Posttraumatic Hydrocephalus in Pediatric Patients After Decompressive Craniectomy

Cesar Carballo-Cuello, Orlando de Jesus, Ricardo J. Fernandez-de Thomas, Maria Garcia, Juan Vigo-Prieto, Aixa de Jesus-Espinosa

■ OBJECTIVE: **The risk for developing posttraumatic hydrocephalus (PTH) is higher when patients undergo decompressive craniectomy as part of their treatment. The purpose of this study is to determine the prevalence of PTH after decompressive craniectomy in pediatric patients and determine associated risk factors that may lead to PTH.**

■ METHODS: **A retrospective analysis was conducted by searching the Puerto Rico neurologic surgery database from 2010 to 2019. All pediatric patients (1–18 years old) at the University Pediatric Hospital of the Puerto Rico Medical Center who had traumatic brain injury and had a decompressive craniectomy were included in the study. Data were reviewed to determine if time to decompressive craniectomy, side of decompressive craniectomy, gender, mechanism of trauma, amount of subarachnoid hemorrhage, and time to cranioplasty were risk factors for the development of PTH.**

■ RESULTS: **Incidence of PTH after decompressive craniectomy was 21%. Neither gender, side of decompressive craniectomy, mechanism of trauma, amount of subarachnoid hemorrhage, time from trauma to decompressive craniectomy, nor cranioplasty intervention had statistical significance for developing PTH. Time from decompressive craniectomy to cranioplasty was significant for development of PTH.**

■ CONCLUSIONS: **Longer time to cranioplasty was associated with an increased likelihood of PTH. We recommend performing cranioplasty as soon as possible to reduce hydrocephalus development.**

INTRODUCTION

Posttraumatic hydrocephalus (PTH) is an active and progressive process of excessive cerebrospinal fluid (CSF) accumulation due to fluid dynamic disturbances following craniocerebral injury. The incidence of PTH has been previously cited to occur from 0.7%–54%.[1-3] In the pediatric population, PTH occurs in approximately 1%[4,5] of children who sustained a traumatic brain injury (TBI) and is more common in those younger than 1 year of age.[4] The risk for developing PTH is higher when patients undergo decompressive craniectomy (DC) as part of their treatment. Studies have shown that in pediatric patients the incidence can range from 15%–40% after DC.[6] Longer duration of coma, increased age, DC, and subarachnoid hemorrhage (SAH) have been reported to increase the risk of developing PTH.[7] The diagnosis of hydrocephalus requires the demonstration of dilated ventricles without sulci enlargement and the development of any associated clinical symptoms, such as headache, nausea/vomiting, and decreased neurologic status.[1] PTH must be differentiated from ventricular enlargement secondary to posttraumatic cerebral atrophy.

The etiology and associated risk factors of PTH must be determined for proper treatment and prevention. Previous studies have shown that factors such as altered intracranial pressure dynamics, mechanical blockage, or inflammation of the arachnoid granulations by postsurgical debris may contribute to developing hydrocephalus.[1] The arachnoid granulations function as pressure-dependent 1-way valves from the subarachnoid space to the draining venous sinuses, making it possible that disruption of pulsatile intracranial pressure dynamics secondary to opening the cranial vault results in decreased CSF outflow.[8] In cases of SAH and traumatic intracranial hematoma, mechanical blockage or inflammation of the arachnoid granulations might promote hydrocephalus.[8]

Key words
- Decompressive craniectomy
- Hydrocephalus
- Pediatric
- Traumatic brain injury

Abbreviations and Acronyms
CSF: Cerebrospinal fluid
CT: Computed tomography
DC: Decompressive craniectomy
PTH: Posttraumatic hydrocephalus
SAH: Subarachnoid hemorrhage
TBI: Traumatic brain injury

Section of Neurosurgery, School of Medicine, University of Puerto Rico, Medical Sciences Campus, San Juan, Puerto Rico, USA

To whom correspondence should be addressed: Orlando de Jesus, M.D. [E-mail: drodejesus@aol.com]

Citation: World Neurosurg. (2020).
https://doi.org/10.1016/j.wneu.2020.01.153

*Journal homepage: **www.journals.elsevier.com/world-neurosurgery***

*Available online: **www.sciencedirect.com***

In pediatric patients, DC only provides benefit in select cases and therefore should only be performed following strict guidelines. Although the mortality rate for children with severe TBI remains high, DC is effective in reducing intracranial pressure and is associated with good outcomes on appropriate cases.[7] The purpose of this study is to determine the prevalence of PTH after DC in pediatric patients and associated risk factors that may lead to PTH.

METHODS

This study was performed at the University Pediatric Hospital of the Puerto Rico Medical Center and approved by the University of Puerto Rico Medical Sciences Campus Institutional Review Board. A retrospective analysis was conducted by searching the neurologic surgery database from November 2010 to March 2019. Inclusion criteria for the study consisted of the following: pediatric patients (1–18 years old) who had TBI and had undergone DC and patients required to have a head computed tomography (CT) scan or a brain magnetic resonance imaging at least 6 months after DC. Clinical and imaging data were reviewed from patient's charts to determine if time to DC, side of DC, gender, mechanism of trauma, amount of SAH on the initial head CT scan, and time to cranioplasty were risk factors for the development of PTH. PTH was defined and confirmed by radiologic evidence of progressive ventricular dilatation (Evans index of at least 0.3) and the presence of clinical deterioration resulting in neurologic worsening over time. The amount of SAH for each case was determined using the Morris-Marshall classification using the initial head CT scan.[9] Cranioplasty was defined as the surgical repair of a defect or deformity of the skull using an autologous bone graft, which was previously saved in the patient's abdomen or an allograft mesh. Uniform criteria and indications for DC were used for the entire series and study time frame. Similar uniform criteria for cranioplasty were used. The endpoint for the surveillance for hydrocephalus was until the cranioplasty was performed. To determine the relationship between the variables of interest and development of posttraumatic hydrocephalus, Fisher exact test and Pearson point-biserial correlation were estimated for categorical and continuous variables, respectively. A P value <0.05 was considered statistically significant. Statistical analysis was performed using R (version 3.4.4).

RESULTS

A total of 39 patients who had TBI and underwent DC were identified between 2010 and 2019 in the Puerto Rico Medical Center Neurological Surgery database. Of these patients, only 34 had clinical and radiologic follow-up at 6 months. The sample mean age was 12.6 (SD = 5.2) years and consisted of 9 females (26%) and 25 males (74%). In 15 cases, the DC was performed on the right side, in 17 cases it was on the left. One bifrontal and 1 bilateral DCs were performed (Table 1). The mechanisms of trauma were widely varied; motor vehicle and pedestrian accidents were the most frequent. The amount of SAH was also widely varied in each case. Twenty-four (70%) cases had a cranioplasty procedure performed after DC. A patient was deemed ready for the cranioplasty as soon as he or she was medically stable to undergo general anesthesia, was free of any infectious source, and brain swelling had decreased to the point where the skin flap was soft and slightly below the bone edge to accommodate the graft. A few patients received the cranioplasty during the initial hospitalization, but the majority were performed during a subsequent hospitalization. If a patient developed permanent hydrocephalus before the cranioplasty, he or she was treated with a shunt and underwent cranioplasty as soon as possible. Ten patients did not have a cranioplasty performed because they died sometime after the inclusion criteria of the 6-month radiologic follow-up was met or the parents did not consent for the surgery due to the critical comatose state of their child. Seven (21%) patients developed PTH after DC.

Table 1. Demographics; Sample Consisted of 34 Cases of Decompressive Craniectomy

Characteristic	Number	%
Sex		
Female	9	26.5
Male	25	73.5
Side		
Left	17	50.0
Right	15	44.1
Bifrontal	1	2.9
Left and right	1	2.9
Cranioplasty		
Yes	24	70.5
No	10	29.4
Hydrocephalus		
Yes	7	20.6
No	27	79.4
Mechanism		
All-terrain vehicle	4	11.8
Bicycle	1	2.9
Ground-level fall	2	5.9
Gunshot wound	3	8.8
Horse	3	8.8
Moto	5	14.7
Motor vehicle accident	8	23.5
Pedestrian	7	20.6
Unknown	1	2.9
Subarachnoid hemorrhage grade		
0	4	11.8
1	11	32.4
2	8	23.5
3	2	5.9
4	9	26.4

Table 2. Risk Factors for Posttraumatic Hydrocephalus Patients

	Hydrocephalus				
	Yes		No		
Risk Factor	Number	%	Number	%	*P* Value
Sex					1
Female	2	22.2	7	77.7	
Male	5	20.0	20	80.0	
Side					0.4025
Left	5	29.4	12	70.5	
Right	2	13.3	13	81.2	
Cranioplasty					0.3943
Yes	4	16.7	20	83.3	
No	3	30.0	7	70.0	
Mechanism					0.2664
All-terrain vehicle	1	25.0	3	75.0	
Bicycle	—	—	1	100	
Ground-level fall	—	—	2	100	
Gunshot wound	2	66.7	1	33.3	
Horse	—	—	3	100	
Moto	—	—	5	100	
Motor vehicle accident	2	25.0	6	75.0	
Pedestrian	1	14.3	6	85.7	
Unknown	1	100	—	0	
Subarachnoid hemorrhage grade					0.6642
0	1	25.0	3	75.0	
1	4	36.4	7	63.6	
2	1	12.5	7	87.5	
3	—	—	2	100	
4	1	11.1	8	88.8	

We analyzed the 7 cases that developed PTH with 5 different variables to determine if gender, side of DC, mechanism of trauma, amount of SAH on the initial head CT scan, or cranioplasty had an association with the development of hydrocephalus, but no statistically significant association for developing PTH was found (Table 2). Using point-biserial correlation, we evaluated if age, time from traumatic event to DC (days), and time to cranioplasty (days) were statistically significant. Our findings demonstrated that mean age for the nonhydrocephalus group was 13.3 and 11.9 for the PTH group, respectively, with a P value of 0.577 (Table 3). In the PTH group, time from trauma to DC showed a mean of 1.0 days and in the nonhydrocephalus group there was a mean of 2.2 days with a P value of 0.333. Time from DC to cranioplasty was measured between the PTH and the nonhydrocephalus group, and the average time in days was 272.3 in the PTH group and 127.5 in the nonhydrocephalus group with a P value of 0.034.

DISCUSSION

TBI is one of the most common causes of death and disability in young people. In a recent meta-analysis including adult and pediatric patients, it was reported that PTH in patients undergoing DC was 17.7%.[6] Our results demonstrate that the incidence of PTH after DC was 21% in pediatric patients, which is smaller when compared with 37.6% reported by the meta-analysis by Fattahian et al,[6] when only the pediatric population was included. Others have reported a pediatric incidence of PTH in 29%–42% of the

Table 3. Association Between Variables and Development of Posttraumatic Hydrocephalus

	Hydrocephalus			
Risk Factor	**Yes Mean (SD)**	**No Mean (SD)**	**Correlation Coefficient**	***P* Value**
Age	11.9 (6.6)	13.3 (5.7)	−0.101	0.577
Time to decompressive craniectomy (days)	1.0 (1.3)	2.2 (3.0)	−0.174	0.333
Time to cranioplasty (days)	272.3 (121.2)	127.5 (116.5)	0.433	0.034

patients with DC.[7,10,11] Several studies have shown significant factors that may lead to PTH after DC. Honeybul and Ho[12] found that the development of hydrocephalus was associated with low-admission Glasgow Coma Scale score and subdural hygroma on head CT. Poor Glasgow Coma Scale score ($\leq$8) or the severity of brain injury and older age ($\geq$65) has also been implicated for high incidence of PTH.[13] Our study data comprise exclusively a pediatric population. We tried to determine if time to DC and/or time to cranioplasty had any effect in PTH in this pediatric population. Randomized controlled trials have shown that younger age is associated with shunt-dependent hydrocephalus after DC.[14] In our study we found that neither gender, age, side of DC, mechanism of trauma, amount of SAH on the initial head CT scan, nor cranioplasty had any statistical significance as a risk factor for developing hydrocephalus. Our results depicted a tendency that linked earlier DC with the PTH group, with the patients who developed PTH having an average time from initial trauma to DC of 1 day in comparison with the nonhydrocephalus group of 2.2 days, as shown in Table 3. It is important to note that an earlier DC is usually associated with a greater severity of the brain injury, which may contribute to the development of PTH.

Patients who underwent earlier cranioplasty were most commonly found in the nonhydrocephalus group. The average in days from DC to cranioplasty was 127.5 in the non-PTH group in comparison with 272.3 in the PTH group. Our findings correlate with previous studies performed by Bonow et al[4] in children and Nasi et al[15] in adults, who showed that delayed cranioplasty was associated with a greater increased likelihood of PTH. Early cranioplasty in children who require craniectomy may reduce the risk for PTH.[4] We think that the cause of PTH after DC is multivariate, and several risk factors may influence its development.

It is important to note that there are certain limitations associated with our results. The small sample size and subsequent small subgroup of patients that developed PTH was a significant limitation in the present study. Only 7 cases (21%) developed the outcome of interest, which was the development of PTH. In addition, the results could have a bias associated with the severity of illness and health status between the analyzed groups. It is likely that DC was performed earlier on patients who reached the maximum threshold of intracranial pressure and had more severe TBI, which could have been the etiology responsible for causing CSF flow disturbances. The time to cranioplasty may also have been affected by this bias as the patients with severe TBI may require more time for optimization for cranioplasty.

CONCLUSIONS

Late cranioplasty was associated with an increased likelihood of PTH. We recommend performing cranioplasty as soon as possible to reduce hydrocephalus development. Our medical center is the only trauma center in Puerto Rico for the pediatric and adult population; therefore our study serves as a framework for further research to obtain knowledge of this pathology. It is a significant problem that needs to be identified because it carries tremendous financial, emotional, and social burden. Future studies with larger population delineating the risk factors are necessary for prompt and proper management.

CRediT AUTHORSHIP CONTRIBUTION STATEMENT

Cesar Carballo-Cuello: Writing - original draft. **Orlando de Jesus:** Writing - original draft. **Ricardo J. Fernandez-de Thomas:** Writing - original draft. **Maria Garcia:** Writing - original draft. **Juan Vigo-Prieto:** Writing - original draft. **Aixa de Jesus-Espinosa:** Writing - original draft.

REFERENCES

1. Choi I, Park HK, Chang JC, Cho SJ, Choi SK, Byun BJ. Clinical factors for the development of posttraumatic hydrocephalus after decompressive craniectomy. *J Kor Neurosurg Soc*. 2008;43:227-231.

2. De Bonis P, Pompucci A, Mangiola A, Rigante L, Anile C. Post-traumatic hydrocephalus after decompressive craniectomy: an underestimated risk factor. *J Neurotrauma*. 2010;27:1965-1970.

3. Di G, Hu Q, Liu D, Jiang X, Chen J, Liu H. Risk factors predicting posttraumatic hydrocephalus after decompressive craniectomy in traumatic brain injury. *World Neurosurg*. 2018;116:e406-e413.

4. Bonow RH, Oron AP, Hanak BW, et al. Post-traumatic hydrocephalus in children: a retrospective study in 42 pediatric hospitals using the pediatric health information system. *Clin Neurosurg*. 2018;83:732-739.

5. Rumalla K, Letchuman V, Smith KA, Arnold PM. Hydrocephalus in pediatric traumatic brain injury: national incidence, risk factors, and outcomes in 124,444 hospitalized patients. *Pediatr Neurol*. 2018; 80:70-76.

6. Fattahian R, Bagheri SR, Sadeghi M. Development of posttraumatic hydrocephalus requiring ventriculoperitoneal shunt after decompressive craniectomy for traumatic brain injury: a systematic review and meta-analysis of retrospective studies. *Med Arch*. 2018;72: 214-219.

7. Jagannathan J, Okonkwo DO, Dumont AS, et al. Outcome following decompressive craniectomy in children with severe traumatic brain injury: a 10-year single-center experience with long-term follow up. *J Neurosurg Pediatr*. 2007;106:268-275.

8. Waziri A, Fusco D, Mayer SA, McKhann GM, Connolly ES. Postoperative hydrocephalus in patients undergoing decompressive hemicraniectomy for ischemic or hemorrhagic stroke. *Neurosurgery*. 2007;61:489-493.

9. Morris GF, Marshall LF. A new, practical classification of traumatic subarachnoid hemorrhage. *Clin Neurol Neurosurg (Supp1)*. 1997;99:S16.

10. 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.

11. Kan P, Amini A, Hansen K, et al. Outcomes after decompressive craniectomy for severe traumatic brain injury in children. *J Neurosurg Pediatr*. 2006;105:337-342.

12. Honeybul S, Ho KM. Incidence and risk factors for post-traumatic hydrocephalus following decompressive craniectomy for intractable intracranial hypertension and evacuation of mass lesions. *J Neurotrauma*. 2012;29:1872-1878.

13. Ban SP, Son YJ, Yang HJ, Chung YS, Lee SH, Han DH. Analysis of complications following decompressive craniectomy for traumatic brain injury. *J Kor Neurosurg Soc*. 2010;48:244-250.

14. Vedantam A, Yamal JM, Hwang H, Robertson CS, Gopinath SP. Factors associated with shunt-dependent hydrocephalus after decompressive craniectomy for traumatic brain injury. *J Neurosurg*. 2018;128:1547-1552.

15. Nasi D, Gladi M, Di Rienzo A, et al. Risk factors for post-traumatic hydrocephalus following decompressive craniectomy. *Acta Neurochir*. 2018;160:1691-1698.

Conflict of interest statement: The author declares that the article content was composed in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Received 12 October 2019; accepted 20 January 2020

Citation: World Neurosurg. (2020).
https://doi.org/10.1016/j.wneu.2020.01.153

Journal homepage: ***www.journals.elsevier.com/world-neurosurgery***

Available online: ***www.sciencedirect.com***