoring of the CEO_2 (or oxygen saturation at the jugular vein bulb for hemoglobin >12 g/l) might therefore be used to direct ventilation and medical therapy in children in the future. However, two out of 45 patients died prior to intended decompressive surgery while being monitored for CEO_2, which points towards the need for shortened monitoring intervals and early surgical decompression.

Prolonged barbiturate therapy inherits a high risk of unwanted therapeutic effects, and revealed small benefits in the outcome in children [22]. In a proven state of refractory absolute hyperemia, selective reduction of the CBF by cerebral vasomodulation (dihydroergotamine, metoprolol and

clonidin [22], or a monotherapy dihydroergotamine respectively [23]) might be considered, but these treatment options are still not for routine application and require very intensive multimodal monitoring.

Brain edema associated with cerebral ischemia requires optimized cerebral perfusion and fluid management. Experimental medical treatment is proposed to lower the ICP and to re-establish sufficient CBF after failure of mannitol and vasopressors to support sufficient CBF. Hypertonic saline (7.2%) as a bolus or an infusion decreased the ICP in adults and children, and may therefore be indicated preferably in hypovolemia [24–26].

As a surgical 'second tier' option, controlled lumbar drainage of cerebrospinal fluid has been proposed. This regimen necessitates an external ventriculostomy and discernible basal cisterns on CT with careful control of both external drainage systems. In a study cohort of 16 pediatric head injury patients, Levy and colleagues [27] reported good control of refractory intracranial hypertension without drainage-related mortality.

Surgical decompression using craniectomy is largely seen as a last resort therapeutic option. This may be due to disappointment from previous anecdotic results based on late intervention. Encouraging results have been reported from studies in adolescent and adult patients indicating an early time point of decompression as extremely important to achieve a favorable outcome [3,8,28].

In addition to the 'optimal' time point for decompression, the extent of brain decompression seems to be important [3]. Restoration of cerebral perfusion by surgical enlargement of the intracranial space is the primary goal of decompression [3]. This may necessitate a large craniotomy with duraplasty. Prospective controlled, randomized studies on the effect of surgical decompression in TBI in childhood are missing. A pilot study by Taylor and colleagues [8] demonstrated an improved neurological outcome of patients who were treated with an early decompressive craniectomy in a cohort of 27 children compared with historical controls. In contrast to our patients, only a small temporal craniectomy without opening the dura was performed. The risk of transtentorial herniation can be lowered in this way, but restoration of the cerebral perfusion can hardly be achieved. However, a benefit from temporal craniectomy without duraplasty has been shown by Taylor and colleagues, which underlines the potential of a larger decompression. Studies in adults demonstrated a greater decrease of the ICP after duraplasty than in cases with craniectomy only [3,29].

Neither in these studies nor in our cohort was a higher rate of complications such as infections or hygroma noted due to duraplasty. Immediate normalization of the ICP after supratentorial surgical decompression was achieved in all patients from our study cohort. A good neurological outcome was achieved in all our patients suffering from TBI treated with decompressive craniectomy and duraplasty. Due to the early timepoint of decompression after failure of first-line treatment options, unwanted effects of prolonged medical therapy (e.g. barbiturate coma) or brain herniation with secondary brain stem compromise could be prevented, and all children survived.

Key messages

- In case of sustained increase in ICP (>20 mmHg) under intensified conservative therapy conditions and **early** decompressive craniectomy including duraplasty has to be considered

There currently seems to be no specific treatment regimen in children compared with adults in severe head injury [21], and there is no preference for a special 'second tier' treatment strategy in pediatric head injury [6]. The presented pilot trial adds an additional argument for surgical decompression at an early stage in case of treatment-refractory intracranial hypertension, and calls for a controlled trial that includes this treatment option in pediatric severe head injury patients.

Conclusion

This pilot trial and the favorable results from the study by Taylor and colleagues [8] demonstrate the necessity of a multicenter, controlled, randomized study to evaluate the indication and standardization of early decompressive craniectomy as a 'second tier' standard therapy in children with severe head injury.

Competing interests

None declared.

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