




Subdural hemorrhage rebleeding in abused children: frequency, associations and clinical presentation

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Abstract

Background Limited documentation exists about how frequently radiologically visible rebleeding occurs with abusive subdural hemorrhages (SDH). Likewise, little is known about rebleeding predispositions and associated symptoms.

Objective To describe the frequency of subdural rebleeding after abusive head trauma (AHT), its predispositions and clinical presentation.

Materials and methods We evaluated children with SDHs from AHT who were reimaged within a year of their initial hospitalization, retrospectively reviewing clinical details and imaging. We used the available CT and MR images. We then performed simple descriptive and comparative statistics.

Results Fifty-four of 85 reimaged children (63.5%) with AHT-SDH rebled. No child had new trauma, radiologic evidence of new parenchymal injury or acute neurologic symptoms from rebleeding. From the initial presentation, macrocephaly was associated with subsequent rebleeding. Greater subdural depth, macrocephaly, ventriculomegaly and brain atrophy at follow-up were associated with rebleeding. No other radiologic findings at initial presentation or follow-up predicted rebleeding risk, although pre-existing brain atrophy at initial admission and initial chronic SDHs barely missed significance. Impact injuries, retinal hemorrhages and clinical indices of initial injury severity were not associated with rebleeding. All rebleeding occurred within chronic SDHs; no new bridging vein rupture was identified. The mean time until rebleeding was recognized was 12 weeks; no child had rebleeding after 49 weeks.

Conclusion Subdural rebleeding is common and occurs in children who have brain atrophy, ventriculomegaly, macrocephaly and deep SDHs at rebleed. It usually occurs in the early months post-injury. All children with rebleeds were neurologically asymptomatic and lacked histories or clinical or radiologic findings of new trauma. Bleeds did not occur outside of chronic SDHs. We estimate the maximum predicted frequency of non-traumatic SDH rebleeding accompanied by acute neurological symptoms in children with a prior abusive SDH is 3.5%.

Keywords Abusive head trauma · Child abuse · Computed tomography · Infants · Magnetic resonance imaging · Subdural hemorrhage · Subdural rebleeding

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Introduction

It is commonly opined that children who have chronic subdural hemorrhages (SDH) can sustain subsequent rebleeds with no or minor trauma [1]. This proposition is often misused in the legal arena to argue that neurologically symptomatic children, often with accompanying radiologic evidence of acute parenchymal or other injuries, did not sustain abusive head trauma (AHT) but instead sustained a non-traumatic rebleed into a pre-existing chronic SDH. Such unjustified legal assertions led the American Association of Neurological Surgeons to censure one of its members [2] and puts abused children at risk of further harm. A few isolated case reports [1, 3, 4] and a single clinical study [5] documented how often children with prior SDH from AHT sustain subsequent radiologically recognizable rebleeds. Thus we sought to determine the frequency and clinical presentation of radiologically apparent SDH rebleeds. We evaluated the initial and follow-up imaging and clinical information on children who had prior hospitalizations for AHT with associated SDH. We sought to determine what percentage of children sustained SDH rebleeding, what was their clinical behavior when they rebled, and which clinical or imaging characteristics from their initial hospitalization and the time of rebleeding predicted the children who were most likely to rebleed.

Chronic SDHs are known to contain a fibrinolytic environment with friable new capillary beds [6] that can predispose to such rebleeds. Likewise fresh red blood cells can be found within chronic SDHs, suggesting microscopic bleeding [7], and radio-tagged red blood cells injected into the lumbar subarachnoid space appear rapidly within chronic SDHs, suggesting transfer from the subarachnoid space [7, 8]. However information about radiologically apparent rebleeding into chronic SDHs and how it presents clinically is less available. Bradford et al. [5] reported the only case series related to this topic. Using serial radiography in 115 infants with AHT, 16% (17/105) of routinely re-imaged subjects had subdural rebleeds [5]. All were asymptomatic. We sought to extend this information about rebleeding rates and to understand rebleeding predispositions and associated symptoms.

Materials and methods

We identified children younger than 3 years with SDH caused by AHT from a previously initiated database of serial cases of AHT; this database had been compiled to study the changing incidence of AHT during the recession [9] and was continued as an administrative database. Only children diagnosed with confirmed AHT by the contributing hospitals' Child Protection Teams were included in that parent study. This determination was made by comparing the history with the injuries, including the constellation of extra-calvarial injuries. It was also supplemented by the

results of routine inter-disciplinary conferences and protective services and police investigations. Most children had additional abusive injuries, histories that did not describe any trauma, described only very minor trauma or were developmentally implausible, or positive legal investigations with confessions or third-party descriptions of abuse. We excluded all infants with an indeterminate abuse diagnosis. This diagnostic methodology continued after the parent study ended. We evaluated children from both Seattle Children's Hospital and Harborview Medical Center from that parent study and similar children from states and counties not included in the parent study. This study included two phases, neither of which was funded. Children in the first phase were initially hospitalized between January 2003 and January 2009. When those data were previously submitted to *Pediatric Radiology*, the editors requested that we expand the study's time span to have a larger subject pool. Thus we undertook a second phase that included all subjects from January 2003 through September 2013. We studied only children who had an initial SDH and at least one available follow-up neuroimaging study prior to 1 year after the initial injury.

Children who sustained rebleeds as complications of medical therapy were not considered to have rebled. Rebleeds immediately concurrent with or secondary to invasive neurosurgical treatment or within the first 3 weeks after surgery were considered to have rebled because of the procedure and were not included as a rebleed. Any shunting or needle drainage of the original SDH was considered a neurosurgical procedure. However children whose new bleeding occurred more than 3 weeks after their invasive procedures or who rebled after previous post-procedure imaging that lacked rebleeding were considered to have rebled. We excluded children with ongoing coagulation disorders, infections, vascular diseases or metabolic disorders that could predispose them to initial or subsequent SDH. We included children with delayed rebleeds during prolonged initial hospitalizations.

We documented clinical details from the initial hospitalization and from the time of repeat neuroimaging. We documented available head sizes/occipital–frontal circumferences (OFCs) from the beginning and end of the initial hospitalization and time of follow-up. For children of 37 weeks' gestation or more, we converted OFCs to z-scores using AnthroCalc, based on U.S. Centers for Disease Control and Prevention (CDC) standards [10]. Z-scores were estimated by measurement from CDC premature infant growth curves for infants born before 37 weeks of gestation. When discharge OFCs were not available but children had been hospitalized 4 days or fewer, the discharge OFC was assumed to be the same as initial OFC because such short stays were unlikely to be accompanied by major brain injury and subsequent OFC changes.

The study neurologist and the child abuse pediatricians (CAPs) obtained clinical data by chart review. Patient functional status at initial hospital discharge had not been prospectively documented by any formal outcome scale. Clinical

reviewers estimated neurological outcome as “normal” (including mild impairment), “moderate” or “severe” impairment based on the child’s clinical description and competence with age-appropriate activities of daily living based on clinical notes prior to discharge. For example, children requiring tube or ostomy feeding or who had severe neurologic impairment or ongoing severe seizure disorders were considered to have severe impairments. Clinical reviewers were not blinded to rebleed status when they decided whether the children were study-eligible or when they collected clinical data.

In the first study phase, cranial imaging was reviewed separately by an attending pediatric neuroradiologist (G.E.I.) with 7 years of experience and a pediatric neurosurgery fellow in his final year of training (S.A.). By the second phase the neurosurgery fellow had graduated and the neuroradiologist was unavailable. A second attending neuroradiologist with 8 years of experience was the reviewer with a CAP (K.W.F.) who had 35 years of experience. They conducted concurrent image review that included the children reviewed during the first study phase. The final data are based on their review. Because imaging choices had been made by the children’s treating physicians, children initially and at follow-up had available CTs, MRIs or both CTs and MRIs. All imaging reviewers reread all cranial imaging from the initial hospitalization and all follow-up imaging obtained during the first post-hospitalization year until they identified SDH rebleeding on subsequent imaging. Our results reflect the information provided by all available imaging. Reviewers were blinded to the initial clinical radiology interpretations and, during the second phase, to the initial study reviews. Rebleeding was defined as the presence or new development of higher/blood density collections within or in relation to prior lower-density SDHs. Some were layered or compartmentalized in membranes and some appeared free within existing chronic SDHs. Children with dependent layering of higher-density SDH fluid within a recent acute SDH and those with only tiny collections of new high density were not considered to have rebled. Likewise, simple expansion or volume increase of lower-density SDHs without new hyperdensity was not considered rebleeding. We identified areas of early abnormal low parenchymal density or loss of normal gray–white matter differentiation on CT and areas of abnormal diffusion on MRI. For the purpose of limiting repetition and shortening the text, we include either or both as “acute parenchymal injury.”

These imaging and clinical findings when rebleeding was identified were recorded as the rebleed event. Children with no SDH rebleeding found on imaging during that first year were considered not to have rebled. Data were collected on the child’s imaging at initial presentation (including, if available, both CT and MRI), last imaging before hospital discharge and first follow-up imaging that exhibited rebleeding. For those without rebleeding on any follow-up imaging, the study

closest to the mean time till rebleeding for children who rebled was chosen for comparison. All intracranial sites, including the posterior fossa and falx, were searched for evidence of rebleeding. During the first phase, we calculated the frontal–occipital ratio (FOR: an index of internal hydrocephalus or ventriculomegaly) [11]. We calculated the FOR by summing the maximum distance between the lateral margins of frontal and occipital horns of the lateral ventricles divided by twice the maximum distance between the skull’s widest inner margins at the level of the third ventricle. Because the initial neuroradiologist thought this was not a reproducible measure and that it added little to the study results, we did not calculate the FOR during the second study phase. Additionally, during the second study phase the study form was modified based on the thoughts of both study neuroradiologists and the concerns of the initial *Pediatric Radiology* journal reviewers. The “maximum SDH depth” was documented. The neuroradiologists used coronal images, if available, of either CT or MRI to measure the maximum distance between the gyral surfaces and inner skull. If only axial images were available, the maximum extra-axial space depth was measured, avoiding the upper cuts. Either measurement was defined as “SDH depth.” The second-phase radiology reviewer recorded his binary subjective judgment whether brain atrophy and ventriculomegaly were present. This radiology judgment also integrated head size information that was provided during the concurrent review by the CAP. Disagreement between first-phase reviewers was resolved by consensus during their concurrent image review. During that first phase, continuous measurements, such as maximum SDH depth and FOR dimensions, were the mean of reviewers’ determinations. Among all three reviewers, there was no disagreement about the presence or absence of rebleeding and only minor variation of lineal measurements, except for the FOR ratio. We evaluated the initial SDH density patterns. However we also analyzed initial SDHs for evidence of the presence of acute SDH and chronic SDH. We recognize that initial mixed-density SDH collections do not equate with both acute SDH and chronic SDH but instead might represent a single recent event of hemorrhage [12–15]. To identify co-existing “acute SDH and chronic SDH” or “chronic SDH” alone, during the second study phase the neuroradiologist and CAP jointly reviewed clinical (e.g., past unexplained apnea, loss of or altered consciousness or seizures, unusually rapid head size growth, macrocephaly) and radiologic (e.g., subdural membranes, old infarcts and parenchymal clefts, split sutures, fractures without soft-tissue swelling) [14] information to determine whether acute SDH, chronic SDH or acute and chronic SDH was present at the onset of the initial hospitalization. Although we did not record which findings in addition to the SDH density pattern allowed this categorization in the individual children, most children with chronic, or acute and chronic SDHs met multiple clinical and radiologic criteria.

Statistics

Data were recorded in RedCap (Institute for Translational Health Science, Nashville, TN), which was imported into SPSS (version 19; IBM, Armonk, NY) for analysis. We used simple descriptive statistics. We compared children with and without SDH rebleeding for continuous variables by *t*-testing, and discrete variables by chi-squared testing, with appropriate corrections for small numbers. Odds ratios with confidence intervals are provided for discrete 2×2 variables with *P*-values less than 0.10. *P*-values less than 0.05 were considered significant.

Human subjects

The study was reviewed by the Seattle Children's Hospital's institutional review board (IRB) and granted approval with waiver of consent. Harborview used Seattle Children's review board's approval. Only previously collected clinical data and imaging were studied.

Results

We identified 160 children with SDH caused by AHT. Seventeen (10.6%) died during their initial hospitalization. Of the remaining 143 abused children, 85 (59.4%) were reimaged during the study time limits; they constituted the study subjects. Eighty children were initially imaged by CT and five by MRI. In all, 49 (57.6%) had MRIs that we utilized early during their initial hospital course. Fifty-four (63.5%) of the 85 children sustained a subdural rebleed within 1 year after injury, based on review of concurrently obtained CT, MRI or both types of imaging. Figures 1 and 2 provide examples of children with AHT-SDH rebleeding. Thus a minimum of 54 of the entire cohort of 143 (37.8%) children with AHT-SDH who survived their initial hospitalization sustained rebleeds. At the time of follow-up or rebleeding, 61 children were initially imaged by CT and 24 by MRI. Thirty-six (66.6%) of the 54 children with AHT-SDH who rebled had had additional interval imaging that showed decreasing SDH density or attenuation between 7 or more days after their initial imaging and separate from invasive neurosurgical procedures, and the time when they rebled. Eight children (9.4%) had a previously placed subdural shunt when they had follow-up imaging; 87.5% of them had rebleeding.

Seventy-nine (92.9%) of the 85 children with AHT-SDH were reimaged during routine head injury follow-up care; they lacked acute symptoms or signs of brain injury or dysfunction. Two children (2.4%) were reimaged for abnormal head growth. Three children (3.5%) were imaged at a clinic visit or hospitalization for seizures that had continued unchanged since their initial hospitalization. Based on the clinical records from the visit at which rebleeding was recognized, none of the

children's symptoms or seizure courses was attributable to the rebleeding. One child (1.2%) was reimaged for a cerebrospinal fluid leak from the shunt site. Three (3.5%) children who rebled during an initial prolonged hospitalization also lacked acute rebleeding symptoms. Thus, no children had acute neurologic symptoms attributable to their rebleeding.

Most children were protected from their original abuser at the time the rebleeding was identified. Twenty-eight of 85 (32.9%) children with AHT-SDH were in foster care and 20 (23.5%) in relative placements. Four other children (4.7%) remained with their parents after a non-parental perpetrator had been identified. Seventeen (20.0%) were with their non-perpetrator mother and two (2.4%) with their non-perpetrator father. Five (5.9%) children remained hospitalized during prolonged initial hospitalizations. Nine (10.6%) were with both parents; these were cases for which no perpetrator had been identified and Children's Protective Services either did not remove or had returned the child.

Sixty-eight children were evaluated at follow-up by neurosurgery (44 of the 54 [81.5%] who rebled), 16 by neurology (13/54, or 24.1% of those who rebled) and 4 by neurodevelopmental clinicians (3/54, or 5.6% of those who rebled). Seventeen (20.0%) of the 85 children had a repeat CAP evaluation at follow-up, of whom 16 (94.1%) had SDH rebleeding. Twelve children (14.1%) had a repeat skeletal survey, of whom 11 (91.7%) had SDH rebleeding. Sixteen children (18.8%) had a follow-up dedicated eye examination, of whom 13 (81.3%) had SDH rebleeding. None of these evaluations revealed evidence of new abusive injury or history, or of clinical or radiologic findings of acute head trauma. The extent of these evaluations had been determined by the attending physicians at the time of the follow-up evaluation. During the study period, our neurosurgeons and CAPs had become more comfortable that most asymptomatic rebleeds did not represent new abuse, so CAP evaluations for abuse beyond a phone discussion, as well as retinal examinations and skeletal surveys, became less frequent.

Subject demographics and initial clinical findings are in Table 1. Initial imaging findings are presented in Table 2. Follow-up clinical and imaging data are in Tables 3 and 4. Ages at initial hospitalization of children who rebled and who did not rebleed did not differ (*P*=0.87). Additionally, 35.2% (19/54) of the children who rebled were younger than 6 months and 48.1% (26/54) were 6–12 months old at injury. Fifty-four (63.5%) of the children were male and the rebleeding rate for males was greater than for females (Table 1).

At the time of follow-up none of the 19 (22.4%) children whose original SDH had completely resolved on imaging had rebled. However 72.7% (8/11) of those with unilateral, 83.6% (46/55) of those with bilateral, and 81.8% (54/66) of those with either unilateral or bilateral residual SDH rebled. Fifty-one (94.4%) AHT rebleeds were located within the region where SDH had been recognized during the initial hospitalization and

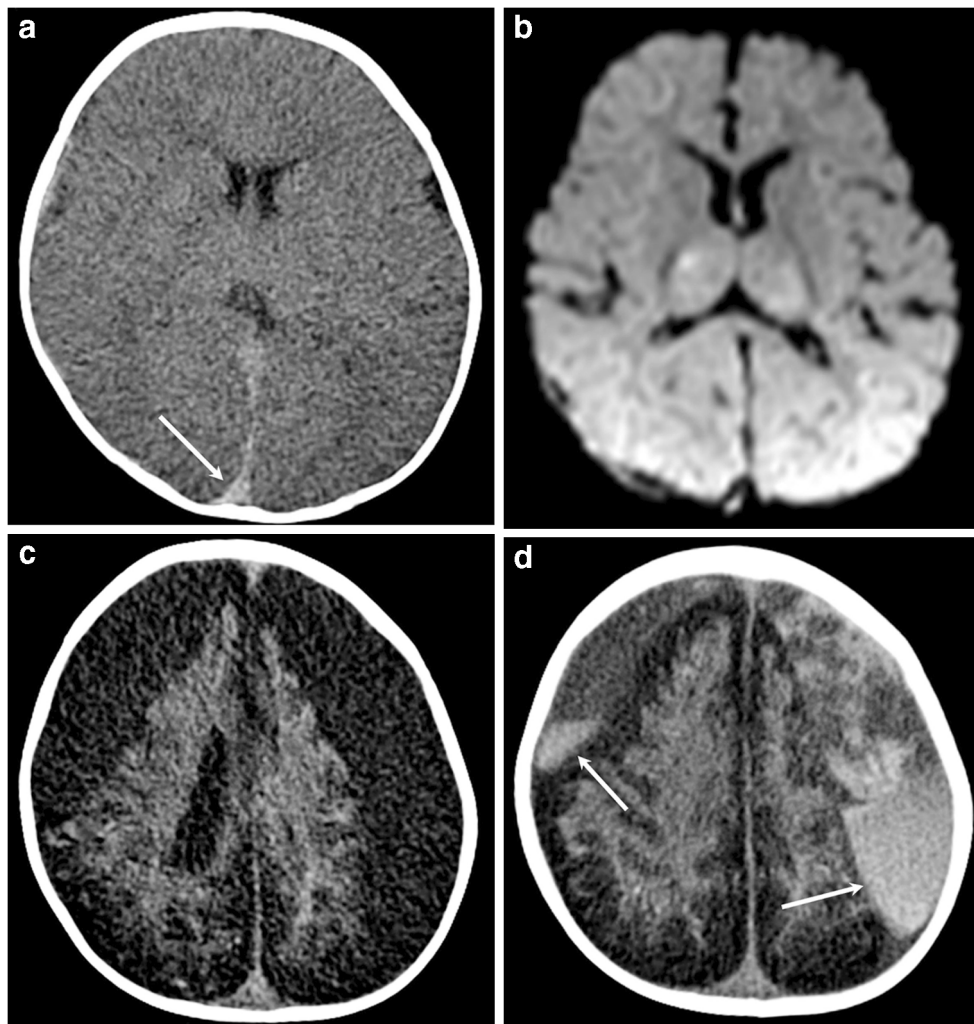


Fig. 1 Imaging in a 4-month-old girl who presented after having been found apneic in a swing by her father. Later, he told law enforcement that she had been crying; he said he grabbed her, jerked her up so that her head whipped back, then slammed her body and head down. She had a history of “easy bruising.” She had bruises, bilateral severe retinal hemorrhages, multiple classic metaphyseal lesions and multiple healing rib fractures, but no skull fractures or scalp swelling. Head size was 98%. At discharge a month after admission, she was spastic, with seizures and need for enteral feeding. **a** At presentation, axial CT shows diffuse swelling and

hypodensity as well as posterior interhemispheric acute subdural hemorrhage (SDH; *arrow*). **b** Axial diffusion-weighted MR imaging the next day demonstrates diffusion restriction throughout the cerebrum. **c** Two months later her axial CT shows diffuse cerebral atrophy with ventricular and sulcal prominence. Large bilateral chronic SDHs exhibit mass effect on the underlying parenchyma. **d** Axial CT obtained 2 months later demonstrates interval development of acute hemorrhage (*arrows*) within large, hypodense bilateral chronic SDHs

lower-density subdural collections remained present at the time of follow-up imaging. Two (3.7%) were located where SDH had not been identified initially and 1 (1.9%) had rebleeding both within and separate from the initial SDH. However all rebleeds were within lower-density subdural collections that were present when the rebleeds were identified. Among the three children who had rebleeding at sites outside their original SDH, two had severe global cerebral atrophy and low-density bilateral effusions and one severe right parietal encephalomalacia and an overlying low-density effusion as the rebleed sites on follow-up imaging.

The time from the onset of initial hospitalization to rebleed recognition averaged 81 ± 58 (standard deviation [SD]) days (Table 3; Fig. 3). Ninety percent of the rebleeds were detected

between initial hospital admission and 21 weeks later. The earliest rebleed was noted 11 days after initial imaging and the latest 341 days after initial imaging. For children without rebleeding, follow-up scans averaged 107 (SD 118) days after their initial hospitalization. As noted in the Materials and methods section, we did not review imaging beyond 365 days after the initial hospitalization.

The presence of initial cranial impact findings — scalp soft-tissue swelling, skull fracture or both — was not associated with rebleeding. Children with retinal hemorrhages at initial evaluation were not more likely to rebleed (Table 1).

We evaluated several factors to try to characterize the severity of children’s initial hospital courses, including initial

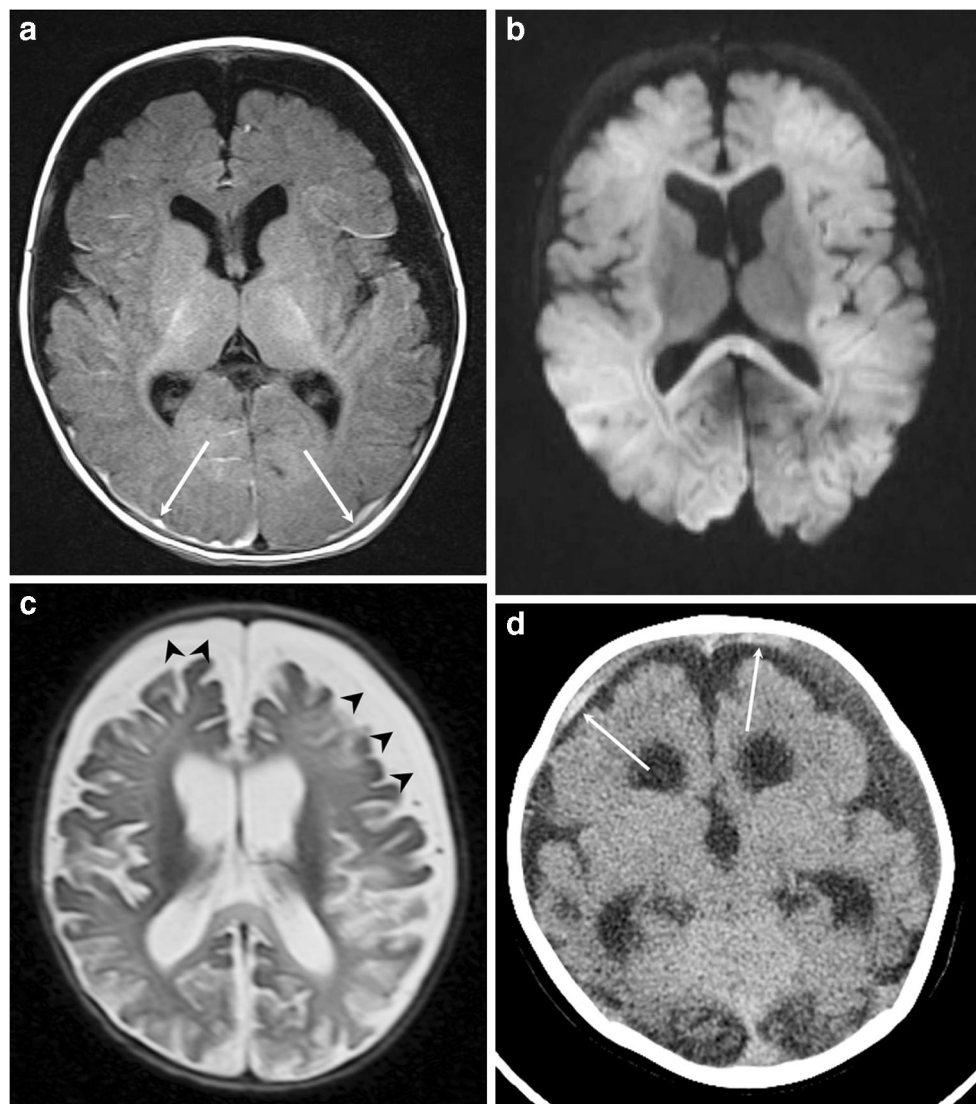


Fig. 2 Imaging in a 3-month-old boy who sustained an arrest while alone with his father. He had a plus-2.26 z-score head size at admission and profuse bilateral retinal hemorrhages but no external or skeletal injuries. At discharge he left with a seizure disorder and severe developmental delay. **a** Axial T1-W fluid-attenuated inversion recovery (FLAIR) MRI at time of presentation demonstrates bilateral hypodense subdural hemorrhages (SDHs) with acute posterior occipital and parafalcine hemorrhage (arrows). The combination of an enlarged occipital–frontal circumference, prominent bilateral subdural collections and lack of significant mass effect on the underlying brain parenchyma yielded a determination of acute and chronic subdural hemorrhage. **b** Axial

diffusion-weighted MR image at presentation demonstrates decreased diffusion throughout the bilateral cerebral hemispheres, confirmed on the apparent diffusion coefficient map (not shown). **c** Axial T2-W half-Fourier acquisition single-shot turbo spin-echo (HASTE) MRI obtained 2 weeks following presentation demonstrates diffuse cerebral atrophy with new enlargement of the subarachnoid spaces, separated from persistent hypointense SDHs by the faintly visualized arachnoid (arrowheads). **d** Axial non-contrast CT obtained 6 months after initial presentation demonstrates bifrontal rebleeding (arrows) into hypodense bilateral SDHs, accompanied by severe brain atrophy

Glasgow Coma Scale (GCS), presence of seizures, need for and duration of mechanical ventilation, hospital length of stay and functional status at discharge from initial hospitalization (Table 1). These factors did not differ between children who rebled and those who did not.

Sixty-seven (78.8%) children with AHT-SDH had bilateral or multifocal initial SDH. Twenty-four children (28.2%) with AHT-SDH had posterior fossa SDH at initial presentation. At the initial hospitalization, posterior fossa hemorrhage, basal

ganglia injury and parenchymal hemorrhages were not more common among those who rebled (Table 2). Twenty-three (42.6%) of the 54 children who rebled had developed acute parenchymal injury patterns on either CT or MRI during their initial hospitalization. Twenty-three of the 49 children who had MR imaging during the initial hospitalization developed diffusion abnormalities, of whom 16 (69.6%) subsequently rebled. However rebleeding was also present in 20 (76.9%) children who lacked initial DWI abnormalities. Acute

Table 1 Clinical comparisons of children with abusive head trauma (AHT), with and without subsequent rebleeding, at the time of initial hospitalization

	Children with AHT subdural hemorrhage (SDH)		Statistics	
	No rebleed <i>n</i> =31	Rebleed <i>n</i> =54	<i>P</i> -values OR (CI) ^a	<i>t</i> -value or chi-square
Demographics				
Age in days; mean (SD)	170 (124)	177 (174)	0.87	−0.16
Male gender mean (SD)	15 (48.4%)	39 (72.2%)	0.028 OR 0.361 (0.143–0.907)	4.83
Evidence of cranial impact injury or retinal hemorrhage				
Scalp injury	6 (19.4%)	14 (25.9%)	0.49	0.47
Skull fracture	7 (22.6%)	11 (27.5%)	0.81	0.06
Either scalp injury or skull fracture	8 (25.8%)	19 (35.2%)	0.37	0.80
Retinal hemorrhage (80 examined)	20/30 (66.6%)	39/50 (78.0%)	0.27	1.24
Clinical indications of the severity of the initial presentation				
Seizures	15 (48.4%)	35 (64.8%)	0.14	2.19
Initial GCS <13	14 (45.2%)	24 (46.3%)	0.95	0.00
Mean GCS ± SD	11.2±4.4	11.4±4.3	0.83	−0.21
Ventilated	13 (41.9%)	23 (42.6%)	0.95	0.00
Days ventilated; mean (SD)	4.2 (4.0)	5.2 (4.4)	0.48	−0.72
Hospital length of stay; mean (SD)	11.7 (15.6)	19.9 (51.2)	0.39	−0.87
Functional status at discharge				
Normal	16 (55.2%)	26 (50.0%)	0.88	0.25
Mild	7 (24.1%)	15 (28.8%)		
Severe	6 (20.7%)	11 (9.1%)		

GCS Glasgow Coma Scale, SD standard deviation

^a In addition to *P*-values, the odds ratio (OR) with confidence interval (CI) is reported for 2×2 variables for cells with *P*<0.10. Significant values by either *P*<0.05 or odds ratios are in bold

parenchymal injury patterns for the children who rebled included six with unilateral, three with focal or patchy (<15%), eight with watershed and six with diffuse (>80%) abnormalities. The presence of any initial hospitalization acute parenchymal injury pattern or abnormal DWI did not differ between those who rebled and those who did not. The initial SDH density patterns also were not significantly associated with rebleeding. We evaluated subdural collections by joint radiologist–CAP judgment whether they represented acute, chronic or both acute and chronic SDH. Acute SDH alone vs. either acute and chronic SDH, or chronic SDH alone was just short of significance (*P*=0.06, OR 1.700 [0.972–2.973]; Table 2). Likewise, the presence of preexisting brain atrophy at initial presentation was just short of significance (*P*=0.05, OR 0.399 [0.074–1.056]). Among the subjects with initial mixed-density SDH patterns, 52.9% (18/34) lacked other evidence to suggest chronic, or acute and chronic SDHs. We think these represented acute hematomas or differential clotting patterns within acute SDHs.

Brain atrophy, macrocephaly and ventriculomegaly at follow-up were significantly associated with rebleeding (Table 3). The maximum SDH depth at rebleeding was the

strongest factor associated with rebleeding (*P*<0.001). The mean maximum subdural depth at the time of rebleeding for children who rebled was 13.2 mm (SD 8.4) vs. 5.2 mm (SD 6.5) for those without rebleeding.

We only calculated FORs during review of the initial 37 children. A FOR ratio of 0.37 is considered the upper limit of normal. The mean scores at initial hospitalization were close to this, suggesting many children already had elevated FOR ratios (Table 2). In turn, this likely reflects prior brain injury. However children with higher FORs at initial presentation and at the time of rebleeding (Table 3) were not more likely to rebleed.

Initial head size z-scores were available for 72 children with AHT-SDH (Table 4). Fifty-seven children with documented head sizes had been born at 37 weeks or greater gestation and 15 were born prematurely. At initial presentation mean OFC z-scores were significantly higher for children who subsequently developed rebleeding. Twenty-seven of 72 (37.5%) children had head sizes greater than +2 z-scores at initial presentation; those with initial OFC z-scores >2.0 rebled more often. Forty-seven children with rebleeding had OFC z-scores from both the times of initial hospital discharge

Table 2 Characteristics of brain imaging and extra-axial hemorrhage at initial presentation for children with abusive head trauma (AHT) with and without subsequent rebleeding

	Children with AHT subdural hemorrhage (SDH)		Statistics	
	No rebleed n=31	Rebleed n=54	P-values OR (CI) ^a	t-value or chi-square
Preexisting brain atrophy	3 (9.7%)	15 (27.8%)	0.05 OR 0.399 (0.074–1.055)	3.8
FOR ratio of 37 children evaluated; mean (SD)	0.33 (0.03)	0.34 (0.03)	0.78	
Cytotoxic edema by either CT or MRI	12 (32.2%)	23 (55.6%)	0.73	0.12
MRI DWI abnormality	7/13 (53.8%)	16/36 (44.4%)	0.56	0.34
Acute inter-parenchymal hemorrhage	7	5	0.10 OR 2.858 (0.821–9.949)	2.64
Basal ganglia injury	4	12	0.29	1.12
Posterior fossa hemorrhage	8 (25.8%)	16 (29.6%)	0.61	0.25
SAH	5 (16.1%)	11 (20.3%)	0.60	0.27
SDH all high density	10 (33.3%)	13 (24.1%)	0.74 ^b	0.59
SDH all low density	3 (9.7%)	6 (11.1%)		
SDH mixed density	18 (58.1%)	34 (63.0%)		
SDH either low or mixed density	21 (67.7%)	40 (74.1%)	0.44 ^c	0.59
Density indeterminate	0 (0%)	1 (2.0%)		
Acute SDH only	17 (54.8%)	18 (33.3%)	0.17 ^d	0.36
Chronic SDH only	2 (6.5%)	6 (11.1%)		
Acute & chronic SDH	12 (32.3%)	29 (53.7%)		
Either chronic SDH or acute & chronic SDH	14 (45.2%)	35 (64.8%)	0.06 ^e OR 1.700 (0.972–2.973)	3.51
Maximum SDH depth; mean (SD)	8.4 (5.8)	10.1 (5.0)	0.16	–1.4

CT computed tomography, DWI diffusion-weighted imaging, FOR ratio frontal–occipital ratio, MRI magnetic resonance imaging, SAH subarachnoid hemorrhage, SD standard deviation

^a In addition to P-values, the odds ratio (OR) with confidence interval (CI) is reported for 2×2 variables for cells with P<0.10. Significant values by either P<0.05 or odds ratios are in bold

^b Compares high vs. low vs. mixed density SDH in 2×3 table

^c Compares high vs. either low or mixed-density SDH

^d Compares acute vs. chronic vs. acute and chronic SDH in a 2×3 table

^e Compares acute vs. chronic or acute/chronic SDH

and rebleed. Seven (14.9%) with rebleeding had increasing z-scores, while 21 (44.7%) had stable and 19 (40.4%) decreasing z-scores. Rebleeding was not significantly different for children with different patterns of OFC z-score changes, but it was more common for those with OFC z-scores >2.0 at the time of rebleeding. Likewise, the mean OFC z-scores at follow-up was higher for those who rebled.

Discussion

Our study confirms that SDH rebleeding is very common in children who have been hospitalized for SDH caused by AHT. Our findings and our 37.7% minimum rebleed rate for AHT survivors is higher than that of Bradford et al. [5], who found that 16% of children hospitalized for SDH subsequently rebled. As with Bradford's subjects, the children we studied

always lacked neurologic symptoms or radiologic findings of acute parenchymal injury in association with their rebleeding. Most of our subjects remained in protective environments when they rebled and no additional new abusive injuries were identified. This lack of symptoms belies the often provided legal testimony that children initially presenting for acute neurologic symptoms who are found to have mixed-density SDH or evidence of acute SDH and chronic SDH have rebled because of minor or no trauma. Although the density of the fluid within the subdural space cannot be utilized alone to determine the age of subdural collections [12–15], a search for other clinical and radiologic signs of older injuries can aid in aging the SDHs [14]. Our results suggest that these children's acute neurologic symptoms should be attributed to new, serious abusive brain trauma. Our prior companion study of abused children initially presenting with both acute and chronic SDH affirmed that 77% of them presented with acute

Table 3 Characteristics of brain imaging at time of rebleed or follow-up imaging

	Children with subdural hemorrhage (SDH) from abusive head trauma (AHT)		Statistics	
	No rebleed <i>n</i> =31	Rebleed <i>n</i> =54	<i>P</i> -values OR (CI) ^a	<i>t</i> -value or chi-square
Time to follow-up from initial presentation in days; mean (SD)	107 (118)	81 (58)	0.18	1.37
Atrophy; mean (SD)	14 (45.2%)	40 (74.1%)	0.01 OR 0.473 (0.112–0.733)	7.0
Ventriculomegaly; mean (SD)	11 (35.5%)	34 (63.0%)	0.015 OR 0.324 (0.129–0.812)	5.97
FOR ratio in 37 children evaluated; mean (SD)	0.35 (0.13)	0.36 (0.06)	0.66	0.01
Maximum SDH depth; mean (SD)	5.2 (6.55)	13.2 (8.4)	<0.001	−4.12

^a In addition to *P*-values, the odds ratio (OR) with confidence interval (CI) is reported for 2×2 variables for cells with *P*<0.10. Significant values by either *P*<0.05 or odds ratios are in bold

FOR ratio frontal–occipital ratio, SD standard deviation

neurologic symptoms [16]. An additional 8% presented for increasing OFCs with rebleeds into existing chronic SDHs, but they had been asymptomatic except for enlarging head sizes. Only rarely, such as in Hymel et al.'s [1] study, are subtle symptoms from atraumatic rebleeding caused by space-occupying blood causing increased intra-cranial pressure rather than by new primary brain injury.

Rebleeding was associated with the presence of brain atrophy, ventriculomegaly, macrocephaly and greater maximum depth of SDHs at the time of rebleeding. We did not study whether the presence or absence of membranes alone was associated with rebleeding, but we used the presence of membranes when we determined whether children had chronic, or acute and chronic SDH. Although initial CT findings of acute parenchymal injury or MRI diffusion abnormalities often could be seen to progress to brain atrophy and deep SDHs in our subjects, neither initial MRI diffusion abnormalities nor CT findings of acute parenchymal injury were significantly associated with rebleeding. This might be because both were analyzed solely as “present” or “absent” and they were common in children both with and without rebleeding. CT, and to a lesser extent MRI, due to image timing, also might not have detected significant acute parenchymal injuries/DWI abnormalities. Because the maximum SDH depth at rebleed was a continuous variable, it was likely more sensitive to the severity of brain injury than those binary data points. The density patterns of the initial SDHs were not significantly associated with rebleeding. Evidence of initial cranial impact trauma or retinal hemorrhage was not associated with rebleeding. Other indices of the severity of the children's initial head injuries — seizures, initial coma, days ventilated, hospital length of stay and functional status at discharge — were not associated with rebleeding. It might be surprising that the children's functional status at initial hospital discharge did not predict rebleeding, but the clinical notes to evaluate functional status were limited

and the functional status soon after injury might have been poorly predictive of the children's eventual outcome.

Children with deep SDHs at rebleeding have in common the presence of large established subdural collections with membranes and previously injured brains that developed atrophy with secondarily increased extra-axial spaces. These factors could cause rebleeding by themselves or through friable subdural neo-membrane vessels and large collections of blood products in fibrinolytic environments [6]. Rebleeding could also be caused by a “small brain-in-a-big-box” phenomenon because of increased brain mobility within the expanded skull. With established subdural membranes and large subdural collections, intact bridging veins should not cross the subdural space, but greater brain mobility inside the skull might still occur. We identified no bridging vein ruptures at the time of rebleeding.

As expected for rebleeding, all rebleeds occurred within established chronic SDHs [16]. Most children bled where SDHs had been present during their initial hospitalization, but three children lacked initially identified SDHs where their rebleeds eventually occurred. However by the time of rebleeding, extensive brain atrophy had resulted in low-density subdural collections at the sites where their rebleeding occurred.

The presence of pre-existing chronic SDHs and pre-existing brain atrophy at initial presentation were both close to significantly related to rebleeding. These issues fit the same chain of rebleeding causation. With additional subjects, these factors might become significant.

The timing of SDH rebleeding we observed is consistent with the recognized patterns of SDH maturation and healing. During initial SDH evolution, inner neo-membranes with friable new vessels develop. However toward the end of the first year the membranes mature and become less vascular [13, 17].

Large OFCs at initial presentation and at follow-up were associated with rebleeding. However rebleeding occurred in many children with stable or decreasing OFC z-scores. In spite

Table 4 Patterns of head size/occipital frontal circumference (OFC)

	Children with abusive head trauma (AHT) subdural hemorrhage (SDH)		Statistics	
	No rebleed <i>n</i> =31	Rebleed <i>n</i> =54	<i>P</i> -values ^a OR (CI)	<i>t</i> -value or chi-square
Mean OFC z-scores at initial presentation	0.77 (1.41)	2.13 (1.60)	<0.001	−3.67
% initial OFC z-scores ≥ 2.0	4 of 27 (14.8%)	23 of 45 (51.1%)	0.002 OR 0.290 (0.050–0.559)	9.49
Mean OFC z-score at rebleed or follow-up	0.87 (1.46)	1.72 (1.70)	0.038	−2.12
% OFC z-scores ≥ 2.0 at rebleed or follow-up	6 of 22 (27.3%)	21 of 47 (44.7%)	0.011 OR 0.182 (0.054–0.616)	2.54
% OFC z-scores ± 0.5 of initial discharge OFC z-score at rebleed or follow-up	11 (50.0%)	21 (44.7%)	0.27	2.66
% OFC z-scores < -0.5 below initial discharge score at rebleed or follow-up	5 (22.7%)	19 (40.4%)		
% OFC z-scores > 0.5 above initial discharge score at rebleed or follow-up	6 (27.3%)	7 (14.9%)		

^a In addition to *P*-values, the odds ratio (OR) with confidence interval (CI) is reported for 2×2 variables for cells with *P*<0.10. Significant values by either *P*<0.05 or odds ratios are in bold

of these stable or decreasing OFC z-scores, many had very large extra-axial collections. This indicates these children were undergoing significant post-injury brain atrophy.

The Hanley rule allows prediction based on the study size of the maximum statistically likely rate at which an event could occur if the study identified no such events [18]. Fifty-four of our subjects plus Bradford et al.'s [5] 17 subjects who rebled all lacked acute neurologic symptoms and were without evidence of any new brain trauma. By the Hanley rule, we estimate the maximum predicted frequency of non-traumatic rebleeding that would be accompanied by acute neurologic symptoms in children with prior SDH to be 3.7%.

There are several limitations of our study. AHT lacks a diagnostic gold standard but instead relies on integrating all

factors of the child's presentation and investigation. Although 143 children with AHT were initially identified as possible subjects, eventually only about 60% met our study criteria. This reduced our study size and probably left us with some equivocal statistical results because of insufficient study power. As a retrospective study, initial and follow-up visit data and imaging were not consistently obtained and did not occur at consistent post-injury intervals. Early during the study, most children who rebled had complete CAP, skeletal and retinal evaluations. However as treating clinicians became more comfortable with the benign, atraumatic nature of these rebleeds, full evaluations became less common. Although we identified no additional injuries in the infants who rebled, we cannot rule out clinically occult injuries in the children who did not have CAP valuations, skeletal surveys or retinal exams.

Based on the IRB-approved protocol, we did not collect information about the children who lacked follow-up imaging. Children's discharge notes rarely specify why or why not follow-up is planned and appointment failures rarely document a reason. However a variety of reasons for lack of follow-up (e.g., initial treating physicians' decision to forgo follow-up care and imaging, distance from our medical center, family animosity over abuse diagnosis, milder initial injury not warranting reimaging) could have variably affected the children's likelihood to be reimaged and to be found to have rebleeding. Anecdotally, many children with severe injuries had come to us from as far away as Alaska and Montana. Thus our subjects might not fully represent the entire population of children with initial AHT-SDH. However it remains quite likely that some children who were not reimaged developed rebleeds, making our total population rebleed rate of 38% a minimum estimate.

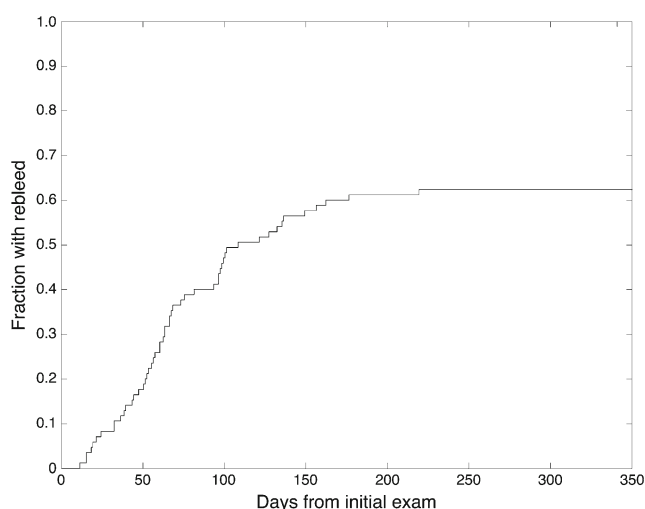


Fig. 3 The cumulative incidence of rebleeding in children with prior subdural hemorrhage from abusive head injury (*n*=85) who subsequently developed subdural rebleeding (*n*=54)

Because this was a retrospective study, the cranial imaging that was obtained was determined by the treating physicians. During the early years of the study, MRIs were less routinely obtained during the children's initial hospitalizations. If they had been routinely obtained several days after injury, we suspect that we would have identified more acute parenchymal injuries. Diffusion studies at that time should have been more sensitive than the nearly universally obtained CTs. Further, because most rebleeding was identified during neurosurgery follow-up encounters and the neurosurgeons were mainly interested in whether intervention was needed for large SDHs, they often used limited-sequence MRIs to avoid the sedation required for full-sequence MRIs and the radiation of CTs. These varied and limited imaging sequences impaired our ability to determine whether rebleeding was into chronic SDHs vs. subdural hygromas.

We attempted to not only document subdural densities at initial presentation, but to also decide, based on additional clinical and radiologic criteria, which children actually had chronic, or both acute and chronic SDH. This required integration of radiologic and clinical data and clinical judgment. Likewise the identification of brain atrophy and ventriculomegaly was a qualitative radiologic judgment.

Conclusion

In children who have been hospitalized for SDH caused by AHT, subsequent rebleeding into established chronic SDHs is quite common but almost always asymptomatic. Children with brain atrophy, ventriculomegaly, macrocephaly and deep SDHs at follow-up are prone to rebleed. All rebleeding occurred within established chronic SDHs and none in children whose SDHs had resolved. Acute parenchymal abnormalities during the initial hospitalization can lead to brain atrophy, which eventually leads to those deep chronic SDHs. The rebleeds we saw occurred without evidence of new clinical or radiologic trauma. We were unable to associate rebleeding risk with initial cranial impacts or the severity of the initial clinical course. Although not seen by us, children with SDH rebleeding rarely might present with symptoms of mass effect from their rebleeds. We estimate that the maximum predicted frequency of acute neurologic symptoms from non-traumatic rebleeding into chronic SDHs resulting from prior AHT is 3.7%. Assertions within the legal realm that children with acute neurologic symptoms accompanying SDH have sustained non-traumatic rebleeding into pre-existing chronic SDHs are specious unless those symptoms are simply related to space-occupying new blood.

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Compliance with ethical standards

Conflicts of interest Drs. Metz, Brown and Feldman have provided legal consultation and testimony in child abuse cases.

References

1. Hymel KP, Jenny C, Block RW (2002) Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. *Child Maltreat* 7:329–348
2. (2013) Notice of disciplinary action: Member censure. AANS Neurosurg 22. <http://v1archives.aansneurosurgeon.org/210613/8/3268>. Accessed 1 May 2019
3. Dias MS, Backstrom J, Falk M, Li V (1998) Serial radiography in infant shaken impact syndrome. *Pediatr Neurosurg* 29:77–85
4. Wuerfel nee Tysiak E, Petersen D, Gottschalk S et al (2012) Progression of chronic subdural hematomas in an infant boy after abusive head trauma. *Eur J Paediatr Neurol* 16:736–739
5. Bradford R, Choudhary AK, Dias MS (2013) Serial neuroimaging in abusive head trauma: timing of injuries. *J Neurosurg Pediatr* 12: 110–119
6. Nomura S, Kashiwagi S, Fujisawa H et al (1994) Characterization of local hyperfibrinolysis in chronic subdural hematomas by SDS-PAGE and immunoblot. *J Neurosurg* 81:910–913
7. Ito H, Yamamoto S, Saito K et al (1987) Quantitative estimation of hemorrhage in chronic subdural hematoma using ^{51}Cr erythrocyte labeling method. *J Neurosurg* 100:862–864
8. Zouros A, Bhargava R, Hoskinson M, Aronyk KE (2004) Further characterization of traumatic subdural collections of infancy. *J Neurosurg Pediatr* 100:512–518
9. Berger RP, Fromkin JB, Stutz H et al (2011) Abusive head trauma in a time of increased unemployment a multicenter analysis. *Pediatrics* 128:637–643
10. (2019) Tools and calculators. BC Children's Hospital. <http://endodiab.bcchildrens.ca/ForProfessionals/AnthropometricCalculators.htm>. Accessed 10 June 2019
11. O'Hayon BB, Drake JM, Ossip MG et al (1998) Frontal and occipital horn ratio: a linear estimate of ventricular size for multiple imaging modalities in pediatric hydrocephalus. *Pediatr Neurosurg* 29:245–249
12. Sieswerda-Hoogendoorn T, Postema FAM, Verbaan D et al (2014) Age determination of subdural hematomas with CT and MRI: a systematic review. *Eur J Radiol* 83:1257–1268
13. Stoodley M, Weir B (2000) Contents of chronic subdural hematoma. *Neurosurg Clin N Am* 11:425–434
14. Adamsbaum C, Morel B, Ducot B et al (2014) Dating the abusive head trauma episode and perpetrator statements: key point for imaging. *Pediatr Radiol* 44:S578–S588
15. Vezine G (2009) Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI. *Pediatr Radiol* 39:586–590
16. Feldman KW, Sugar NF, Browd SR (2015) Initial clinical presentation of children with acute and chronic vs. acute subdural hemorrhage due to abusive head trauma. *J Neurosurg Pediatr* 16:177–185
17. Lee K-S (2004) Natural history of chronic subdural hematoma. *Brain Inj* 18:351–358
18. Hanley JA, Lippman-Hand A (1988) If nothing goes wrong, is everything alright? *J Am Med Assoc* 249:1743–1745

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