Hydrocephalus associated with childhood nonaccidental head trauma

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OBJECTIVE The incidence of posttraumatic ventriculomegaly (PTV) and shunt-dependent hydrocephalus after nonaccidental head trauma (NAHT) is unknown. In the present study, the authors assessed the timing of PTV development, the relationship between PTV and decompressive craniectomy (DC), and whether PTV necessitated placement of a permanent shunt. Also, NAHT/PTV cases were categorized into a temporal profile of delay in admission and evaluated for association with outcomes at discharge.

METHODS The authors retrospectively reviewed the cases of patients diagnosed with NAHT throughout a 10-year period. Cases in which sequential CT scans had been obtained ($n = 28$) were evaluated for Evans' index to determine the earliest time ventricular dilation was observed. Discharge outcomes were assessed using the King's Outcome Scale for Childhood Head Injury score.

RESULTS Thirty-nine percent (11 of 28) of the patients developed PTV. A low admission Glasgow Coma Scale (GCS) score predicted early PTV presentation (within < 3 days) versus a high GCS score (> 1 week). A majority of PTV/NAHT patients presented with a subdural hematoma (both convexity and interhemispheric) and ischemic stroke, but subarachnoid hemorrhage was significantly associated with PTV/NAHT ($p = 0.011$). Of 6 patients undergoing a DC for intractable intracranial pressure, 4 (67%) developed PTV ($p = 0.0366$). These patients tended to present with lower GCS scores and develop ventriculomegaly early. Only 2 patients developed hydrocephalus requiring shunt placement.

CONCLUSIONS PTV presents early after NAHT, particularly after a DC has been performed. However, the authors found that only a few PTV/NAHT patients developed shunt-dependent hydrocephalus.

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KEY WORDS biomarker; child abuse; excitotoxicity; hypoxia-ischemia; shaken baby syndrome; traumatic brain injury

THE natural history of hydrocephalus after nonaccidental head trauma (NAHT) has not been evaluated. This is partly due to the extremely dismal prognosis that NAHT portends and to the fact that it often occurs within 24 hours, correlating with a significant rate of mortality. As a matter of fact, the Center for Disease Control and Prevention in a Spring 2008 report (<http://www.cdc.gov/ViolencePrevention>) listed NAHT as the leading cause of childhood death; the incidence reported in Pennsylvania, for example, was 14.7–26.0 cases per 100,000 person-years.¹¹ This high incidence is not unique to the United States, as a similar rate was observed internationally: 12.8–21 cases per 100,000 person-years in

England,⁸ for instance, and 14.7–19.6 cases per 100,000 person-years in New Zealand.¹⁰ Collectively, these statistics suggest that outcomes after NAHT are universally poor, with only 50% of patients admitted for NAHT living beyond the first 24 hours after injury and with reintegration into society difficult in 50% of surviving children due to severe morbidity.¹² These poor outcomes may be in part due to the effects of hydrocephalus.

The incidence of ventricular dilation after traumatic head injury may be as high as 39%–44%, and on average, this pathology becomes apparent at the 4-week postinjury interval.^{13,15} The incidence of posttraumatic ventriculomegaly (PTV) is unclear, as is that of subsequent development

ABBREVIATIONS CPCT = child protection consultation team; GCS = Glasgow Coma Scale; IVH = intraventricular hemorrhage; KOSCHI = King's Outcome Scale for Childhood Head Injury; NAHT = nonaccidental head trauma; PTV = posttraumatic ventriculomegaly; SAH = subarachnoid hemorrhage; SDH = subdural hematoma; TBI = traumatic brain injury.

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to shunt-dependent hydrocephalus after NAHT. Marmarou et al.¹³ classically used a dye injection method to demonstrate altered CSF dynamics due to poor absorptive properties associated with subarachnoid hemorrhage (SAH) or intraventricular hemorrhage (IVH) in a majority of patients. Alternatively, operative procedures such as decompressive craniectomies have also been implicated in playing a role in promoting the development of hydrocephalus^{2,22} and subsequently could result in poorer outcomes.^{2,14,19}

Here, we evaluated our cohort of patients with perpetrator-identified NAHT to address the involvement of hydrocephalus. We quantified the cumulative incidences of PTV and shunt-dependent hydrocephalus. We analyzed the data for epidemiological factors and radiographic signs associated with these primary findings. In addition, we questioned whether secondary factors such as a delay in seeking medical care and/or a decompressive craniectomy promoted hydrocephalus.

Methods

With approval from the institutional review board, we performed a retrospective, single-center, chart review of all patients who were evaluated by the medical child protection consultation team (CPCT) and who were reported to the New York State Central Register, documented between the 2001 and 2010.²⁰ The total number of NAHT patients registered was 48. Inclusion criteria included cases in which the perpetrator was specifically identified by the Child Protection Services of Nassau, Suffolk, Brooklyn, and Queens counties and in which the perpetrator was considered the primary suspect in a criminal trial or was convicted by the local/state law enforcement agencies.^{4,20} Fourteen cases were excluded from this review because perpetrator status could not be identified, and 6 of the 34 cases in which a perpetrator was identified were excluded because the charts could not be reviewed at the time of this study as they were under review in preparation for a criminal trial or were being used during a criminal trial. The remaining 28 cases were evaluated by our hospital CPCT and pediatric neurosurgical department during the study period and included a multidisciplinary evaluation during admission.

The diagnosis of NAHT was indicated after clear evidence of intracranial injury was demonstrated on radiographs and after careful evaluation by CPCT.^{4,20}

All admission CT scans and subsequent CT and MRI scans were collected and reviewed by both a neuroradiologist and neurosurgeon. PTV was defined using the Evans' index (≥ 0.30). Hydrocephalus requiring shunt placement was determined by the Evans' ratio linear index for ventriculomegaly and persistent or worsening symptoms.^{15,16} We determined the temporal profile of each case by identifying the time of injury/ictus as verified by the child protection and criminal investigation teams, regardless of whether the stated mechanism was accurate. We then identified the time of admission to the pediatric emergency room. This elapsed time was then categorized into the following temporal classification: 1) no delay, 0–6 hours; 2) moderate delay, 6–12 hours; and 3) severe delay, > 12 hours.²⁰

Outcomes assessments took into account the following

variables: serial imaging, in-hospital morbidity, and neurosurgical interventions. Discharge outcomes were graded using the King's Outcome Scale for Childhood Head Injury (KOSCHI) scores (1 = death, 2 = vegetative state, 3 = severe disability, 4 = moderate disability, and 5 = good recovery).^{1,20}

Statistical assessment involved the Biostatistics Unit of The Feinstein Institute for Medical Research and included univariate analysis of the patient age, sex, Glasgow Coma Scale (GCS; low score of 3–8; high score of 9–15), SAH, IVH, ischemic stroke, subdural hematoma (SDH), PTV, shunt-dependent hydrocephalus, decompressive craniectomy, admission delay, and KOSCHI score.²⁰ For statistical purposes, scoring was represented in nonparametric ranks to preserve the order of the scoring. A Kruskal-Wallis test or Mann-Whitney U-test was performed for all univariate analyses, and a scatter plot was generated to represent statistical findings. Associations between categorical variables (admission delay, SDH, infarct, SAH, IVH, and midline shift) and PTV during hospitalization were examined using Fisher's exact test. Associations between continuous variables (age, GCS score at admission) and PTV during hospitalization were examined using exact logistic regression. All data were evaluated using SAS version 9.2 statistical software (SAS Institute, Inc.). A *p* value < 0.05 was considered significant.

Results

Incidence of Ventriculomegaly and Time of Presentation

Of the 28 children diagnosed with NAHT, we identified 11 (39%) with ventricular dilation according to the Evans' index (Table 1). Of NAHT patients presenting with low GCS scores (*n* = 16), 7 developed PTV (44%), whereas of 12 NAHT patients presenting with high GCS scores, 4 developed PTV (33%) (Table 1). Despite the fact that more NAHT patients presented with low GCS scores, GCS score on arrival did not prove predictive of subsequent hydrocephalus development. In our assessment of the earliest time of PTV presentation, PTV occurred as early as within 1 day of trauma but more commonly within 10 days of trauma (82%; 9 of 11; Fig. 1 upper).

Radiological Features Associated With PTV

The most common radiological finding observed in our patients was SDHs; additionally, we observed a high incidence of retinal hemorrhages, a low presenting GCS score, higher male to female sex ratio, and age of less than 2 years, as previously described in our previous report.²⁰ We investigated the association of additional radiological findings specifically to those NAHT patients who developed PTV (*n* = 11; Table 1). Eight patients (73%) had hemispheric SDHs and 6 patients (55%) had interhemispheric or tentorial SDHs. Additionally, in 7 patients (64%), the CT scan displayed hypodensities suggestive of ischemic stroke, another predominant sign. Despite these trends, only SAH, seen in 3 patients (27%), proved significant (*p* = 0.011). Historically, SAH and IVH are thought to contribute to ventricular dilation, but in only 1 case of PTV (1%) was IVH observed, and this was not statistically significant (Table 1).

TABLE 1. Summary of PTV data and their associated radiological findings

Characteristic	No. of Patients	p Value
PTV incidence in NAHT		
Overall PTV incidence	11/28 (39%)	
PTV incidence w/o craniectomy	7/22 (32%)	
PTV incidence w/ craniectomy	4/6 (67%)	0.0366
PTV & GCS		
Mild/moderate NAHT (GCS Score 9–15)	4/12 (33%)	
Severe NAHT (GCS Score 3–8)	7/16 (44%)	
Permanent VPS placed*	2/28 (7%)	
VPS placed after craniectomy†	1/6 (17%)	
Admission CT findings‡		
Convexity SDH (unilat and bilat)	8	
Interhemispheric/tentorial SDH	6	
SAH	3	0.011
IVH	1	
Ischemic stroke	7	

VPS = ventriculoperitoneal shunt.

* Both cases presented with severe NAHT (GCS score of 3–8) and without delayed hospital admission.

† Limited temporal craniectomy with large superior frontoparietal craniotomy.

‡ Total number of PTV/NAHT cases was 11.

Temporal Profile of Delay in Admission and PTV

We recently reported on the concern about delays in obtaining acute medical care after NAHT, questioning the impact that delayed treatment may have on in-hospital discharge outcomes, including data on inpatient survival analysis. Patients who had a moderate delay in care—in other words, those who arrived to the hospital between 6 and 12 hours after onset of the first reported symptom (injury ictus)^{18,20,21}—had demonstrated worst outcomes compared with patients who presented without delay (0–6 hours postinjury ictus) or after a significant delay (≥ 12 hours postinjury ictus).²⁰ We questioned if time between injury and seeking medical care had any role in observations of PTV. Seven (64%) of 11 patients with severe head injury (a low GCS score) had PTV, and this imaging finding was demonstrated within 3 days after injury. A majority of these patients presented without delay in arrival (0–6 hours) (Table 2). However, the other 4 patients (36%), those with high a GCS score, had PTV, but it was demonstrated at least a week after injury. When we performed a subgroup analysis based on delay in arrival, we found that cases in which patients had a no or moderate delay in seeking medical care were associated with PTV development before 1 week postinjury, and in those cases in which patients arrived after severe delay (> 12 hours) PTV developed more than 2 weeks after injury (Fig. 1 lower). Low GCS score and no or moderate time delay in arrival for medical care were associated with earlier demonstration of PTV.

Ventriculomegaly and Discharge Outcome

We considered whether development of PTV has any impact on hospital discharge outcomes by comparing pa-

FIG. 1. Bar graphs showing the development of PTV after NAHT presentation in patients with a low or high GCS score (upper) and with or without delay in seeking acute medical care after injury (lower).

tients with and without PTV. Considering patients with early mortality who may not have undergone serial scanning, we were unable to perform any meaningful statistical comparison. However, when we examined those NAHT patients demonstrating PTV early (< 1 week) versus late (> 1 week), poorer outcomes at inpatient discharge were observed in patients with early PTV than in those presenting with late PTV (KOSCHI score < 3 ; Fig. 2).

PTV and Decompressive Craniectomy

Decompressive craniectomies may be life-sustaining measures in patients with traumatic brain injury (TBI). Postoperative sequelae in such patients have been a subject of recent debate, including the development of hydrocephalus. Here, we analyzed NAHT/PTV patients who underwent decompressive craniectomy. No patient had PTV on arrival at our hospital. Large frontotemporoparietal craniectomies (n = 5) or a temporal craniectomy (n = 1), which included dural openings, were performed in a total 6 (21%) of 28 NAHT patients when significant cerebral edema or mass effect was present due to either intracranial findings on CT scans or intractable intracranial pressure. PTV was observed to occur in 4 of these patients (67%). NAHT patients who did not undergo a decompressive craniectomy

TABLE 2. Hospital admission GCS score and temporal profile of delay in arrival scores in NAHT/PTV patients

GCS Score	Delay in Seeking Care		
	None	Moderate	Severe
3–8	5	2	0
9–15	1	1	2

were also observed to develop PTV (7 of 22 patients; 32%) (Table 1). We asked whether those NAHT patients who underwent a decompressive craniectomy were more likely to be associated with PTV. We found decompressive craniectomy to be statistically significantly associated with NAHT/PTV development ($p = 0.0366$; Table 1).

Shunt Dependence

Only 2 cases (7%) of NAHT/PTV required permanent CSF shunting (Table 1). One of the patients had undergone a full frontotemporoparietal craniectomy. With only 2 cases, there was no statistical analysis that could address the role of decompressive craniectomy on shunt-treated hydrocephalus. Overall, shunted hydrocephalus was observed to occur infrequently in our cohort.

Discussion

To the best of our knowledge, this is the first study to examine the natural history of hydrocephalus after NAHT in children. Hydrocephalus may represent an important factor in discharge outcomes. Dias et al. documented that imaging findings such as intraparenchymal hypodensities on CT scans may be present as early as 3 hours after injury in contrast to historical documentation of presentation 6–24 hours after injury. Furthermore, chronic SDHs may present as early as 20 hours, much earlier than the 1–4 weeks traditionally viewed.³ For these reasons, we questioned whether the incidence of PTV and hydrocephalus in NAHT patients was similar to the incidence of PTV and hydrocephalus in other etiologies of TBI. To address these concerns, we first evaluated PTV, a radiological sign that can be demonstrated, with or without clinical correlation. We found the incidence of PTV in our series to be 38%, on par with more general studies of TBI (adult TBI/PTV approximately 44%).¹³ One important limitation of our study observations was nonprotocolled neuroimaging surveillance. We did not acquire routine CT scans and instead obtained imaging studies based on clinical parameters including neurological examination. In previous work, we questioned the post-NAHT time to seeking medical care as an important variable,²⁰ and this, in addition to findings reported here, may prompt future investigation into the early need for serial imaging. The second limitation was that we used the Evans' index as a linear index common to several neuroradiology divisions across a large number of children's hospitals. Several studies including a recent report directly assessing the various methods have suggested the frontooccipital horn ratio may better serve as an estimate of ventricular volume in those for whom there is concern for hydrocephalus.¹⁶

FIG. 2. Bar graph showing KOSCHI scores at the time of inpatient discharge in NAHT patients stratified by early or late PTV.

Pediatric patients with TBI can develop delayed hydrocephalus: 20% of patients with severe injuries,¹³ 29.5% after a decompressive craniectomy (accidental trauma),⁷ and 40% of patients with pediatric TBI (accidental and non-accidental trauma).⁹ It's unclear from these studies what the natural history of developing hydrocephalus is in the NAHT population. Past studies in NAHT have evaluated mostly short-term outcomes, partly due to the high rate of infant mortality within the first 24 hours.⁵

We evaluated hydrocephalus association by stratifying time of arrival to medical attention, development of PTV, and time to shunt-dependent hydrocephalus. By the time of inpatient discharge, only 2 patients required a permanent shunt (2 of 28 patients [7%] and 2 of 24 surviving patients [8%]). This is not surprising when comparing this finding to observations in the reported literature that intraparenchymal hypodensities allow for more brain compliance and thus greater ventricular enlargement and blunting of cortical subarachnoid spaces as seen in our few cases involving SAH. This observation was not statistically significant, and several possible explanations may be the following: 1) poor clinical status at admission may not have allowed enough time to evaluate for development of delayed hydrocephalus, 2) a limited population was evaluated in this cohort of perpetrator-identified cases only, and 3) using dye-injection CT cisternography made direct assessment impossible.¹³

There has been concern that decompressive craniectomies themselves may contribute to the development of hydrocephalus. When we assessed the role of this procedure in our population, we found a statistically significant association between decompressive craniectomy and PTV. Mathematical modeling for hydrocephalus, as described in the article by Rekaté et al.,¹⁷ may explain why removal of part of the cranium can cause significant change in CSF dynamics. Hochwald et al.⁶ demonstrated blunted CSF absorption after craniectomies and investigated impaired periventricular venous circulation. In the present study, we could not address the effect of craniectomy size directly. It is possible that large craniectomies with a superior border within 2.5 cm of the sagittal sinus can lead to a greater incidence of PTV.² It's unclear whether this parameter is age dependent. Importantly, selection bias in offering a decompressive craniectomy to individuals with neurological compromise likely may be brought about by signifi-

cant intracranial findings such as hypodensities related to stroke or intracranial hemorrhage, thus having a greater propensity toward developing PTV.

Conclusions

This is the first report to identify the cumulative indices of ventriculomegaly and shunt-dependent hydrocephalus after NAHT. We observed the rate of PTV in our cohort to be similar to that after accidental TBI, and this may, in part, be related to decompressive craniectomies. Furthermore, we observed a low rate of subsequent shunt-dependent hydrocephalus. Potentially a larger collaborative prospective accrual of patients undergoing standardized hydrocephalus assessment and treatment with shunt placement protocols may confirm the observations reported here.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Vadivelu. Acquisition of data: Vadivelu, Esernio-Jenssen. Analysis and interpretation of data: Vadivelu, Rekate. Drafting the article: Vadivelu. Critically revising the article: Vadivelu, Rekate. Reviewed submitted version of manuscript: Vadivelu, Rekate, Esernio-Jenssen. Approved the final version of the manuscript on behalf of all authors: Vadivelu. Statistical analysis: Vadivelu. Administrative/technical/material support: Mittler, Schneider. Study supervision: all authors.

Supplemental Information

Previous Presentations

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