Post-Traumatic Hydrocephalus in Children: A Retrospective Study in 42 Pediatric Hospitals Using the Pediatric Health Information System

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BACKGROUND: Post-traumatic hydrocephalus (PTH) is a potentially treatable cause of poor recovery from traumatic brain injury (TBI) that remains poorly understood, particularly among children.

OBJECTIVE: To better understand the risk factors for pediatric PTH using a large, multi-institutional database.

METHODS: We conducted a retrospective cohort study using administrative data from 42 pediatric hospitals participating in the Pediatric Health Information System. All patients ≤21 yr surviving a hospitalization with an International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) code for TBI were identified. The primary outcome was PTH, defined by an ICD-9-CM procedure code for surgical management of hydrocephalus within 6 mo. Data were analyzed using multivariable logistic regression.

RESULTS: We identified 91 583 patients ≤21 yr with TBI, 846 of whom developed PTH. Odds of PTH were significantly higher in children <1 yr compared to older age groups. A total of 48.7% of PTH cases were victims of abuse (adjusted odds ratio [aOR] 2.62, 95% confidence interval [CI] 2.16-3.18). PTH was more common after craniotomy (aOR 1.60, 95% CI 1.30-1.97). Craniectomy without early cranioplasty was associated with markedly increased odds of PTH (aOR 3.67, 95% CI 2.66-5.07), an effect not seen in those undergoing cranioplasty within 30 d (aOR 1.19, 95% CI 0.75-1.89).

CONCLUSION: PTH was seen in 0.9% of children who sustained a TBI and was more common in those <1 yr. Severe injury, abuse, and craniectomy with delayed cranioplasty were associated with greatly increased likelihood of PTH. Early cranioplasty in children who require craniectomy may reduce the risk for PTH.

KEY WORDS: Craniotomy, Decompressive craniectomy, Hydrocephalus, Pediatrics, Traumatic brain injury

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Hydrocephalus has been a recognized sequela of traumatic brain injury (TBI) since its initial description by Dandy and Blackfan in 1914.[1] Post-traumatic hydrocephalus (PTH) may present with headache, nausea, and lethargy in patients with satisfactory neurological recovery after injury. Impaired cognitive function, neurological deficits, and autonomic dysfunction can also occur, and some patients demonstrate regression in functional status or delayed recovery.[2-4] Those who remain comatose may not demonstrate overt signs of PTH other than enlarged ventricles and transependymal flow on imaging.[5,6] Importantly, treatment of PTH with cerebrospinal fluid (CSF) diversion can lead to clinical improvement in appropriately selected patients.[2,4,6]

PTH, therefore, represents an important, treatable cause of poor recovery from TBI, yet risk factors remain poorly understood. This knowledge deficit is particularly pronounced among the pediatric population, where little research on this topic has been conducted. In

ABBREVIATIONS: aOR, adjusted odds ratio; **CI,** confidence interval; **CSF,** cerebrospinal fluid; **ICP,** intracranial pressure; **ICU,** intensive care unit; **PHIS,** Pediatric Health Information System; **PTH,** post-traumatic hydrocephalus; **SAH,** subarachnoid hemorrhage; **TBI,** traumatic brain injury

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the studies that have been performed in adult and mixed-age populations, PTH appears to be relatively rare among TBI survivors, with a prevalence of between 0.7% and 3.6%.[2,7] Studies focusing solely on more severely injured patients report higher rates between 2.4% and 45%, suggesting that patients with more severe TBI are at higher risk.[3,4,6,8] The same pattern is observed in patients who undergo decompressive craniectomy for severe TBI, where 34.5% of adults and 40% of children are reported to develop PTH.[9,10]

However, many of these publications do not directly compare TBI patients who develop hydrocephalus to those who do not, thereby limiting their ability to reliably report characteristics associated with PTH. From those that do make this comparison, the results have been inconsistent. Low Glasgow Coma Scale, advanced age, traumatic subarachnoid hemorrhage (SAH), traumatic intraventricular hemorrhage, and decompressive craniectomy have all been associated with hydrocephalus in some studies, but not others.[3,6,8,9,11,12]

These variable results may be due to differences in patient-selection criteria and nonstandard definitions of hydrocephalus; some authors have relied solely on radiographic findings, whereas others incorporated clinical information, including intracranial pressure measurements. Furthermore, the existing literature is derived exclusively from single-institutional series, which limits sample sizes and increases the likelihood that differences in practice between centers could skew results. Finally, PTH can present anywhere from weeks to months after injury,[2-4] which may complicate identification of cases in research studies using data from single admissions.

To bridge this knowledge gap and improve our understanding of PTH in children, we conducted a retrospective cohort study using the Pediatric Health Information System (PHIS), an administrative dataset that receives contributions from not-for-profit, freestanding pediatric hospitals in the United States. Uniquely, PHIS allows linkage of serial healthcare encounters over time, enabling identification of delayed outcomes. The aim of this study was to identify risk factors associated with the development of PTH in children.

METHODS

Study Design and Participants

This study was approved by the local institutional review board, which waived the need for patient consent. The release of PHIS data was approved by the Child Health Corporation of America. Data from PHIS were evaluated for quality and completeness. Duplicate records and those submitted by facilities with known quality issues were removed prior to analysis.

Study participants consisted of all patients aged 21 yr and younger who were discharged from a PHIS hospital between January 2004 and March 2015 with one or more of the standard International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) diagnosis codes for TBI (800.0-801.9, 803.0-804.9, 850.0-854.1, 950.1-950.3, 959.01, and 995.55).[13] To ensure specific capture of clinically important hydrocephalus, PTH was defined based on the presence of an ICD-9-CM procedure code for either an endoscopic third ventriculostomy (ETV) or placement of a CSF shunt (02.31, 02.32, 02.33, 02.34, 02.35, 03.71, 03.72, and 03.79) within 6 mo of admission for the index TBI.

We minimized the likelihood of including children with hydrocephalus due to other etiologies by excluding those with conditions known to be associated with hydrocephalus, such as brain neoplasm, neural tube defects, and intraventricular hemorrhage of the newborn. Children who died during the index hospitalization or who had ICD-9-CM procedure or diagnostic codes related to hydrocephalus in their records prior to the index trauma were also excluded. To minimize bias from early mortality, we did not include patients with a known malignancy.

Variable Definitions

Detailed information was extracted about each patient's TBI admission, including demographic characteristics, ICD-9-CM coding data, medications administered, and the presence of an intensive care unit (ICU) admission. Age was treated as a categorical variable, broken into 4 groups of approximately equal size using standard cutoffs.[14] Procedure codes entered on subsequent inpatient visits after the index hospitalization were also recorded. Neurosurgical interventions were categorized into intracranial pressure (ICP) monitor placement (either with an external ventricular drain or another type of ICP monitor; ICD-9-CM procedure codes 01.09, 01.10, 01.16, 01.17, and 02.21), craniotomy (01.24, 01.23, 01.31, 01.32, 01.39, and 02.02), craniectomy (01.25), and cranioplasty (02.03, 02.04, 02.05, and 02.06). Due to inconsistent nomenclature and coding practice variations for craniotomy and craniectomy, craniotomies followed by a cranioplasty within 6 mo were reclassified as craniectomies. Abusive head trauma was identified using the relevant ICD-9-CM E-codes and diagnosis codes for inflicted injuries (E967.0-E967.9 and 995.50-995.59). This diagnosis is based on confirmed evidence of child abuse; suspected abuse is indicated by a separate set of ICD-9-CM codes, which were not considered in our analysis.

Because PHIS contains purely administrative records without neurological examinations, physiological data, or imaging results, we created proxy measures for TBI severity using the available information. The use of an ICP monitor, hypertonic saline, or mannitol served as indicators of a severe TBI. Gastrostomy (ICD-9-CM 43.11 and 43.19) or tracheostomy (ICD-9-CM 31.1, 31.21, and 31.29) was used as surrogate measures of poor neurological outcome.

Finally, we studied the 2-yr shunt failure rate in the subset of patients who were treated with a CSF shunt 2 or more years prior to the end of the study period. To obtain this information, billing records were queried for ICD-9-CM procedure codes for shunt removal or revision (02.41, 02.42, 02.43, 03.97, and 03.98) within 2 yr of the original shunt placement.

Statistical Analysis

The characteristics of patients with and without PTH were summarized descriptively, and bivariate relationships were tested for statistical significance using Pearson's chi-square test. The primary analysis tested associations between predictor variables and PTH using a multivariable logistic regression, adjusting for facility as a fixed covariate to account for practice pattern variations across hospitals. The following data were included in the model: age, gender, race/ethnicity, payer status, child abuse, specific TBI diagnoses, ICU admission, ICP monitor use, receipt of hyperosmolar therapy, need for gastrostomy or tracheostomy,

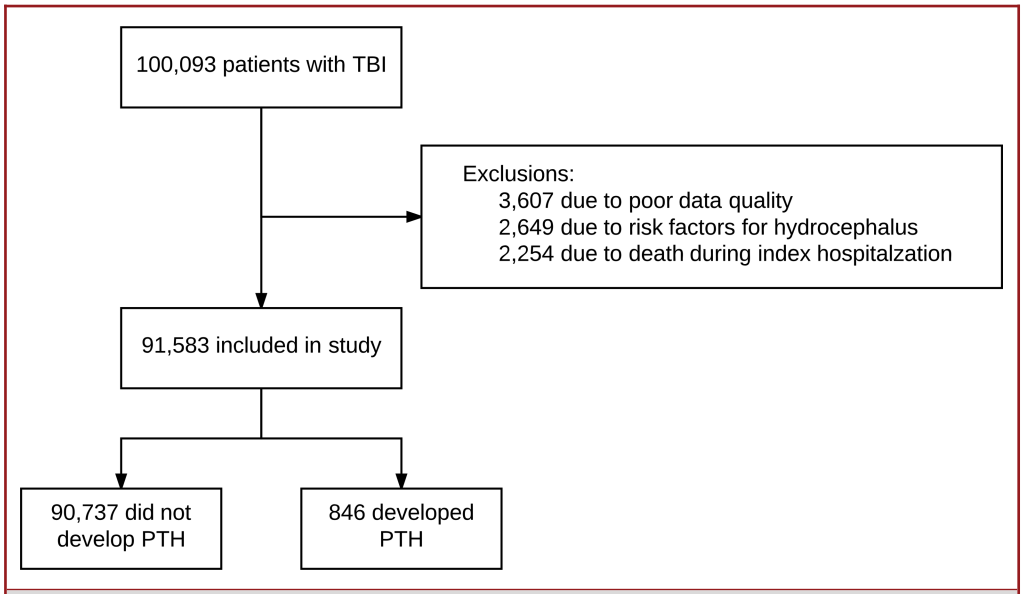

FIGURE. *Creation of the study cohorts. TBI, traumatic brain injury; PTH, post-traumatic hydrocephalus.*

and surgical management of the index TBI. Since others have reported possible associations between the timing of cranioplasty and hydrocephalus,[15] a 3-tiered craniectomy variable was created: no craniectomy, craniectomy followed by cranioplasty within 30 d, and craniectomy not followed by cranioplasty within 30 d. If a patient developed hydrocephalus within 30 d, only cranioplasty procedures performed prior to shunt placement or ETV were included in these definitions. A sensitivity analysis was conducted using the same modeling technique in patients who required hyperosmolar therapy or ICP monitoring to examine these relationships among patients with severe TBI. The null hypothesis was rejected for $P < .05$. Data processing and statistical analysis were performed using R statistical software version 3.3.2 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

A total of 100 093 children with TBI-related admissions and complete records were identified. After excluding those with data quality issues, 96 486 patients from 42 separate facilities remained. Of these, 2649 were excluded due to either pre-existing hydrocephalus or other conditions known to be associated with a high risk of hydrocephalus. A further 2254 children did not survive the index hospitalization, leaving a final cohort of 91 583 children with TBI. A total of 846 required surgical management of hydrocephalus within 6 mo, yielding a PTH rate of 0.9% (Figure).

The demographic and clinical characteristics of patients with and without PTH are shown in Table 1. Patients with PTH tended to be younger and to be recipients of government health insurance. Concussion and skull fracture without intracranial hemorrhage were rare in the PTH group, whereas subdural hematoma and skull fracture with intracranial hemorrhage were more common.

The median time to intervention for PTH was 21 d (interquartile range, 7-49). 62.8% of patients with PTH received a shunt or an ETV within 30 d of the trauma, and 66% were treated during the index hospitalization. Patients whose shunts were placed during the index hospitalization had longer lengths of stay, were more likely to have been female, and were more likely to be older. These children were also more likely to have received an ICP monitor, hyperosmolar therapy, and a gastrostomy or tracheostomy; surgical management with craniotomy and subdural hematomas were more common in those who developed PTH after discharge (**Table**, **Supplemental Digital Content 1**).

Among those treated for PTH, 92.8% received a ventriculoperitoneal shunt, 2.1% another type of ventricular shunt, and 5.2% an ETV. 698 children treated with a ventricular CSF shunt had 2 yr of follow-up data available, and of these 24% required one or more repeat shunt operations in that timeframe.

Patients with PTH tended to require more medical and procedural interventions than those without PTH (Table 2). ICU admission, hyperosmolar therapy, and ICP monitor use were all more common among children who ultimately developed PTH. These children also had a higher likelihood of poor neurological outcomes, as evidenced by the greater proportion of these patients who required placement of a gastrostomy or a tracheostomy.

In a multivariable logistic regression, young age was an important predictor of PTH (Table 3). Compared to children aged less than 1 yr, those between 1 and 5 yr were at significantly lower odds of PTH; the odds decreased further among children aged 6 to 11, where it plateaued. Additional factors

TABLE 1. Demographic and Clinical Characteristics

	No PTH		PTH		
	n = 90 737	%	n = 846	%	*P*-value
Age in years					<.001
<1	23 197	25.6%	499	59.0%	
1-5	25 085	27.6%	196	23.2%	
6-11	19 309	21.3%	66	7.8%	
12-21	23 146	25.5%	85	10.0%	
Male gender	57 421	63.3%	549	64.9%	.35
Insurance					<.001
Private/other	47 556	52.4%	271	32.0%	
Government	43 181	47.6%	575	68.0%	
Race/ethnicity					<.001
White	49 523	54.6%	395	46.7%	
African American	14 094	15.5%	164	19.4%	
Hispanic/Latino	15 997	17.6%	157	18.6%	
Other	11 123	12.2%	130	15.3%	
Diagnosis[a]					
Concussion	14 671	16.2%	4	0.5%	<.001
Skull fracture without hemorrhage	23 127	25.5%	12	1.4%	<.001
Skull fracture with hemorrhage	24 051	26.5%	324	38.3%	<.001
Cerebral contusion	3582	3.9%	46	5.4%	.03
SAH	3520	3.9%	71	8.4%	<.001
Subdural hematoma	7495	8.3%	401	47.4%	<.001
Epidural hematoma	1952	2.2%	11	1.3%	.11
Other intracranial hemorrhage	13 603	15.0%	28	3.3%	<.001
Child abuse	5306	5.8%	412	48.7%	<.001

[a]Some patients have multiple diagnosis codes. PTH, post-traumatic hydrocephalus.

independently associated with PTH included abusive head trauma and the specific types of TBI present; subdural hematoma increased the likelihood of requiring treatment for hydrocephalus, whereas children who had concussions or skull fractures without intracranial hemorrhage had lower odds of developing PTH. More aggressive care, a proxy for injury severity, also correlated with the odds; ICU admission, hyperosmolar therapy, and ICP monitor placement were all independently associated with PTH. Poor neurological outcome as defined by the need for gastrostomy or tracheostomy was independently associated with hydrocephalus. Most of these associations were maintained in a sensitivity analysis incorporating only patients who were injured severely enough to require ICP monitoring or hyperosmolar therapy, although the contributions of individual diagnoses were less evident (Table 3).

In the main model including children with any TBI, craniotomy was associated with significantly increased odds of PTH (Table 3). Children who required craniectomy were nearly 4 times more likely to develop PTH if they did not have a cranioplasty within 30 d, an increase that was not seen in children who underwent cranioplasty within 30 d. These associations differed somewhat when the analysis was limited to children with severe TBI. In these patients, the influence of craniotomy was no longer seen, but the effect of craniectomy without early cranioplasty was more pronounced. Children with early cranioplasty after craniectomy were found to be at increased odds of PTH, although this increase was substantially lower than in those who did not undergo early cranioplasty.

DISCUSSION

Hydrocephalus is an important, potentially reversible cause of poor recovery from TBI that remains incompletely understood. Relatively few studies have directly examined patient characteristics that predispose to PTH, and none of these have focused on the pediatric population. Moreover, findings have been inconsistent, which may be a consequence of the small sample sizes that result from the rarity of the condition. Inconsistent case definitions and practice pattern variations may also contribute, as the existing series all derive from single-institutional experiences. The delayed onset of PTH may also make detection of cases challenging, particularly when using datasets focused on single hospital admissions. We addressed many of these weaknesses by using PHIS, which provides a large convenience sample of administrative data on pediatric healthcare from over 40 freestanding

TABLE 2. Medical and procedural management of patients with and without hydrocephalus

	No hydrocephalus		Hydrocephalus		
	n = 90 737	%	n = 846	%	*P*-value
Hyperosmolar therapy	6464	7.1%	382	45.2%	<.001
ICP monitor					<.001
IPM alone	1262	1.4%	190	22.5%	
EVD alone	223	0.2%	41	4.8%	
IPM and EVD	146	0.2%	35	4.1%	
ICU admission	27 131	30.0%	627	74.1%	<.001
Craniotomy	5435	6.0%	211	24.9%	<.001
Craniectomy	968	1.1%	118	13.9%	<.001
Cranioplasty	681	0.8%	35	4.1%	<.001
Supportive procedures					
Gastrostomy	951	1.0%	146	17.3%	<.001
Tracheostomy	454	0.5%	61	7.2%	<.001

TBI, traumatic brain injury; ICP, intracranial pressure; IPM, intraparenchymal ICP monitor; EVD, external ventricular drain; ICU, intensive care unit.

pediatric hospitals. As a result, we were able to curate a cohort of PTH patients that is an order of magnitude larger than any prior study.[4]

We found that 0.9% of children admitted with TBI required surgical management of hydrocephalus. This finding is in line with published series, which report PTH rates between 0.7% and 3.6% among patients with TBI of any severity.[2,7] Studies based on cohorts limited to patients with severe TBI tend to report substantially higher rates of PTH, with some authors reporting that as many as 40% to 45% of these severely injured patients required CSF diversion.[6,10,16] While PHIS does not contain clinical measures of TBI severity such as Glasgow Coma Scale or pupillary response, we did find that children with markers of more severe TBI were significantly more likely to require surgical intervention for PTH. Additionally, we found that PTH was associated with poor neurological outcomes, as evidenced by the high rate of tracheostomy and gastrostomy placement during the index hospitalization. Unfortunately, our data do not allow us to examine long-term neurological outcomes or response to PTH treatment.

Severity of injury is also likely to account for the strong association between abusive TBI and PTH. Nearly half of all PTH cases were associated with child abuse, and as a group these patients were twice as likely to require management for PTH. Indeed, victims of abusive head trauma have been shown to present with more severe injuries that confer lower rates of survival and higher rates of neurological deficits among those who survive.[17,18] These children are also more likely to have a subdural hematoma and to require neurosurgical intervention,[17-19] both of which were independently associated with the development of PTH in our analysis. Prior work has not identified a relationship between abuse and PTH, but small sample sizes may have limited statistical power in those studies.[18]

The specific type of TBI considerably influenced the odds of PTH. Children with a subdural hematoma had about twice the odds compared to those who did not have a subdural. By contrast, patients with only an isolated skull fracture and no hemorrhage had significantly lower odds of PTH. We did not find an association between PTH and SAH, epidural hematoma, intraparenchymal contusion, or skull fracture with an associated hemorrhage. While aneurysmal SAH is a well-recognized cause of hydrocephalus, the association with traumatic SAH is less clear; some authors have found PTH to be more common in patients with SAH, whereas others have seen no link.[3,8,12,20,21]

Children whose intracranial injuries required neurosurgical intervention in the form of a craniotomy had substantially higher odds of developing PTH in our main model. This finding may be related to TBI severity not adequately captured and accounted for by our severity measures, as children who require a craniotomy are likely to have been more badly injured. That the effect of craniotomy is not seen when severely injured children managed with ICP monitors or hyperosmolar therapy are separately analyzed supports this hypothesis, and suggests that craniotomy is not likely to be pathogenic in PTH.

By contrast, craniectomy without early cranioplasty was associated with increased likelihood of developing PTH, both in the main model and in the subgroup analysis of children whose injuries required ICP monitoring or hyperosmolar therapy. That this effect persisted even among the severely injured suggests that craniectomy may be pathogenic in PTH, and not simply a marker for worse injury. Indeed, widely opening the skull and removing a bone flap significantly reduces intracranial pressure and improves compliance, but also dissipates the pulsatile CSF waveforms that are normally present. Others have posited that the peak pressures of these pulsations are important in driving the reabsorption of CSF, and that diminished pulsatility in the setting of an open skull therefore leads to CSF retention.[15] Consistent with this hypothesis, we found that patients who underwent craniectomy followed by cranioplasty within 30 d of injury were at lower risk of PTH than those without early cranioplasty, perhaps because replacing the bone flap early restores CSF hydrodynamics and reestablishes the driving force for CSF reabsorption before it becomes chronically impaired.[22-24] A putative benefit of early cranioplasty in preventing hydrocephalus was reported in one study of adults who underwent decompressive hemicraniectomy for stroke. Patients who did not develop hydrocephalus had cranioplasty performed a mean of 23 d after decompression, compared to 69 d in those who did develop hydrocephalus.[15] While this finding has not been universally reproduced, studies that did not find an association between cranioplasty timing and hydrocephalus have examined patients who underwent cranioplasty greater than 60 d after decompression, which may be too late to derive benefit. Waziri et al[15] have suggested that delayed cranioplasty

TABLE 3. Multivariable Logistic Regression

	Any TBI			Severe TBI[a]		
	aOR	95% CI	*P*-value	aOR	95% CI	*P*-value
Age in years						
<1	[ref]			[ref]		
1-5	0.44	0.77, 1.04	<.001	0.46	0.35, 0.61	<.001
6-11	0.28	0.77, 1.04	<.001	0.26	0.17, 0.38	<.001
12-21	0.32	0.77, 1.04	<.001	0.28	0.20, 0.41	<.001
Gender						
Male	[ref]			[ref]		
Female	0.89	0.77, 1.04	.14	0.99	0.81, 1.21	.91
Race						
White	[ref]			[ref]		
African American	1.16	0.93, 1.43	.18	1.15	0.87, 1.52	.34
Latino	1.06	0.85, 1.33	.59	0.95	0.70, 1.30	.76
Other/unknown	1.08	0.86, 1.36	.50	1.16	0.85, 1.58	.34
Payer status						
Private	[ref]			[ref]		
Government	1.11	0.93, 1.32	.24	1.05	0.83, 1.32	.70
Child abuse						
No child abuse	[ref]			[ref]		
Any child abuse	2.62	2.16, 3.18	<.001	1.64	1.25, 2.16	<.001
Diagnosis						
Concussion[b]	0.08	0.03, 0.22	<.001	0.00	0.00, Inf	.97
Skull fracture, no ICH[b]	0.08	0.04, 0.16	<.001	0.29	0.11, 0.75	.01
Skull fracture, with ICH[b]	0.92	0.63, 1.35	.67	0.79	0.48, 1.29	.35
SAH[b]	0.86	0.63, 1.16	.32	0.93	0.62, 1.38	.72
Subdural hematoma[b]	1.83	1.27, 2.64	.001	1.13	0.70, 1.82	.63
Epidural hematoma[b]	0.51	0.26, 1.00	.05	0.36	0.14, 0.97	.04
Cerebral contusion[b]	0.95	0.61, 1.47	.81	0.79	0.45, 1.37	.40
Other TBI[b]	0.39	0.23, 0.67	.001	0.63	0.31, 1.27	.20
Inpatient management						
ICU admission[b]	2.10	1.73, 2.54	<.001	1.05	0.76, 1.47	.76
G-tube or tracheostomy[b]	2.57	2.03, 3.25	<.001	2.27	1.78, 2.89	<.001
Hyperosmolar therapy[b]	1.84	1.52, 2.23	<.001	–	–	–
ICP monitor[b]	3.64	2.99, 4.43	<.001	–	–	–
Surgical management of TBI						
Craniotomy[b]	1.60	1.30, 1.97	<.001	1.00	0.78, 1.28	.97
Craniectomy w/o cranioplasty within 30 d[b]	3.67	2.66, 5.07	<.001	4.77	3.44, 6.60	<.001
Craniectomy w/cranioplasty within 30 d[b]	1.19	0.75, 1.89	.45	1.64	1.00, 2.69	.048

[a]Severe TBI defined by use of either ICP monitor or hyperosmolar therapy. [b]Compared to patients without this diagnosis or procedure. TBI, traumatic brain injury; aOR, adjusted odds ratio; CI, confidence interval; ICH, intracranial hemorrhage; ICU, intensive care unit; G-tube, gastrostomy tube; ICP, intracranial pressure; Inf, infinity.

may lead to permanent derangement of CSF hydrodynamics, as has been seen in chronically shunted patients with hydrocephalus.

Finally, our data also showed that age was a significant risk factor for PTH, with children under 1 yr having the highest odds. While young children are certainly at higher risk for abusive head trauma,[25] this variable was considered in the multivariable regression and should not confound the effect of age. It is possible that the compressible, unmyelinated brain and open cranial sutures of young children facilitate dissipation of intracranial pressure waves, as in those who underwent craniectomy. Young children could therefore be susceptible to PTH by a similar mechanism.

Limitations

This study is subject to several limitations that should be considered when interpreting our results. The primary outcome of our study was surgical treatment of hydrocephalus, which was chosen to identify clinically important hydrocephalus. Nonetheless, variation between practitioners and institutions

regarding indications for treatment may be present, and such heterogeneity may impact the associations we observed. In addition, all data were obtained via a query of administrative records, which can result in patient misclassification due to coding errors. This potential bias is of concern with regard to ICD-9-CM codes, which may lack specificity in their classification of some neurosurgical procedures and TBI diagnoses, such as intraventricular hemorrhage. It is also possible that child abuse was miscoded in some cases; however, at least 1 study has shown that the sensitivity and specificity of ICD-9-CM coding for abusive head trauma are greater than 90%.[26] The reliance on administrative data and lack of clinical information precluded the use of standard definitions of TBI severity, and thus our definition of severe TBI differs from other studies. Without detailed clinical data and information about decision making, we are limited in our ability to draw conclusions and make practice recommendations. This limitation is particularly germane to our finding that children who underwent early cranioplasty after craniectomy were less likely to be treated for PTH. While early cranioplasty may be protective against the development of PTH, the association could be subject to confounding by indication; it is possible that patients who develop PTH are less likely to receive an early cranioplasty as a result of their hydrocephalus. Prospective validation of this finding is warranted before definitive practice recommendations can be made.

CONCLUSION

Using a national dataset, we were able to demonstrate that young age, severe TBI, and abusive head trauma all increased the likelihood of PTH. Children who underwent craniectomy without an early cranioplasty were at substantially increased risk, an association that held even when severely injured patients were analyzed separately. This effect was blunted in patients whose craniectomy was followed by a cranioplasty within 30 d, suggesting that early cranioplasty may help to prevent hydrocephalus in this population.

Overall, 0.9% of children admitted with TBI were found to require treatment for PTH. While small as a proportion of all children with TBI, it nonetheless represents a large number in absolute terms. Given the considerable social, psychological, and financial costs of caring for children with hydrocephalus,[27] an improved understanding of the disease is vital from a public health perspective.

Disclosures

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COMMENT

The paper describes information useful in everyday practice of pediatric Neurosurgery. The association between traumatic head injury and hydrocephalus is significant, and this paper confirms the special circumstances that increase frequency of occurrence in pediatric traumatic brain injury such as young age and non-accidental trauma. The authors have made a great effort to find proxies for known important factors such as Glasgow Coma Scale and injury severity. It remains to be seen if there is truly equivalency and the cited conditions such as tracheostomy, gastrostomy, and use of hyperosmolar therapy can truly represent the clinical conditions.

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