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Retinal hemorrhage and brain injury patterns on diffusion-weighted magnetic resonance imaging in children with head trauma

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Abstract

Purpose—To evaluate associations between retinal hemorrhage severity and hypoxic-ischemic brain injury (HII) patterns by diffusion-weighted magnetic resonance imaging (DW-MRI) in young children with head trauma.

Methods—DW-MRI images of a consecutive cohort study of children under age 3 years with inflicted or accidental head trauma who had eye examinations were analyzed by two independent masked examiners for type, severity, and location of primary lesions attributable to trauma, HII secondary to trauma, and mixed injury patterns. Retinal hemorrhage was graded retrospectively on a scale from 1 (none) to 5 (severe).

Results—Retinal hemorrhage score was 3–5 in 6 of 7 patients with predominantly post-traumatic HII pattern and 4 of 32 who had traumatic injury without HII ($P < 0.001$) on DW-MRI imaging. Severe retinal hemorrhage was observed in absence of HII but only in inflicted injury. Retinal hemorrhage severity was correlated with HII severity ($\rho = 0.53$, $P < 0.001$) but not traumatic injury severity ($\rho = -0.10$, $P = 0.50$). HII severity was associated with retinal hemorrhage score 3–5 ($P = 0.01$), but traumatic injury severity was not ($P = 0.37$).

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Conclusions—During inflicted head injury, a distinct type of trauma occurs causing more global brain injury with HII and more severe retinal hemorrhages. HII is not a necessary factor for severe retinal hemorrhage to develop from inflicted trauma.

Understanding of pediatric abusive head trauma has evolved over the last five decades. In 1962 Kempe and colleagues[1] published their landmark paper "The Battered Child Syndrome," the first real recognition of child abuse as a disease and of the responsibility physicians held for its diagnosis and prevention. The paper identified intracranial hemorrhage in young children as a hallmark sign in many cases.[1] Nine years later, in 1971, Guthkelch[2] was the first to suggest shaking as a form of abusive injury. He reported on 23 children (22 <18 months of age) presenting with various combinations of subdural hemorrhage, fractures, parenchymal brain injury, and retinal hemorrhages.[2] Shortly thereafter, Caffey[3–5] coined the term *whiplash-shaken infant syndrome*. Both Guthkelch and Caffey noted a frequent absence of external signs of trauma and suggested the role of torn bridging vessels in the brain as the cause of the intracranial hemorrhage. Using autopsy evidence and a dummy model, Duhaime and colleagues[6] in 1987 suggested that blunt impact trauma may be a prerequisite to generate sufficient deceleration forces for the characteristic injuries to occur; however, consistency across perpetrator confessions suggests that shaking alone is sufficient to cause such injuries,[7] and actual injury threshold levels for infant brains have yet to be established. There are currently multiple hypothesized factors in the pathogenesis of brain pathology and retinal hemorrhage in abusive head trauma, including deceleration and sheering injury, hypoxic-ischemic injury (from decreased perfusion or apnea), blunt impact, neck flexion-extension, and raised intracranial or venous pressures.[8] However, the relative importance of these factors cannot be determined precisely based on the published data.

The availability of diffusion-weighted magnetic resonance imaging (DW-MRI) and noninvasive vascular imaging techniques now make it possible to evaluate the role of hypoxic-ischemic injury (HII) in traumatic pediatric head injuries.[9] DW-MRI enables identification of acute cellular injury and cytotoxic edema, which in the context of head trauma may result from hypoxic-ischemic injury, direct traumatic injury, or both. Brain tissue damage causes shifting of water molecules from extracellular to intracellular compartments, which can be identified as reduced diffusion of water on DW-MRI, in comparison to undamaged areas. Such changes can be identified early, hours or days before changes in the appearance of tissue on T2-weighted sequences.[9] DW-MRI (versus T1 or T2) is particularly helpful in infants, whose brains have a high water content and immature myelination. Biousse and colleagues[10] reported a high incidence of possible HII in a cohort of infants with presumed abusive head trauma. In 2007 Ichord and colleagues[11] demonstrated a relationship between HII and inflicted trauma using DW-MRI in a cohort of children with both accidental and inflicted head injuries.

It has been clearly established that both the presence and increasing severity of retinal hemorrhages are highly associated with abusive versus accidental injury in children presenting with traumatic intracranial hemorrhage[12]; however, the mechanisms underlying retinal hemorrhages are still not clearly established, and there is limited information in the literature addressing HII as it relates to ocular findings in the setting of pediatric head trauma. The purpose of this study was to evaluate the associations between retinal hemorrhage severity and HII patterns as identified on DW-MRI in young children with accidental and inflicted head trauma.

Subjects and Methods

The study was approved by the Children's Hospital of Philadelphia Institutional Review Board and conformed to the requirements of the US Health Insurance Portability and Accountability Act of 1996.

Patients were enrolled in a consecutive cohort study at the Children's Hospital of Philadelphia (CHOP). Children <3 years of age admitted from July 2001 to December 2004 with inflicted or accidental head trauma and who had a brain DW-MRI within 1 week of injury (average, 2 days) were included. Children with coagulopathy, penetrating head injury or burns, cervical cord injury, or cardiopulmonary resuscitation for >5 minutes were excluded in order to avoid any potentially confounding effects. Subjects were identified from prospectively maintained CHOP trauma and abuse registries. The primary analysis of brain injury patterns on DW-MRI has been previously published.[11] The primary report did not include ocular findings, and only the subset of children with eye examinations were included for this study. While the imaging analyses described below were performed explicitly for the study, eye findings were abstracted from the medical record retrospectively, as detailed below.

We rigorously applied a published and validated algorithm that excludes eye findings and HII to categorize a child as having had an inflicted injury.[13] Accidental injury was defined as well-documented, witnessed accidental trauma. Children with injuries from indeterminate mechanisms of trauma were excluded.

Magnetic resonance images were obtained on a 1.5-Tesla system. Images included sagittal T1, axial T2, fluid attenuated inversion recovery (FLAIR), gradient echo, and diffusion sequences with apparent diffusion coefficient (ADC) maps. MRI images were centrally analyzed and scored for type, severity, and location of (1) lesions due to HII secondary to trauma and (2) lesions due to the primary traumatic injury by one or both of two independent masked readers (a pediatric neuroradiologist and a pediatric neurologist) using a semiquantitative scoring system, which has been described in detail elsewhere[11] and may be summarized as follows. Intracranial trauma severity was scored separately from HII severity. Traumatic injury lesions included cortical contusions, white matter shear injury, and intracranial hemorrhage (subdural epidural, subarachnoid, intraventricular, and intraparenchymal hemorrhage). Criteria defining HII lesions included (a) "increased T2 signal with concomitant diffusion restriction in a vascular" (definite HII) or not-typical vascular (suspected HII) distribution or (b) "increased T1 signal indicative of cortical laminar necrosis in a vascular distribution."[11] Vascular distributions included "watershed territories, large vessel arterial occlusive infarcts, symmetric deep grey nuclei injury (basal ganglia, thalamus), or diffuse cerebral edema."[11] A comprehensive list of vascular distributions has been published.[11] Severity was scored on a 1 to 4 scale ("1 for small unilateral lesions, 2 for large unilateral lesions, 3 for small bilateral lesions, and 4 for large bilateral lesions"[11]) for each type of lesion and summed separately for HII and traumatic lesions. Inter-rater reliability of this image analysis method was previously reported to be high.[11] Among 14 cases scored by both readers, the intra-class correlation coefficient was 0.97 (95% CI, 0.91–0.99) for HII severity score and 0.92 (95% CI, 0.77–0.97) for traumatic injury severity score.

Five patterns of brain injury were previously identified on MRI[11] and described as follows: (1) predominantly post-traumatic HII, in which HII is the predominant finding, with associated traumatic lesions not anatomically contiguous or colocalizing with HII, although all had traumatic lesions (Figure 1); (2) mixed, in which focal or multifocal HII and traumatic lesions are anatomically contiguous or colocalizing; (3) predominantly traumatic

injury lesions, in which traumatic injury lesions are the major finding without HII lesions (Figure 2); (4) indeterminate, in which focal lesions do not conform to vascular patterns and lack features suggestive of trauma, such as hemorrhage or edema; and (5) none.[11]

All patients had dilated fundus examinations by a pediatric ophthalmologist using indirect ophthalmoscopy within 48 hours of presentation. The clinical notes of these examinations for each subject were retrospectively scored by two independent, masked pediatric ophthalmologists and averaged. The scoring system used was as follows: no retinal hemorrhages, 1; few, intraretinal, posterior pole hemorrhages, 2; numerous intraretinal, posterior pole hemorrhages, 3; numerous, intra- and preretinal, diffuse hemorrhages, 4; and extensive retinal hemorrhages in multiple layers and/or the presence of retinoschisis or retinal fold, 5. If a child had asymmetric eye findings, the score for the eye with more extensive retinal hemorrhage was used as the score for a given subject.

All statistical analyses were performed using SAS statistical software version 9.2 (SAS INC, Cary, NC). Comparison of means was done using two-sample *t* test. Comparison of medians was done using Mann-Whitney-Wilcoxon test. Comparisons of proportions for categorical variables were performed using the Fisher exact test. Multivariate logistic regression models were used to determine the associations between moderate to severe retinal hemorrhage (a score of 3 to 5) and the severity of HII or the severity of traumatic injury lesions.

Results

A total of 45 children met inclusion criteria for this study (Table 1). Of these, 16 children had accidental head injury; 29, inflicted head injury. The median age was 4.5 months (range, 0.5–35 months) for accidental cases and 2.3 months (range, 0.4–16 months) for inflicted injury cases ($P = 0.28$).

Traumatic injury lesions on DW-MRI were present in 14 of 16 accidental injuries and all 29 inflicted injuries ($P = 0.12$). HII lesions on DW-MRI were found in none of the children with accidental trauma and 10 children (35%) with abusive head trauma ($P = 0.008$), for whom the brain injury pattern was predominantly post-traumatic HII in 7 and mixed in 3. All 7 children with a predominantly post-traumatic HII pattern of abnormalities also had traumatic injury lesions that were of lesser morphologic extent when compared to the HII lesions, mostly extra-axial hemorrhages. These cases were classified as predominantly post-traumatic HII because the predominant finding was hypoxic-ischemic in nature. Of the 45 children, 42 (93%) had extracranial injuries identifiable on physical examination or radiographic study (Table 2).

Retinal hemorrhage was found in 2 of 16 accidental cases and 11 of 29 inflicted cases (12.5% vs 37.9%; $P = 0.09$). Moderate to severe retinal hemorrhages (scores of 3–5) were present in 6 of 7 children who had a predominantly post-traumatic HII pattern and 4 of 32 who had predominantly traumatic injury lesions without HII (86% vs 12.5%, $P < 0.001$). Retinal hemorrhage severity was correlated with HII severity (Spearman correlation coefficient, $\rho = 0.53$; $P < 0.001$) but not with traumatic injury severity ($\rho = -0.10$, $P = 0.52$). In multivariate regression analysis, HII severity was associated with moderate to severe (score 3–5) retinal hemorrhage ($P = 0.01$, OR = 1.32 [95% CI 1.06–1.64]) but traumatic injury severity was not ($P = 0.37$, OR = 0.85 [95% CI, 0.60–1.21]). All children with HII also had traumatic injury.

Discussion

This study found an association between severity of retinal hemorrhage and HII brain injury patterns as identified on DW-MRI, in young children with head trauma. These findings might be explained by the severity of trauma and/or by the involvement of more than one mechanism underlying both retinal hemorrhage and HII in these children with inflicted or accidental injuries. Retinal hemorrhage severity was not correlated with traumatic intracranial injury severity even though traumatic injury lesions were present in all cases with a predominantly post-traumatic HII pattern. In addition, HII was only seen in inflicted trauma. These findings suggest that a distinct type of traumatic injury may occur during inflicted head trauma that causes both more global brain injury with HII and more severe retinal hemmorrhage but not necessarily more severe traumatic intracranial injury lesions.

Severity of “traumatic intracranial injury,” as defined in this study, was measured by an imaging scoring system in which hemorrhagic lesions, both intraparenchymal and extra-axial, contributed the most weight to the score. More diffuse cellular injury of a nonhemorrhagic nature produced by trauma, such as axonal shearing, may not be fully visualized by conventional clinical MRI sequences and thus may not be fully captured with this scoring strategy. While epidural hematoma and hemorrhagic stroke are examples of conditions in which the amount of blood correlates with the severity of disease, some brain injuries might only be markers for the type of injury rather than the severity of the injury. For example, it is quite common even in fatal cases of abusive head trauma to see only small amounts of intracranial hemorrhage.[14] In contrast, MRI imaging is exquisitely sensitive in depicting extent and anatomic distribution of hypoxic ischemic parenchymal injury. Thus the HII score may be a more sensitive and precise marker of severity of HII parenchymal damage than is the trauma severity score a marker of severity of traumatic parenchymal injury.[11] A difference in sensitivity and precision of HII scores compared to traumatic injury scores in fully representing severity of these different types of injury could also explain why we found that severity of retinal hemorrhage correlated with severity of HII but not with severity of “traumatic” injury (ie, hemorrhage).

Numerous mechanisms contributing to HII have been described, many of which are interrelated but most not clearly established to be independently causative in the setting of infant head trauma. HII in the setting of trauma may relate to poor systemic perfusion, decreased oxygenation, raised intracranial pressure, intravascular microthrombosis, reactive vasospasm adjacent to intracranial hemorrhage lesions, cervicomedullary injuries, strangulation, disrupted autoregulation, or apnea.[10,11,15,16] However, none of these factors in isolation has been shown to cause severe retinal hemorrhage. Furthermore, in our study, predominantly HII injury patterns were seen only in inflicted head injury, and moderate to severe retinal hemorrhages were also only present in cases of inflicted trauma. These findings, taken together with the well-described strong association between severe retinal hemorrhage and abusive versus accidental head injury,[12] raise the possibility that the mechanisms responsible for HII may play a role in the pathogenesis of retinal hemorrhage in the setting of trauma. It is important to recognize that we did not evaluate nontraumatic causes of HII alone and thus cannot use our study data to evaluate if hypoxia-ischemia contributes directly to the pathogenesis of retinal hemorrhage, as opposed to traumatic forces causing both retinal hemorrhage and HII. Nearly all children had extracranial findings consistent with a traumatic injury, such as bony fractures, soft tissue swelling, bruises, and other signs, and were unlikely to have an isolated, nontraumatic cause of HII. We can observe, however, that HII is not a necessary factor for severe retinal hemorrhage, as severe retinal hemorrhages were noted in the absence of HII in children with inflicted trauma in the study. Furthermore, in a recent study of retinal findings in critically ill children, respiratory

failure was associated with a lower likelihood of having retinal hemorrhage, and nontraumatic encephalopathy had no association with retinal hemorrhage.[17]

Strengths of this study included well-defined inclusion and exclusion criteria, strict basis for categorization of inflicted versus accidental trauma that excluded eye findings, masked central review and scoring of MRI images by a pediatric neuroradiologist and a pediatric neurologist, masked independent grading of retinal hemorrhage findings by two pediatric ophthalmologists, and well-defined brain injury pattern and scoring criteria. There were important limitations as well. Inclusion in the study depended on an MRI having been performed, and the decisions to obtain an MRI and eye examination were based on clinical judgment and not on predefined study criteria. In addition, the injuries scored were determined through the use of a single time point, even though acute brain injury may evolve over time. The heterogeneity represented among children with head injuries, whether inflicted or accidental, may affect the generalizability of the study findings. Finally, eye findings were evaluated and scored using a retrospective review of medical records.

In this study using masked, structured evaluations of diffusion-weighted MRI films, increased retinal hemorrhage severity was associated with radiologically confirmed brain HII in children with head trauma. Severe retinal hemorrhage was observed in the absence of HII but only in cases of inflicted injury, and retinal hemorrhage severity did not correlate with radiologically defined traumatic intracranial injury severity. These findings suggest that during inflicted head injury a distinct type of trauma occurs that causes both more brain HII and more severe retinal hemorrhage but not necessarily more severe traumatic intracranial injuries. We did not investigate the underlying pathophysiologic or biomechanical mechanisms involved, and we can not draw a conclusion regarding a causative role between HII and retinal hemorrhage, because the children did not have isolated HII without trauma. However, we observed that HII is not necessary for the development of severe retinal hemorrhage, as some children had severe retinal hemorrhages without HII. Future investigation is required to assess the association between retinal hemorrhage and nontraumatic causes of HII to address this question directly.

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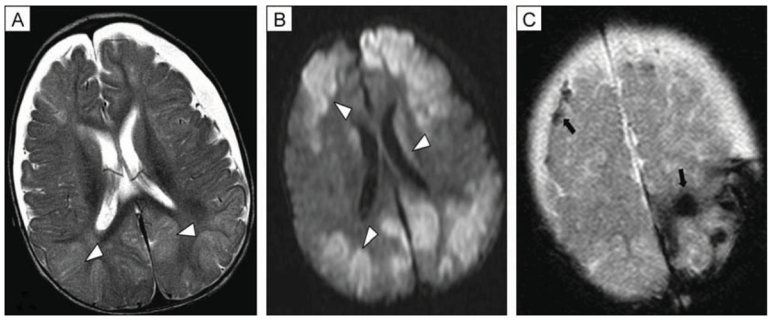

FIG 1.

Predominantly post-traumatic hypoxic-ischemic injury pattern in a 4 month old infant with abusive head trauma. A, MRI T2-weighted image demonstrating multiple foci of subtle hyperintensity and loss of gray-white differentiation in watershed zones and the putamen (arrow heads), as well as chronic subdural hygromas. B, Diffusion-weighted image showing corresponding multiple confluent areas of restricted diffusion (arrow heads). C, Susceptibility-weighted sequence showing acute extra-axial hemorrhage over the convexity posteriorly and frontally (arrows). Reprinted with permission from J Neurotrauma 2007;24:106–18.

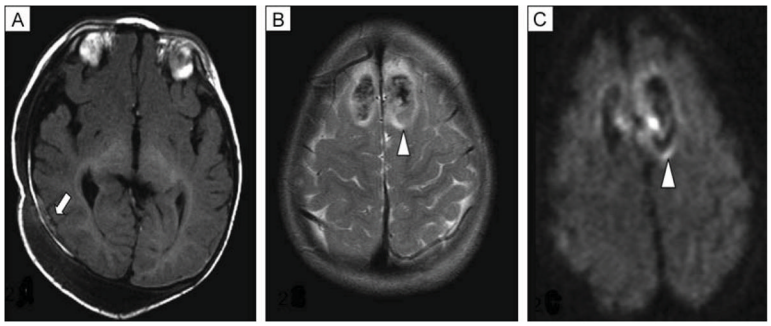

FIG 2.

Examples of predominantly traumatic brain injury patterns. A, T1-weighted sequence shows small subdural hemorrhage over the right posterior temporal region (arrow) in a 6-month-old infant with accidental trauma. B, T2-weighted image in a 16-month-old child with inflicted trauma shows two frontal contusions with intraparenchymal hemorrhage (arrowhead), and diffusion weighted image. C, Surrounding rims of restricted diffusion. Reprinted with permission from J Neurotrauma 2007;24:106–18.

Table 1

Comparison of accidental head injury and inflicted injury

	Accidental head injury (n = 16)	Inflicted injury (n = 29)	*P* value
Age, months			
Median (min, max)	4.5 (0.5, 35)	2.3 (0.4, 16)	0.28[a]
Mean (SD)	6.2 (8.3)	3.2 (3.0)	0.19[b]
Time to MRI, days			0.42[c]
0	2 (12.5)	2 (6.9)	
1	6 (37.5)	12 (41.4)	
2	7 (43.8)	7 (24.1)	
3	0 (0.0)	5 (17.2)	
4	1 (6.3)	2 (6.9)	
8	0 (0.0)	1 (3.5)	
Retinal hemorrhage, N (%)			
None present	14 (88)	18 (62)	0.09[c]
Present	2 (12)	11 (38)	
Retinal hemorrhage score, N (%)			
0–2 (none to mild)	16 (100)	18 (62)	0.004[c]
3–5 (moderate to severe)	0 (0)	11 (38)	
Traumatic intracranial injury lesions on MRI, N (%)			
No	2 (12)	0 (0.00)	0.12[c]
Yes	14 (88)	29 (100)	
Traumatic intracranial injury severity score			
Median (min, max)	3 (0, 11)	4 (1, 13)	0.002[a]
Mean (SD)	3 (3)	5 (3)	0.01[b]
Post-traumatic HII lesions on MRI, N (%)			
No	16 (100)	19 (65)	0.008[c]
Yes	0 (0)	10 (35)	
HII severity score,			
Median (min, max)	0 (0, 0)	0 (0, 23)	0.009[a]
Mean (SD)	0 (0)	3 (6)	0.008[b]
Brain Injury Pattern, N (%)			0.01[c]
Predominantly post-traumatic HII	0 (0)	7 (24)	
Mixed	0 (0)	3 (10)	
Predominantly traumatic injury lesions	14 (88)	18 (62)	
Indeterminate	0 (0)	1 (4)	
None	2 (12)	0 (0.00)	

HII, hypoxic ischemic injury; *MRI*, magnetic resonance imaging.

[a] Mann-Whitney-Wilcoxon test.

[b] Two sample *t* test of means.

[c] Fisher exact test.

Table 2

Extracranial injuries among 45 infants with abusive or accidental head trauma

	Abuse	Accident	Total
	(n = 29)	(n = 16)	(n = 45)
	n (%)	n (%)	n (%)
Any extracranial injury	28 (96.6)	14 (87.5)	42 (93.3)
External sign of injury on physical exam (swelling, bruise, abrasion, or laceration)	23 (79.3)	13 (81.3)	36 (80.0)
Mouth (including frenulum tear)	4 (13.8)	0 (0.0)	4 (8.9)
Head	15 (51.7)	12 (75.0)	37 (82.2)
Face	16 (55.2)	4 (25.0)	20 (44.4)
Neck	1 (3.5)	0 (0.0)	1 (2.2)
Chest	5 (17.2)	0 (0.0)	5 (11.1)
Abdomen or back	6 (20.7)	1 (6.3)	7 (15.6)
Genital or buttock	4 (13.8)	0 (0.0)	4 (8.9)
Upper or lower extremity	8 (27.6)	1 (6.3)	9 (20.0)
Radiographic evidence of injury			
Fracture	23 (79.3)	11 (68.8)	34 (75.6)
Skull (includes sutural diastasis)	18 (62.1)	10 (62.5)	28 (62.2)
Orbit	0 (0.0)	1 (6.3)	1 (2.2)
Long bone	11 (37.9)	1 (6.3)	12 (26.7)
Rib	10 (34.5)	0 (0.0)	10 (22.2)
Clavicle	1 (3.4)	0 (0.0)	1 (2.2)
Other	5 (17.2)	0 (0.0)	5 (11.1)
Upper cervical ligamentous injury	3 (10.3)	0 (0.0)	3 (6.7)
Visceral abdominal injury (liver, spleen)	1 (3.4)	0 (0.0)	1 (2.2)
Pulmonary contusion	1 (3.4)	0 (0.0)	1 (2.2)