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Impact of timing of decompressive craniectomy on outcomes in pediatric traumatic brain injury

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Original Article

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ABSTRACT

Background: Decompressive craniectomy (DC) can be utilized in the management of severe traumatic brain injury (TBI). It remains unclear if timing of DC affects pediatric patient outcomes. Further, the literature is limited in the risk assessment and prevention of complications that can occur post DC.

Methods: This is a retrospective review over a 10-year period across two medical centers of patients ages 1 month–18 years who underwent DC for TBI. Patients were stratified as acute (<24 h) and subacute (>24 h) based on timing to DC. Primary outcomes were Glasgow outcome scale (GOS) at discharge and 6-month follow-up as well as complication rates.

Results: A total of 47 patients fit the inclusion criteria: 26 (55.3%) were male with a mean age of 7.87 ± 5.87 years. Overall, mortality was 31.9% (n = 15). When evaluating timing to DC, 36 (76.6%) patients were acute, and 11 (23.4%) were subacute. Acute DC patients presented with a lower Glasgow coma scale (5.02 ± 2.97) compared to subacute (8.45 ± 4.91) (P = 0.030). Timing of DC was not associated with GOS at discharge (P = 0.938), 3-month follow-up (P = 0.225), 6-month follow-up (P = 0.074), or complication rate (P = 0.505). The rate of posttraumatic hydrocephalus following DC for both groups was 6.4% (n = 3).

Conclusion: Although patients selected for the early DC had more severe injuries at presentation, there was no difference in outcomes. The optimal timing of DC requires a multifactorial approach considered on a case-by-case basis.

Keywords: Cranioplasty, Decompressive craniectomy, Posttraumatic hydrocephalus, Traumatic brain injury

INTRODUCTION

For decades, the leading cause of morbidity and mortality in the pediatric population has been traumatic brain injury (TBI).^[1,10,12,13,15,20,29,31,32,35] Severe TBI can lead to long-term disability and cognitive deficits; therefore, treating these injuries promptly and effectively is essential. TBI can be divided into primary and secondary brain injury. Primary injury refers to the injury sustained at the time of the initial insult and is typically not responsive to treatment. Secondary injury is resultant of the subsequent pathogenic autoregulatory mechanisms within the brain following primary injury.^[24,32] The cerebral swelling and elevated intracranial pressure (ICP) associated with secondary brain injury can cause decreased cerebral blood flow, decreased cerebral perfusion pressure (CPP), and herniation leading to compromised blood supply. This is frequently followed

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by ischemia and hypoxic injury resulting in irreversible tissue damage.^[8,10,12,13,20,24,26,32,37]

Treatment of severe TBI is divided into first tier and secondary tier therapies. Current first and secondary tier therapies are based on the third edition of guidelines for the management of pediatric severe TBI by Kochanek et al.[17,18] Primary treatment involves sedation and pain management, intubation, osmotic agent administration, cerebrospinal fluid (CSF) drainage through external ventricular drain (EVD), and head elevation. ICP monitors are often placed for better management of intracranial hypertension.^[2,10,15,18] Second-tier treatments are typically utilized in instances where the patient's ICP is refractory to primary treatment methods. Secondary interventions can include induction of a barbiturate coma, cerebral blood flowguided hyperventilation, higher doses of osmolar agents, and neurosurgical intervention such as decompressive craniectomy (DC). This is different than previous recommendations, which had DC as a last-resort treatment.[10,15,17,18]

DC is a potentially life saving procedure that is used in the treatment of severe TBI. It is utilized on a case-by-case basis for treatment of severe cerebral edema and intractable ICP.^[10,13] The procedure involves the removal of a part of the cranium to allow for the evacuation of any mass lesions and to create space for the brain to swell without causing detrimental increases in ICP.^[8,18] The timing of the procedure can be separated into early/acute (within 24 h) or late (after 24 h); however, the optimal timing of DC remains unclear.[10,20] Complication rates following DC are known to be high. Posttraumatic hydrocephalus (PTH) and incidence of hygromas following DC are common, and the sequelae of these disease processes can negatively impact patient outcomes.^[26] The literature is limited in potential management strategies to mitigate these complications. We aimed to assess the impact of postsurgical drain usage on PTH and hygroma incidence.

DC comes with inherent risks, so it is commonly utilized as a second-tier therapy. However, some recent studies indicate that DC in children can be done effectively and safely.^[2,18,20,26] The efficacy of DC remains undecided within the adult population, which has translated into the pediatric population as well.^[12,13,15,20,26] Studies involving outcomes, timing, indications, and complications of DC within pediatric patients are lacking. Of the studies that do exist, many have low patient numbers and lack statistical power.^[2,10,12,13,15,20,24,29,31] Even fewer studies exist looking at the significance of early versus late DC within the pediatric population. More studies are needed to elucidate the potential benefit of DC, especially early DC, along with analysis of outcomes and prognostic factors. ^[10,29,31,35] This study aims to assess outcomes and complication rates in pediatric neurosurgical patients in relation to the timing of DC and the impact of postsurgical drain usage on PTH and hygroma rates. We hypothesized that postsurgical EVDs and subgaleal drains would decrease the rates of PTH. Furthermore, we hypothesized that pediatric patients overall would have good outcomes after undergoing DC.

MATERIALS AND METHODS

Study population

Following Institutional Review Board approval, we performed a retrospective review of pediatric patients who underwent DC from 2012 to 2021. This study was conducted at Covenant Women and Children's Hospital and University Medical Center, a Level I Trauma Center that serves as the primary teaching hospital and tertiary referral center for a large rural population. Patients included were younger than 18 years of age and underwent a DC for TBI at one of our institutions. Patients who received a DC for an indication other than TBI were excluded from the study. We obtained a list of patients from the IT department at each institution of patients 18 years or under who presented to the emergency department (ED) for TBI. This list was then manually screened, and all patients who had DC for TBI with or without hematoma evacuation were included in the study.

Data acquisition

All data were extracted from the electronic medical record. Demographic data, including age, sex, mechanism of injury, and type of injury, were obtained for all patients. ICP, CPP, and Glasgow coma scale (GCS) scores were obtained from chart notes at admission, pre-surgery, and post-surgery. Admission data were collected from the first recorded measurement following arrival at our institution. Some patients, especially those in the acute DC group, did not have admission/preoperative ICP or CPP measurements; however, all had admission GCS scores documented. Pre-surgery data were obtained from documentation immediately prior to the operation. Post-surgery data were obtained from notes between 24 and 48 hours after the operation based on when the first postoperative measurements were documented. For pre-surgery ICP measurements in patients who did not receive ICP monitoring before surgery, some patients had ICP monitors placed in the operating room prior to the DC, and these measurements were utilized. Not all patients had ICP and CPP monitoring before surgery; however, all were measured following their operation.

Data were also collected regarding the type of neurologic treatment patients received before surgery and the locations where they received this treatment. Possible treatment locations included the ED, the pediatric intensive care unit (PICU), either outside the hospital or by emergency medical services, and the surgical intensive care unit. The type of DC received, the timing (acute versus subacute) of the DC, and the indications for the operation were documented for each patient. Preoperative imaging data included computed tomography (CT) and magnetic resonance imaging (MRI). CT studies were utilized for scoring the Rotterdam score and

Marshal classification. Imaging studies were also utilized for the description of mass lesions before surgery and progression following DC. After the operation, complications, including any incidence of infection, CSF leak, hydrocephalus, subdural/subgaleal fluid collection, and new or progression of hematomas, were recorded from imaging studies and neurosurgical notes for each patient. Hydrocephalus was defined as ventriculomegaly requiring shunting following the DC. Complications were noted, whether a follow-up operation was indicated or not, as some complications can delay operations such as cranioplasty. Significant progression of cerebral swelling and herniation through the skull defect was also documented based on imaging studies and neurosurgical notes. The usage of drains, the type of drains, and the amount of CSF drained from each patient were also documented.

Primary outcomes included total length of stay (LOS), ICU LOS, and Glasgow outcome scale (GOS). GOS was utilized as it has previously been validated in head injury and has been used in other similar studies.^[13,16,22,23] GOS was recorded from discharge and from 3 to 6-month follow-up notes. Favorable outcomes were labeled as GOS of 4 and 5, indicating moderate to no disability with preserved patient independence. A GOS of 3 indicates severe disability with loss of independence, 2 indicates a persistent vegetative state, and a GOS of 1 indicates death. All patients had the same follow-up period of 6 months. Due to the rural population served by these two hospitals, longer follow-up periods were not consistently present, so a maximal period of 6 months was used to keep the follow-up period equal between all patients.

All DC procedures and the decision to operate were decided on a case-by-case basis with a multifactorial decisionmaking method. The decision to operate preoperative medical management was guided by the 2019 TBI guidelines by Kochanek et al.[18] Surgical indications were based on clinical presentation/deterioration, cerebral swelling and ICP, herniation/midline shift, and the presence of mass lesions. DC type was also surgeon-dependent, but it was based on the location of the injury, severity, and extent of the injury. Four different surgeons operated on the patients included in this study. DCs were frontal, bifrontal, frontotemoroparietal, or frontotemporal, depending on the amount of cerebral edema and size of a mass lesion. A durotomy was also completed at the time of the operation. The dura was opened in a stellate fashion and was closed with a dura matrix and 4-0 nylon sutures. Patients were divided into acute and subacute groups based on time to DC following arrival to the institution. Patients who received a DC in under 24 h were categorized as acute, and those undergoing DC after 24 h were categorized as subacute, similarly to what was considered an early DC by Taylor et al.[35] There was not sufficient data in the charts to measure timing based on presentation to outside hospitals in patients who were transferred from outside hospitals.

Statistical analysis

Statistical analyses used two-sided P-values, independent samples, and a significance level of $\alpha = 0.05$. Continuous variables are summarized using the mean and standard deviation (SD). Categorical variables are summarized using counts and percentages. Differences in independent interval level variables are tested using the permutational unequal variance Welch t-test based on 1000 permutations. Differences in independent nominal level and binary variables are tested using Fisher's test. The standardized mean difference is used as the standardized effect size for nominal-level, binary, and interval-level variables. Differences between ordinal level variables are tested using the Mann-Whitney U-test with Cliff's δ as the standardized effect size. Associations between independent interval or ordinal level variables are tested using Spearman correlation. For repeated measures pre-surgery to post-surgery, the permutational dependent samples t-test or the sign test has been applied. To aid the interpretation of nonsignificant hypothesis tests, Supplementary Figure 1 provides *post hoc* power analyses corresponding to the sample sizes of the project. To simplify interpretation and to balance the Type I and Type II error probabilities, adjustments to P-values to control the family-wise error rate and false-positive rate have not been made. Confidence intervals for effect sizes and population parameters are not reported since hypothesis testing is the inferential area of focus, and the relatively small sample size implies relatively wide confidence intervals.

RESULTS

Demographics

A total of 47 patients underwent a DC; 25 (53.2%) were male, and the mean age was 7.87 (SD = 5.87) years. The most common etiology of these patients was motor vehicle



Figure 1: Differences in pretreatment methods/location between the acute and subacute groups. Asterix refers to statistical significance, if there is an asterix statistical significance was met. ED: Emergency department, PICU: Pediatric intensive care unit, EVD: External ventricular drain, ICP: Intracranial pressure.

collision (42.6%), followed by known accidental trauma (21.3%), nonaccidental trauma (19.1%), and gunshot wounds (17.0%). The overall mortality rate was 31.9% (n = 15). The average admission GCS for all patients was 5.8 (SD = 3.7). The mean GOS of the survivors was 3.68 (SD = 1.06) at discharge and 4.25 (1.00) at six-month follow-up, with 75.0% of the survivors having a favorable outcome (GOS 4 or 5) at their 6-month follow-up.

Timing of DC

There were 36 (76.6%) patients who underwent an acute procedure, whereas 11 (23.4%) underwent a subacute procedure. There were no statistically significant differences in age, sex, mechanism of injury, or original insult between the two groups [Table 1]. The group undergoing acute DC was more frequently treated in the ED (P = 0.018), whereas the subacute group more commonly received treatment in the PICU (P = 0.001) before surgical intervention [Table 2]. Three patients in the acute group were treated in the PICU and in the ED. Both acute and subacute groups received treatment before the DC at a similar rate (88.9% vs. 100%, respectively); however, the type of treatment varied between the two groups. The subacute group received a wider range of treatments including higher usage of ICP monitors (P = 0.001), EVDs (P < 0.001), barbiturates (P = 0.036), 3% saline (P = 0.003), and head elevation above 30° (P = 0.011) [Figure 1]. Indications between the two groups also varied. The subacute group had more instances of intractable ICP (P < 0.001) and stroke, though this trend did not reach statistical significance (P = 0.051). Conversely, the acute group had more subarachnoid hematomas per patient (0.44 vs. 0.09) (P = 0.013).

The acute group presented with lower GCS scores than the subacute group (P = 0.030) [Table 3]; however, there was no significant difference in admission, pre-surgery or post-

surgery ICP, CPP, or mean arterial pressure (MAP) [Table 4]. There was no difference in complication rate (P = 0.505) or type of complications encountered. There was a significant positive association between the timing of DC and ICU stay (P = 0.033); however, total LOS was not significantly different (P = 0.057). Acute and subacute DC groups had comparable outcomes at discharge measured by GOS (mean of 2.94 vs. mean of 2.90, respectively, P = 0.938) [Figure 2]. Patients who underwent acute DC had better outcomes at 6-month follow-up with an average GOS of 4.5 (SD = 0.76) compared to the subacute GOS of 3.60 (SD = 1.30); however, it did not reach statistical significance (P = 0.074). The mortality rate was 33.3% in the acute and 18.2% in the subacute group (P = 0.461).

Outcomes

The overall complication rate was 36.6% [Table 4]. Patients who underwent DC for intractable ICP and anoxic brain injury had worse discharge GOS (P = 0.021 and P = 0.021, respectively). Following surgical decompression, herniation of the brain through the skull defect and continued progression of brain swelling were significantly associated with worse discharge GOS (P = 0.031 and P = 0.001). Pre- and post-surgery GCS (P = 0.001 and P < 0.001, respectively), pre-surgery ICP (P = 0.017), and pre-and post-surgery CPP (P = 0.050 and P = 0.001, respectively) also correlated with poor discharge GOS. All complications together were not significantly associated with GOS at discharge (P = 0.235). In the acute group, ICP was significantly lower following surgery, whereas CPP was not (P = 0.019 and P = 0.602,respectively). However, in the subacute group, both ICP and CPP were significantly lower following surgery (P < 0.001and P = 0.007, respectively). This difference could be a result of only 0.42% (n = 15) of acute patients receiving an ICP monitor compared to 0.82% (n = 9) in the subacute group [Tables 5, 6 and Figure 3].

Table 1: Demographic data for the acute and subacute groups.					
	Acute	Subacute	SMD	P value	
Male	21/36 (58.3)	4/11 (36.4)	0.451	0.303	
*Age	8.24 (5.77)	6.69 (6.32)	0.263	0.479	
MOI			0.457	0.837	
MVC	15/36 (41.7)	5/11 (45.5)			
Known accidental	8/36 (22.2)	2/11 (18.2)			
NAT	6/36 (16.7)	3/11 (27.3)			
Unknown	0/36 (0.0)	0/11 (0.0)			
GSW	7/36 (19.4)	1/11 (9.1)			
Insult			0.859	0.305	
Closed head injury	17/36 (47.2)	3/11 (27.3)			
Skull fracture	12/36 (33.3)	7/11 (63.6)			
Penetrating head wound	7/36 (19.4)	1/11 (9.1)			

NAT: Nonaccidental trauma, GSW: Gunshot wound, MVC: Motor vehicle accident, MOI: Mechanism of action, SMD: Standardized mean difference. *The average age with the standard deviation in the parenthesis. The number with the slash is the ratio and the number in the parenthesis is the average

Table 2: Pretreatment methods/location and the indications for decompressive craniectomy between the acute and subacute groups.						
	Acute	Subacute	SMD	P value		
Pretreatment	32/36 (88.9)	11/11 (100.0)	-0.500	0.560		
Pretreatment in ED	13/36 (36.1)	0/11 (0.0)	1.063	0.018		
Pretreatment in PICU	12/36 (33.3)	10/11 (90.9)	-1.475	0.001		
Pretreatment at OSH or EMS	17/36 (47.2)	4/11 (36.4)	0.222	0.731		
Pretreatment SICU	2/36 (5.6)	0/11 (0.0)	0.343	>0.999		
ICP monitoring	9/36 (25.0)	9/11 (81.8)	-1.386	0.001		
Intubation	31/36 (86.1)	9/11 (81.8)	0.117	0.659		
EVD	6/36 (16.7)	9/11 (81.8)	-1.718	< 0.001		
Fentanyl, benzodiazepines, propofol	25/36 (69.4)	10/11 (90.9)	-0.559	0.244		
Barbiturates	15/36 (41.7)	9/11 (81.8)	-0.907	0.036		
3% saline	18/36 (50.0)	11/11 (100.0)	-1.414	0.003		
Mannitol	15/36 (41.7)	4/11 (36.4)	0.109	>0.999		
Rocuronium	13/36 (36.1)	6/11 (54.5)	-0.377	0.312		
VP shunt	0/36 (0.0)	0/11 (0.0)	0.000	>0.999		
Head of bed above 30°	5/36 (13.9)	6/11 (54.5)	-0.948	0.011		
Intractable ICP	10/36 (27.8)	11/11 (100.0)	-2.280	< 0.001		
Neurologic deterioration	9/36 (25.0)	3/11 (27.3)	-0.052	>0.999		
Subarachnoid space decompression	1/36 (2.8)	0/11 (0.0)	0.239	>0.999		
Subarachnoid space compression	3/36 (8.3)	0/11 (0.0)	0.426	>0.999		
Vascular compression	2/36 (5.6)	0/11 (0.0)	0.343	>0.999		
Stroke	0/36 (0.0)	2/11 (18.2)	-0.667	0.051		
Anoxic brain injury	5/36 (13.9)	2/11 (18.2)	-0.117	0.659		
Hemorrhage or swelling	29/36 (80.6)	7/11 (63.6)	0.384	0.256		
Midline shift	23/36 (63.9)	5/11 (45.5)	0.377	0.312		
Herniation	14/36 (38.9)	6/11 (54.5)	-0.318	0.489		
*Number of hematomas	1.92 (1.25)	1.27 (1.10)	0.534	0.105		
*Number of epidural hematomas	0.19 (0.47)	0.09 (0.30)	0.228	0.265		
*Number of subdural hematomas	1.06 (0.98)	0.73 (1.01)	0.334	0.359		
*Number of subarachnoid hematomas	0.44 (0.65)	0.09 (0.30)	0.593	0.013		
*Number of intraparenchymal hematomas	0.28 (0.57)	0.36 (0.50)	-0.144	0.567		

SMD: Standardized mean difference, ED: Emergency department, PICU: Pediatric intensive care unit, OSH: Outside hospital, SICU: Surgical intensive care Unit, EMS: Emergency medical services, ICP: Intracranial pressure, EVD: External ventricular drain, VP: Ventriculoperitoneal. *This is the average number with the standard deviation in the parenthesis. The number with the slash is the ratio and the number in the parenthesis is the average

Table 3: In hospital GCS, GOS at discharge and at follow-ups, and CT classifications between acute and subacute groups.

	Acute	Subacute	Cliff's delta	<i>P</i> value
Admission GCS	5.03 (2.97)	8.45 (4.91)	-0.412	0.030
Pre-surgery GCS	4.17 (2.24)	4.82 (1.99)	-0.232	0.178
Post-surgery GCS	5.23 (2.89)	5.00 (2.56)	0.008	0.968
Discharge GOS	2.89 (1.53)	2.90 (1.30)	0.015	0.938
3-month GOS	4.05 (1.12)	3.56 (1.13)	0.270	0.225
6-month GOS	4.50 (0.76)	3.63 (1.30)	0.394	0.074
Rotterdam score	2.74 (1.31)	2.18 (0.60)	0.291	0.131
Mashal classification	4.16 (1.25)	4.14 (1.21)	0.227	0.206
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GCS: Glasgow coma scale, GOS: Glasgow outcome scale, CT: Computed tomography. The numbers in the table are averages with the standard deviation in the parenthesis

Drain usage

Of all the patients who underwent DC, 26 (55.3%) had an EVD, and 39 (83.0%) had a subgaleal drain postoperatively. The average time for a patient to have the subgaleal drain in place was 3.12 (SD = 2.34) days. Patients who had an

EVD had an average of 1.8 EVDs (SD = 0.93) during their stay. The usage of EVDs was associated with an increased rate of infection (P = 0.007), while the number of EVDs used was associated with an increased rate of subdural fluid collections (P = 0.036). Similarly, the number of days spent with a subgaleal drain was associated with an



Figure 2: (a and b) Outcome comparison between acute and subacute decompressive craniectomy. GOS: Glasgow outcome scale



Figure 3: Pre- and post-surgery characteristics in the acute versus subacute decompressive craniectomy groups. (a) Pre- and post-surgery intracranial pressure. (b) Pre- and post-surgery cerebral perfusion pressure. (c) Pre- and post-surgery mean arterial pressure. (d) Pre- and post-surgery Glasgow coma scale. ICP: Intracranial pressure, DC: Decompressive craniectomy, SMD: Standardized mean difference, GCS: Glasgow coma scale, MAP: Mean arterial pressure, CPP: Cerebral perfusion pressure. The numbers correlate to the number of patients who shared the same point.

increased incidence of infection (P = 0.040). There was a greater incidence of CSF leak with increasing days with a subgaleal drain, though not reaching statistical significance (P = 0.063). There were no significant differences in subgaleal fluid collection, subdural fluid collection, or hydrocephalus with either EVD or subgaleal drain. The total overall incidence of subgaleal fluid collections was 2.1%, subdural fluid collections were 8.5%, and hydrocephalus

was 6.4%. The median amount of CSF drained was 200-300 mL/24 h. Pressure settings of EVDs fluctuated in patients based on the need for CSF drainage.

DISCUSSION

The role of DC following severe TBI remains heavily debated in the neurosurgery community.^[10,18,24,26,29,32,38] The idea of

subacute groups.					
	Acute	Subacute	SMD	P value	
Admission ICP	32.78 (17.49)	25.29 (9.98)	0.508	0.295	
Admission CPP	59.56 (20.57)	63.29 (21.12)	-0.179	0.722	
Admission MAP	86.47 (21.20)	85.82 (22.23)	0.030	0.929	
Pre-surgery ICP	36.00 (23.29)	32.33 (14.19)	0.179	0.639	
Pre-surgery CPP	48.27 (25.87)	48.22 (7.40)	0.002	0.993	
Pre-surgery MAP	78.89 (20.63)	77.45 (15.62)	0.073	0.822	
Post-surgery ICP	21.39 (21.17)	11.82 (4.77)	0.514	0.057	
Post-surgery CPP	53.39 (19.58)	59.18 (8.02)	-0.330	0.192	
Post-surgery MAP	71.83 (17.65)	72.64 (11.53)	-0.049	0.881	
*Hydrocephalus	3/36 (8.3)	0/11 (0.0)	0.426	>0.999	
*Subdural fluid collection	2/36 (5.6)	2/11 (18.2)	-0.398	0.229	
*Subgaleal fluid collection	1/36 (2.8)	0/11 (0.0)	0.239	>0.999	
*Infection	7/36 (19.4)	2/11 (18.2)	0.032	>0.999	
*CSF leak	5/36 (13.9)	1/11 (9.1)	0.151	>0.999	

Table 4: Complications, intracranial pressure, cerebral perfusion pressure, and mean arterial pressure compared between acute and subacute groups.

SMD: Standardized mean difference, ICP: Intracranial pressure, CPP: Cerebral perfusion pressure, MAP: Mean arterial pressure, CSF: Cerebrospinal fluid. *These are the proportion of patients with it listed as a % in the parenthesis. The numbers are the averages with the standard deviation in the parenthesis

Table 5: Pre-surgery to post-surgery changes for acute and subacute decompressive craniectomy.						
	n	Pre-surgery ^a	Post-surgery	Change	SMD ^b	<i>P</i> value ^c
Acute ICP (mmHg)	15	*36.0 (23.3)	23.4 (22.6)	-12.6	-0.609	0.019
101 (19)	10	0010 (2010)	2011 (22:0)	(20.7)	0.000	01017
CPP (mmHg)	15	*48.3 (25.9)	52.3 (20.1)	4.0 (27.2)	0.147	0.602
MAP (mmHg)	36	*78.9 (20.6)	71.8 (17.7)	-7.1 (26.8)	-0.264	0.135
Subacute						
ICP (mmHg)	9	*32.3 (14.2)	12.0 (5.3)	-20.3	-1.425	< 0.001
-				(14.3)		
CPP (mmHg)	9	*48.2 (7.4)	61.1 (7.2)	12.9 (8.3)	1.562	0.007
MAP (mmHg)	11	*77.5 (15.6)	72.6 (11.5)	-4.8 (17.5)	-0.276	0.402

^aMean (standard deviation), ^bstandardized mean difference, ^cmatched pairs permutational *t*-test using 1000 simulations. SMD: Standardized mean difference, ICP: Intracranial pressure, CPP: Cerebral perfusion pressure, MAP: Mean arterial pressure. *Average with the standard deviation in the parenthesis

Table 6: Pre-surgery to post-surgery changes in ordinal and binary level variables.

0 / 1	0 / 0	1		
	Pre-surgery	Post-surgery	Change (-, 0, +)	P value ^a
Acute (<i>n</i> =36)				
*GCS (3 to 15) ^b	3.0 (1.3)	3 (4.0)	(6, 18, 12)	0.238
Pupil reactivity (reactive) ^c	**18/36 (50.0)	18/36 (50.0)	(4, 28, 4)	1.000
Pupil size (nondilated)	**16/36 (44.4)	25/36 (69.4)	(1, 25, 10)	0.012
Subacute (<i>n</i> =11)				
GCS (3 to 15)	4.0 (3.5)	3.0[3.5]	(2, 7, 2)	1.000
Pupil reactivity (reactive)	**6/11 (54.5)	4/11 (36.4)	(2, 9, 0)	0.500
Pupil size (nondilated)	**6/11 (54.5)	10/11 (90.9)	(1, 5, 5)	0.219
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^a*P* values computed using the sign test, ^bgcs summarized as median (interquartile range), ^cpupil size and pupil reactivity summarized as count (%). GCS: Glasgow coma scale. ^{*}This is the median with the standard deviation in the parenthesis, ^{**}This is the proportion of patients with the % listed in the parenthesis

removing part of the cranium to allow for greater brain expansion secondary to swelling and edema has been around for centuries.^[9,32] Despite its longevity, the surgical procedure has fallen in and out of favor over the years.^[13,19] Recently, much

of the controversy around DC stems from the lack of strong evidence-based recommendations. Most studies in the literature conducted to evaluate the efficacy of DC are in the adult population. Pediatric DC has been studied less than in the adult population.^[10,18,20,31] Further, many of the studies on pediatric DC are limited by a low sample size. Moreover, only one randomized control trial has been conducted, with most studies being retrospective. Of the existing literature, DC has been shown to decrease intractable ICP, thus increasing CPP effectively. This is due to the increase in volume and ability of the expansion of the brain. Early effective control of ICP has been associated with better long-term outcomes. DC is also associated with favorable outcomes in surviving patients in pediatrics.[10,11,13,15,23,32,36] Within our pediatric patient population, DC was a viable option in the treatment of severe TBI and elevated ICP, irrespective of the timing of the procedure. After undergoing DC, 75.0% of survivors recovered with a favorable outcome (GOS 4 or 5) at six-month follow-up, and our mortality rate (33.3%) was similar to prior studies.^[10,12,13,15,16,23,29,30] We experienced a much lower incidence of PTH (6.9%) than commonly reported in the literature (29-42%).[13,15,26]

The impact of timing to DC following the primary insult and hospital arrival remains unclear in the pediatric population.^[10,20] Some studies have indicated that early DC may provide some benefit in patient outcomes, whereas others have indicated that there is no effect.^[2,7,12,14,23,32] Many studies do not compare early versus late DC, however. In studies by Mhanna et al. and Josan and Sgouros, the conclusion is that early DC is better for the management of intracranial hypertension and improves functional outcomes; however, the control group consists of patients who did not receive a DC rather than those undergoing a late DC. [14,23] Similarly, Csokay et al. concluded that early DC is beneficial to avoid sudden increases in ICP that could be life-threatening; however, their patient population consisted of eight patients, only one of which had a DC later than 24 h.[7] We found that early DC did not correlate with better patient outcomes at discharge or patient mortality rates. This study is one of the few existing studies in which an early DC group is directly compared to a late DC group and has a larger population size compared to the previous studies. Similar outcomes could be a result of the heterogeneity of TBI, with some not necessitating surgical intervention until later in the disease process.

Patel *et al.* found that after controlling for mechanism of injury and injury severity, rural pediatric TBI patients had the same mortality and in hospital complications as compared to urban patients.^[25] However, it is worth noting that Patel *et al.* may have been subject to population bias, as a similar study by McCowan *et al.* postulates that a larger proportion of severely injured rural patients do not make it to the hospital than their urban counterparts, resulting in lower in-hospital mortality.^[21] Studies indicating that the amount of time a patient's ICP is above 20 mmHg correlated to worse outcome led to the idea that early DC was more efficacious than late DC. This indicates that it is important to get patients decompressed once ICP criteria have been

met. This further supports the notion the clinical condition should dictate timing to DC.^[3,11,13,27,32,36] Patients that were in the early decompression group did present with lower GCS scores than subacute. This suggests that the patients in the early group presented with more severe injuries, which may account for the necessity of their acute procedure. Based on our findings, timing to DC can be based on clinical status rather than timing from initial injury.

Following the results and limitations of the DECRA and rescue ICP studies, the potential benefits and limitations of DC remain unclear. However, within the pediatric population, many studies report surviving patients with favorable outcomes following DC.^[3,10,12,13,20,23,24,28,31,36] Many of the studies measuring outcomes following DC in children are limited due to sample size. Patel et al. and Rutigliano et al. reported favorable outcomes in 100% and 83% of patients, respectively; however, their patient population was made up of seven and six patients, respectively. Thomale et al. remain the only pediatric randomized control trial on DC. They reported a favorable outcome in 92% of their 14 patients. There are, however, some larger studies that have also experienced good outcomes in 81% to nearly 100% of survivors.^[13,15,28] However, in a recent study by Bruns et al., patients undergoing a DC had less favorable outcomes, with patients more likely to experience death or an unfavorable outcome (27.6% vs. 16.1%).^[5] Our experience further demonstrates the positive outcome of pediatric patients following DC. Although our patients were in severe clinical distress on presentation, three-quarters (75%) of the survivors had favorable outcomes. This corroborates previous articles that DC can be a very effective tool for managing secondary brain injury and highlights the benefit of DC within the pediatric population.

Controversy surrounding the usage of DC is in part due to the relatively high complication rate.^[26] PTH, hygroma, infection, wound breakdown, and CSF fistula are all well-known complications following the procedure.^[26] Complication rates following DC have been reported to be from 0% to 100%, with most studies in the range of 14-40%.^[2,13,15,26,31] However, many of the studies are case series or small institutional reviews of <10 patients, so it is difficult to find comparable complication rates. The complication rate within our patient population was 31.9%, which is comparable to previously reported complication rates. In terms of wound breakdown, our infection rate (19.1%) and CSF fistula rate (12.8%) are similar to the previous studies, including one by Ballestero et al., who had an infection rate of 18.8%. Of our patients who had a CSF fistula (n = 6), all but one had a congruent infection.^[4] The infections were successfully treated with antibiotics and did not require surgical drainage. Kan et al. had an infection rate of 8.6% and noted that the patients who developed postoperative infections had scalps that were difficult to close at the time of the infection.^[15]

Furthermore, in a study by Adamo *et al.*, three patients (50%) developed CSF fistulas after the operation. Following fistula formation, EVDs were inserted to drain excess CSF.^[2]

Despite the high prevalence of PTH, few studies exist on the topic. PTH rates are usually reported to be around 21–40%, with one smaller study by Adamo *et al.* reporting rates as high as 100%.^[2,13,15,26,28] In this present study, only three patients (6.9%) developed hydrocephalus, which is well below the commonly reported average.^[6] It is possible that the lower incidence of PTH was due to the usage of postsurgical drains. About 55.3% of patients had an EVD in place following initial decompression, and 83.0% had a subgaleal drain. In prior studies in which EVDs were noted in patients, the total number of drains used was not reported. Other publications do not detail the use of EVDs and subgaleal drains in preventing PTH following DC, as our study suggests; therefore, there is no good comparison data.

In a study by Carballo-Cuello et al. specifically looking at PTH following DC, they found that PTH was more prominent in patients receiving later cranioplasties when compared to patients undergoing earlier cranioplasties.^[6] Patients with hydrocephalus had a mean time to cranioplasty of 272.3 days, whereas patients without hydrocephalus had a mean time to cranioplasty of 127.5 days. The earlier placement of the bone flap allows for earlier restoration of normal pressure within the cranium and CSF flow mechanics, thus decreasing the risk of hydrocephalus.^[6] In this present study, the mean time to cranioplasty was 26.93 days, as is institutional practice, which also potentially contributed to our low rates of PTH. Similarly, in a case report by Scollato et al., phase contrast MRI was utilized to image CSF flow dynamics in a patient who developed hydrocephalus after undergoing a DC.[33] MRI demonstrated arrested pulsatile CSF flow at the Sylvian aqueduct, which they believed was the cause of the hydrocephalus. Notably, after cranioplasty, CSF flux was restored at the aqueduct. Early cranioplasty in most of the patients could then be another contributor to our lower rates of hydrocephalus when compared to the average reported rates.

Another commonly encountered complication after DC is subdural and subgaleal hygromas. It is believed that the development of hygromas is linked to the development of PTH. A study by Pechmann *et al.* supports the notion that all patients who developed PTH had a preexisting subdural hygroma.^[6,26] Complication rates for hygromas have been reported to be as high as 83%; however, many studies do not report the incidence.^[26] The rate in our current study is 10.6%, which is below the commonly reported values. Most patients in this study also received an epidural drain for an average of 3.12 days following DC. We attribute the low number of hygromas to the ubiquitous usage of subgaleal drains and EVDs. Due to the association between the development of hygroma as a risk factor for PTH, it may also be possible that

our low rate of PTH can also be attributed to the control of hygroma formation with the use of subgaleal drains and EVDs.

With respect to the use of postoperative EVDs and subgaleal drains, we believe that these are pivotal in reducing complications and aiding in the healing process. Care must be taken in the usage of drains as it has been noted that oversiphoning of CSF can result in slit ventricle syndrome with spikes in ICP secondary to venous engorgement.^[34] The EVDs and subgaleal drains were set in such a way that the EVD did not drain too much CSF (and was adjusted when draining too much), and the subgaleal drain balanced the removal of CSF intraventrically such that ventricles did not collapse, causing drain failure. This is another potential benefit of the subgaleal drain in these circumstances.

Limitations

A limitation of this study is the retrospective nature, which can include bias due to the lack of randomization. Due to the small sample size coupled with the numerous covariables present in the study, we were not able to perform any multivariable regression analysis of independent predictors of outcome following DC. The sample size of 47 does hold some limitations on the generalizability of the study sample. Type II errors cannot be completely ruled out due to the pilot scale of the study sample. We were unable to capture the exact timing from injury to DC and the exact timing of ICP, CPP, and MAP measurements, which were collected as admission, preoperation, and postoperation. Finally, decision-making was surgeon preference, with four different surgeons operating on the patient population. Due to the pilot size of the study sample, we were able to collect substantial data on each patient. This gave us the benefit of having ample descriptive characteristics that were included within the study. A prospective study is now needed to adequately randomize patients and collect data that are not readily available within patient charts and should be considered for future work.

CONCLUSION

Timing to DC following arrival to the hospital after a TBI did not significantly correlate with better outcomes in the pediatric population. Pediatric patients have further demonstrated positive outcomes following DC, and our rates of PTH are the lowest recorded. This further demonstrates the safety and efficacy of DC in children following severe traumatic brain injuries; however, more studies with increased patient populations are needed.

Ethical approval

The author(s) declare that they have taken the ethical approval from IRB. (IRB approval number #00000096, 7/24/2020).

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

- 1. Adamo MA, Drazin D, Smith C, Waldman JB. Comparison of accidental and nonaccidental traumatic brain injuries in infants and toddlers: Demographics, neurosurgical interventions, and outcomes. J Neurosurg Pediatr 2009;4:414-9.
- 2. Adamo MA, Drazin D, Waldman JB. Decompressive craniectomy and postoperative complication management in infants and toddlers with severe traumatic brain injuries. J Neurosurg Pediatr 2009;3:334-9.
- Ardissino M, Tang A, Muttoni E, Tsang K. Decompressive craniectomy in paediatric traumatic brain injury: A systematic review of current evidence. Childs Nerv Syst 2018;35:209-16.
- Ballestero MF, Furlanetti LL, Augusto LP, Chaves PH, Santos MV, de Oliveira RS. Decompressive craniectomy for severe traumatic brain injury in children: Analysis of longterm neuropsychological impairment and review of the literature. Childs Nerv Syst 2019;35:1507-15.
- Bruns N, Kamp O, Lange K, Lefering R, Felderhoff-Müser U, Dudda M, *et al.* Functional short-term outcomes and mortality in children with severe traumatic brain injury: Comparing decompressive craniectomy and medical management. J Neurotrauma 2022;39:944-53.
- 6. Carballo-Cuello C, de Jesus O, Fernandez-de Thomas RJ, Garcia M, Vigo-Prieto J, de Jesus-Espinosa A. Posttraumatic hydrocephalus in pediatric patients after decompressive craniectomy. World Neurosurg 2020;136:e690-4.
- Csókay A, Emelifeonwu JA, Fügedi L, Valálik I, Láng J. The importance of very early decompressive craniectomy as a prevention to avoid the sudden increase of intracranial pressure in children with severe traumatic brain swelling (retrospective case series). Childs Nerv Syst 2011;28:441-4.
- Csókay A, Nagy L, Pentelénvi T. "Vascular tunnel" formation to improve the effect of decompressive craniectomy in the treatment of brain swelling caused by trauma and hypoxia. Acta Neurochir (Wien) 2001;143:173-5.

- 9. Cushing H. I. Subtemporal decompressive operations for the intracranial complications associated with bursting fractures of the skull. Ann Surg 1908;47:641-4.1.
- Elsawaf Y, Anetsberger S, Luzzi S, Elbabaa SK. Early decompressive craniectomy as management for severe traumatic brain injury in the pediatric population: A comprehensive literature review. World Neurosurg 2020;138:9-18.
- 11. Figaji AA, Fieggen AG, Argent AC, Le Roux PD, Peter JC. Intracranial pressure and cerebral oxygenation changes after decompressive craniectomy in children with severe traumatic brain injury. Acta Neurochir Suppl 2008;102:77-80.
- 12. Güresir E, Schuss P, Seifert V, Vatter H. Decompressive craniectomy in children: Single-center series and systematic review. Neurosurgery 2012;70:881-8.
- Jagannathan J, Okonkwo DO, Dumont AS, Ahmed H, Bahari A, Prevedello DM, *et al.* Outcome following decompressive craniectomy in children with severe traumatic brain injury: A 10-year single-center experience with longterm follow up. J Neurosurg 2007;106:268-75.
- 14. Josan VA, Sgouros S. Early decompressive craniectomy may be effective in the treatment of refractory intracranial hypertension after traumatic brain injury. Childs Nerv Syst 2006;22:1268-74.
- 15. Kan P, Amini A, Hansen K, White GL Jr., Brockmeyer DL, Walker ML, *et al.* Outcomes after decompressive craniectomy for severe traumatic brain injury in children. J Neurosurg 2006;105:337-42.
- 16. Khan SA, Shallwani H, Shamim MS, Murtaza G, Enam SA, Qureshi RO, *et al.* Predictors of poor outcome of decompressive craniectomy in pediatric patients with severe traumatic brain injury: A retrospective single center study from Pakistan. Childs Nerv Syst 2014;30:277-81.
- 17. Kochanek PM, Tasker RC, Bell MJ, Adelson PD, Carney N, Vavilala MS, *et al.* Management of pediatric severe traumatic brain injury: 2019 consensus and guidelines-based algorithm for first and second tier therapies. Pediatr Crit Care Med 2019;20:269-79.
- Kochanek PM, Tasker RC, Carney N, Totten AM, Adelson PD, Selden NR, *et al.* Guidelines for the management of pediatric severe traumatic brain injury, third edition: Update of the brain trauma foundation guidelines. Pediatr Crit Care Med 2019;20:S1-82.
- 19. Kurland DB, Khaladj-Ghom A, Stokum JA, Carusillo B, Karimy JK, Gerzanich V, *et al.* Complications associated with decompressive craniectomy: A systematic review. Neurocrit Care 2015;23:292-304.
- 20. Manfiotto M, Beccaria K, Rolland A, Paternoster G, Plas B, Boetto S, *et al.* Decompressive craniectomy in children with severe traumatic brain injury: A multicenter retrospective study and literature review. World Neurosurg 2019;129:e56-62.
- 21. McCowan CL, Swanson ER, Thomas F, Handrahan DL. Outcomes of pediatric trauma patients transported from rural and urban scenes. Air Med J 2008;27:78-83.
- 22. McMillan T, Wilson L, Ponsford J, Levin H, Teasdale G, Bond M. The Glasgow outcome scale 40 years of application and refinement. Nat Rev Neurol 2016;12:477-85.
- 23. Mhanna MJ, Mallah WE, Verrees M, Shah R, Super DM.

Outcome of children with severe traumatic brain injury who are treated with decompressive craniectomy. J Neurosurg Pediatr 2015;16:508-14.

- 24. Patel N, West M, Wurster J, Tillman C. Pediatric traumatic brain injuries treated with decompressive craniectomy. Surg Neurol Int 2013;4:128.
- 25. Patel PD, Kelly KA, Chen H, Greeno A, Shannon CN, Naftel RP. Measuring the effects of institutional pediatric traumatic brain injury volume on outcomes for rural-dwelling children. J Neurosurg Pediatr 2021;28:638-46.
- 26. Pechmann A, Anastasopoulos C, Korinthenberg R, van Velthoven-Wurster V, Kirschner J. Decompressive craniectomy after severe traumatic brain injury in children: Complications and outcome. Neuropediatrics 2015;46:5-12.
- 27. Polin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, Bocchicchio B, *et al.* Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. Neurosurgery 1997;41:84-92.
- Prasad GL, Gupta DK, Mahapatra AK, Borkar SA, Sharma BS. Surgical results of growing skull fractures in children: A single centre study of 43 cases. Childs Nerv Syst 2015;31:269-77.
- 29. Prasad GL, Gupta DK, Mahapatra AK, Sharma BS. Surgical results of decompressive craniectomy in very young children: A level one trauma centre experience from India. Brain Inj 2015;29:1717-24.
- Rallis D, Poulos P, Kazantzi M, Kalampalikis P. Rescue decompressive craniectomy in children with severe traumatic brain injury. J Pediatr Intensive Care 2018;7:33-8.
- 31. Ruf B, Heckmann M, Schroth I, Hügens-Penzel M, Reiss I, Borkhardt A, *et al.* Early decompressive craniectomy and duraplasty for refractory intracranial hypertension in children: Results of a pilot study. Crit Care 2003;7:R133-8.
- 32. Rutigliano D, Egnor MR, Priebe CJ, McCormack JE, Strong N,

Scriven RJ, *et al.* Decompressive craniectomy in pediatric patients with traumatic brain injury with intractable elevated intracranial pressure. J Pediatr Surg 2006;41:83-7.

- Scollato A, Gallina P, Bahl G, Di Lorenzo N. Decompressive craniectomy arrests pulsatile aqueductal CSF flux: An *in vivo* demonstration using phase-contrast MRI. Case report. Br J Neurosurg 2015;29:440-2.
- Sood S, Kumar CR, Jamous M, Schuhmann MU, Ham SD, Canady AI. Pathophysiological changes in cerebrovascular distensibility in patients undergoing chronic shunt therapy. J Neurosurg 2004;100:447-53.
- 35. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, *et al.* A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. Childs Nerv Syst 2001;17:154-62.
- Thomale UW, Graetz D, Vajkoczy P, Sarrafzadeh AS. Severe traumatic brain injury in children-a single center experience regarding therapy and long-term outcome. Childs Nerv Syst 2010;26:1563-73.
- Wilson MH. Monro-Kellie 2.0: The dynamic vascular and venous pathophysiological components of intracranial pressure. J Cereb Blood Flow Metab 2016;36:1338-50.
- 38. Zhang D, Xue Q, Chen J, Dong Y, Hou L, Jiang Y, *et al.* Decompressive craniectomy in the management of intracranial hypertension after traumatic brain injury: A systematic review and meta-analysis. Sci Rep 2017;7:8800.

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Supplementary Figure 1: Power analysis. SMD: Standardized mean difference.