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Editorial

External Hydrocephalus as a Cause of Infant Subdural Hematoma: Epidemiological and Radiological Investigations of Infants Suspected of Being Abused



PEDIATRIC NEUROLOGY

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ABSTRACT

Background: Acute subdural hematoma (ASDH) and chronic subdural hematoma (CSDH) in infants have been regarded as highly specific for abuse. Other causes of CSDH have not been investigated in a large population.

Purpose: The purpose of this study was to investigate to what extent external hydrocephalus is present in infants with ASDH and CSDH undergoing evaluation for abuse.

Material and methods: Eighty-five infants suspected of being abused, with ASDH (n = 16) or CSDH (n = 69), were reviewed regarding age, risk factor profiles, craniocortical width (CCW), sinocortical width (SCW), frontal interhemispheric width (IHW), subarachnoid space width (SSW), and head circumference (HC). In infants with unilateral subdural hematoma (SDH), correlations between contralateral SSW and ipsilateral CCW and SDH width were investigated.

Results: Infants with CSDH had significantly lower mortality, were more often premature and male, and had significantly higher CCW, SCW, IHW, and SSW than infants with ASDH (P < 0.05). Ipsilateral CCW (R = 0.92, P < 0.001) and SDH width (R = 0.81, P < 0.01) correlated with contralateral SSW. Increased HC was more prevalent in infants with CSDH (71%) than in infants with ASDH (14%) (P < 0.01). Forty-two infants, all with CSDH, had at least one of CCW, SCW, or IHW \geq 95th percentile. Twenty infants, all with CSDH, had CCW, SCW, and IHW >5 mm, in addition to increased HC.

Conclusion: A substantial proportion of infants with CSDH who had been suspected of being abused had findings suggesting external hydrocephalus.

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Conflict of interest: Jacob Andersson has no conflicts of interest to declare. Johan Wikström has served as a mostly unpaid expert for the defense in a few cases of suspected AHT in Swedish and Norwegian courts and has on one occasion assisted the police in an investigation of suspected AHT. Ulf Högberg has served as a mostly unpaid expert witness for the prosecution or the defense in a few cases of suspected infant abuse. Ingemar Thiblin has written statements and appeared in court in child abuse cases at the request of the Legal Counsel of the National Board of Health and Welfare, the prosecutor or the defense, all as part of his regular duties. Knut Wester has served as a mostly unpaid expert witness for the court or the defense in a few cases of suspected AHT in Norwegian and Swedish courts. * Communications should be addressed to: Dr. Andersson; Forensic Medicine;

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Introduction

Subdural hematoma (SDH) is encountered more frequently in infants than in older children.¹ A Swedish national registry study found an incidence of 16.5 infants with SDH per 100,000 infants, with a mean age of 3.3 months, median age of 2.5 months, and a male preponderance for all SDH subgroups.² One limitation of that study was that the International Statistical Classification for Diseases—Tenth Revision (ICD-10) does not differentiate between acute subdural hematoma (ASDH) and chronic or mixed SDH/ hygroma (CSDH). No study on a large population has investigated the possibility that infants referred with suspected abusive head

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trauma (AHT) could be suffering from external hydrocephalus (benign external hydrocephalus [BEH], benign enlargement of subarachnoid space [BESS], macrocephaly etc.) complicated by a CSDH.³⁻⁷ External hydrocephalus has a marked male preponderance,⁸ and prematurity is relatively frequent⁹; the condition has also been suggested as a possible pitfall in the diagnosis of AHT.^{4,10-12}

Purpose

It is reasonable to expect that a traumatic event will cause an ASDH; however, there are also rare nontraumatic causes such as bleeding disorders, central venous thrombosis, vessel malformations, or genetic diseases. The nontraumatic differential diagnoses are to a large degree possible to confirm by thorough medical examination, although small-vessel malformations can be missed in neuroimaging, and central venous thromboses have a range of etiologies.^{13,14} CSDH, on the other hand, has multiple possible etiologies that may be hard to identify at the time of detection, for example, birth^{15,16} or BEH.^{5,6,10,12,17}

Based on earlier studies,^{2,4,5,12,17,18} we hypothesized that BEH would be present more often in infants with CSDH than in those with ASDH. In Sweden, such cases would have been referred to the National Board of Forensic Medicine owing to suspected AHT. The aim of the present study was thus to investigate the proportion of BEH in infants with ASDH and CSDH undergoing evaluation for abuse.

Material and Methods

The present study is a descriptive review of a national series of infants aged younger than one year, with an SDH or subdural hygroma, who had been subjected to forensic investigation owing to the presence of SDH without an obvious cause. The presence or nonpresence of other medical findings or history of trauma was not considered for inclusion/exclusion. SDHs due to obstetric or neonatal malpractice, traffic accidents, multistorey falls, or similar high-energy trauma were not included. The cases were retrieved from the computerized register of the Swedish National Board of Forensic Medicine and had been registered during the period January 1, 1994, to December 12, 2018.

A total of 1380 infants were identified from that period, of whom 497 had been subjected to clinical medicolegal investigation and 883 had died and had therefore undergone an autopsy.

In this total, 1249 infants did not have any SDH or an SDH as a result of an obvious perinatal or obstetrical complication, a traffic accident, or a multistorey fall. Among the remaining 130 infants, neuroimaging at the time of diagnosis was available in 96 cases and 85 of those had subdural fluid that could be classified as ASDH or CSDH. These 85 cases comprised the study population. See the flowchart in Fig 1 for an overview of the database.

Our data consist of information available from the aforementioned register and included forensic reports and, to a varying extent, hospital records and birth records.

Some infants had other findings such as skull fracture, extracranial fractures, retinal hemorrhages, subarachnoid hemorrhage, cerebral venous thrombosis, hypoxic ischemic injury, or other parenchymal injury. Our aim in the present study was however only to study findings related to BEH on a group level and not to finally conclude whether an infant had suffered abuse or a spontaneous SDH.

Neuroimaging procedure

Neuroimaging data were collected from the respective hospitals and interpreted by a specialist in neuroradiology (J.W.). Both magnetic resonance imaging (MRI) and computed tomography (CT) data were available in 65 cases, only CT in 27 cases, and only MRI in four cases. Among the cases that had a confirmed SDH on the neuroimaging (n = 85), both MRI and CT were available in 61 cases, only CT in 20 cases, and only MRI in four cases.

Cases were divided into two groups for later analysis of risk factors and neuroimaging comparison. Where CT showed subdural fluid predominantly hyperattenuating compared with adjacent brain parenchyma, the hematoma was defined as an ASDH, while a hematoma with predominantly isodensity/hypodensity was considered to be a CSDH. In cases without CT, T1-and T2-weighted sequences on MRI were used to assess the type of SDH, where signal intensity similar to cerebrospinal fluid was interpreted as a sign of a CSDH. Craniocortical width (CCW), sinocortical width (SCW), and frontal interhemispheric width (IHW) are possible to measure on either CT or MRI. Although subarachnoid space width (SSW) could be measured on MRI in most cases, this was possible on CT in only a few cases, as the subarachnoid and subdural spaces are more difficult to distinguish. If the MRI was performed more than four weeks after the CT (n = 4), we did not measure SSW on MRI because the MRI could not be considered representative for the measurements at the time of diagnosis.

Risk profiles were analyzed with regard to age at diagnosis, sex, prematurity, and multiple birth and death; differences were estimated with Fisher's exact tests and proportion tests.

Infants were reviewed with regard to available HC charts, HC at the time of diagnosis as measured on CT, and radiological characteristics of BEH, such as left and right measures of CCW, SCW, SSW,

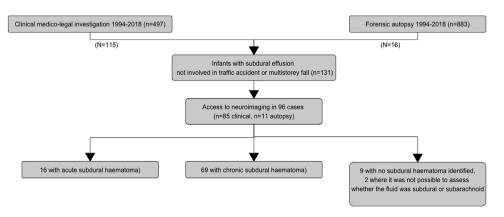


FIGURE 1. Flowchart for inclusion.

in addition to IHW. All measurements were made in millimeters at the level of the foramen of Monro. Wilcoxon's signed-rank test was used to calculate differences of means. Spearman's rank-order correlation was used to estimate a correlation between contralateral SSW and ipsilateral CCW and SDH width (CCW-SSW) in infants with unilateral SDH located at the level of the foramen of Monro (n = 10), to investigate the possibility that the SDH was simply replacing a pre-existing widened subarachnoid space.

Increased head circumference

In Sweden, HC is routinely measured during infancy at child care centers and HC charts are plotted with standard deviations (SDs). For infants with HC data from growth charts or with repeated measurements of HC, data were plotted manually and the infants were subsequently assigned to one of four categories: (1) normal HC; (2) rapidly increasing HC exceeding two SDs from normal range; (3) rapidly increasing HC exceeding two SDs from normal range and an HC \geq 3 SDs from normal range; (4) no rapid increase in HC, but a large head from birth—defined as an HC \geq 3 SDs from normal range.

Infants with increased HC and ASDH or CSDH, categories 2-4, were compared with infants with normal HC and ASDH or CSDH, category 1, using Fisher's exact test.

HC was also measured on the initial axial CT scan at the level of the foramen of Monro.

SDs for males and females were imported into R from the World Health Organization's website (https://www.who.int/childgrowth/ standards/hc_for_age/en/), and the HC of each infant was inserted. Data for premature infants were age-adjusted.

Benign external hydrocephalus

BEH is usually defined as a rapid increase of HC in combination with increased SSW; however, it is difficult to find a distinct, generally accepted radiological definition.⁸ Lam et al.¹⁹ have suggested using CCW, SCW, and IHW as indicative measures of BEH, while also taking age into account. They created a function that can be used to estimate normal values. In their definition, infants with CCW, SCW, or IHW $\ge 95^{\text{th}}$ percentile should be viewed as possibly having BEH.

A plot was created based on the functions for CCW, SCW, and IHW (Table 1) from the study by Lam et al., and the measurements for each infant were inserted into the plot.

Hussain et al. did not find a correlation between SSW and age and described an overlap between infants with and without BEH.²⁰ They recommended that infants who did not have clinical features of BEH should be considered to belong to the normal population.

We therefore used a definition in which all of the measurements of CCW, SCW, and IHW had to be above 5 mm and HC had to be

TABLE 1.

Normal Values of CCW, SCW, and IHW as a Function of Age-From the Study by Lam et al.19

Measurement	Function (Mean + coef1 \times age - coef2 \times age ²)
CCW	$2.32845 + 0.208036 imes age - 0.003709 imes age^2$
SCW	$2.220131 + 0.12409 imes age - 0.002552 imes age^2$
IHW	$2.874066 + 0.12765 \times age - 0.002084 \times age^2$

Abbreviations:

CCW = Craniocortical width

IHW = Interhemispheric width SCW = Sinocortical width

Function: mean = mean value at birth: coef1 = increase coefficient up to 28 weeks: age = age in weeks; coef2 = decrease coefficient after 28 weeks.

increased for BEH to be indicated. The differences were compared using Fisher's exact test.

Assuming that CSDH could be a complication of BEH and that ASDH to a greater extent would be of traumatic origin, one would logically expect that risk factor profiles and CCW, SCW, IHW, and SSW would differ substantially between the CSDH and the ASDH groups.

The correlation between the ipsilateral CCW and SDH width and contralateral SSW in infants with unilateral SDH was used to investigate the hypothesis that the SDH only filled a pre-existing space in infants with increased SSW.

Software

The R studio packages tidyverse, ggpubr, and rstatix were used for descriptive statistics and statistical computations.

Ethical approval

This study was approved by the Regional Ethical Review Board in Uppsala 2015/039 and 2015/040.

Results

It was possible to determine the type of subdural fluid in 85 of 96 cases. Sixteen infants had ASDH (19%), and 69 had CSDH (81%). Nine cases had been diagnosed with an SDH, but no subdural fluid could be identified at the re-assessment. In two cases, it was not possible to assess whether the fluid was subdural or subarachnoid (only CT imaging was available in these cases).

Risk factor profiles

There were marked differences between ASDH and CSDH regarding prematurity (P < 0.05—more common in CSDH) and mortality (*P* < 0.001—higher in ASDH) and a numerical difference for being male (P = 0.39). When comparing sex distribution with an expected frequency of 50 %, infants with CSDH, but not ASDH, had a higher proportion of male infants (P > 0.01) (see Table 2).

There was a significantly higher proportion of male infants with CSDH (68%, P > 0.01) than an expected frequency of 50%; this was not seen in infants with ASDH (56%, P = 0.8). See Table 1 for test statistics.

Most infants with ASDH were identified during the first month of life (0-30 days), with mean and median ages of 2.6 and 2.0 months, respectively, whereas infants with CSDH were most often identified during the third month of life, with mean and median ages of 3.0 and 2.5 months, respectively. See Fig 2 for age distribution details for the two groups.

Fifty-six percent of the infants with ASDH were males, one (6%) was born preterm (week 31), 13% had a twin, and the mortality was high. at 44%.

In the CSDH group, there were 68% males, 34% were born preterm (week 21-36), 12% had a twin, and the mortality was low, at 4%

See Fig 2 for age distribution among infants with ASDH and CSDH and Table 2 for risk profile comparison.

Increased head circumference

HC charts or repeated HC measurements were available for 53 infants. It was significantly more likely for infants with CSDH (71%) than for infants with ASDH (14%) to have an increased HC (P < 0.01). HCs measured on initial CTs can be seen in Fig 3.

TABLE 2.

Risk Factor Comparison Between	Infants With	CSDH and ASDH
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Variable	n	Estimate	P-value	conf.low	conf.high	Method	Alternative	p.signif	Statistic
Deceased	85	0.06 (OR)	0.00	0.01	0.33	Fisher's exact test	Two-sided	***	NA
Prematurity	72	7.55 (OR)	0.03	1.01	340.49	Fisher's exact test	Two-sided	*	NA
Male	85	1.65 (OR)	0.39	0.46	5.75	Fisher's exact test	Two-sided	ns	NA
Twin	85	0.92 (OR)	1.00	0.16	9.81	Fisher's exact test	Two-sided	ns	NA
ASDH male proportion	16	0.56 (Prop)	0.80	0.31	0.79	Prop test	Two-sided	ns	0.06 (Chisq)
CSDH male proportion	69	0.68 (Prop)	0.00	0.56	0.79	Prop test	Two-sided	**	8.35 (Chisq)

n = number of infants available for analysis; estimate calculated with CSDH as numerator and ASDH as denominator, OR = odds ratio; Prop = proportion; Chisq = value of Pearson's chi-squared test statistic; ns = not significant, * = P < 0.05; ** = P < 0.01; *** = P < 0.001; NA = not available; ASDH = acute subdural hematoma; CSDH = chronic subdural hematoma.

Neuroimaging measurements and correlation

Measurements for CCW, SCW, and IHW were obtained in all 85 cases. SSW could be measured in 68 cases (CSDH = 57, ASDH = 11). Infants with CSDH had significantly higher CCW, SCW, IHW, and SSW than infants with ASDH (see Fig 4). In infants with unilateral SDH, there were significant correlations between contralateral SSW and ipsilateral CCW (P < 0.001) and SDH width (P < 0.01), respectively, which can be seen in Fig 5.

CCW, SCW, and IHW \geq 95th *percentile*

No infants with ASDH had a CCW, SCW, or IHW $\ge 95^{th}$ percentile, as suggested by Lam et al.,¹⁹ but 42 infants with CSDH (49% of all infants, 60% of the infants with CSDH) had at least one measurement which was $\ge 95^{th}$ percentile. See Table 3 and Fig 6.

CCW, SCW, and IHW >5 mm and increased HC (category 2-4)

There were 53 infants with HC charts or repeated HC measurements and data on CCW, SCW, and IHW available. Twenty infants with CSDH (38% of all 53 infants, 44% of the infants with CSDH) had all measurements CCW, SCW, and IHW >5 mm and an increased HC, but no infants with ASDH had this combination (P = 0.02).

Discussion

The aim of the present study was to investigate the hypothesis that BEH would be more prevalent in infants with CSDH than in those with ASDH. The empirical consequences would be different risk profiles, HC, and neuroimaging characteristics of infants with CSDH and ASDH who were referred with suspected AHT.

Infants with CSDH were more likely to be premature, had a lower mortality, and were more often male than infants with ASDH. These observations indicate that infants with CSDH and ASDH should be viewed as separate groups.

There was a clear difference between infants with CSDH and ASDH regarding HC, CCW, SCW, IHW, and SSW, indicating that substantial proportion of infants with CSDH had findings suggestive of BEH.

SDH is known to be a complication of BEH. It is believed that the expansion of the subarachnoid space stretches the bridging veins that extend from the cortex to the dural sinuses. The stretching of the veins may cause spontaneous bleedings^{3–6} or bleedings after

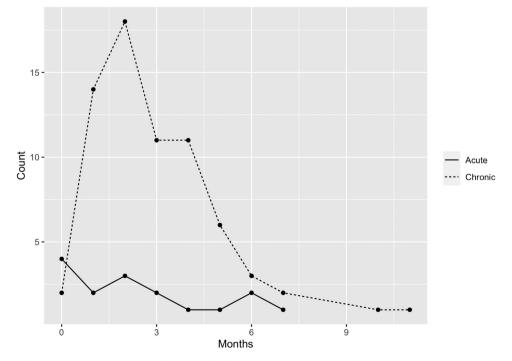


FIGURE 2. Infants with ASDH and CSDH had different age distributions. CSDH, chronic subdural hematoma; ASDH, acute subdural hematoma.

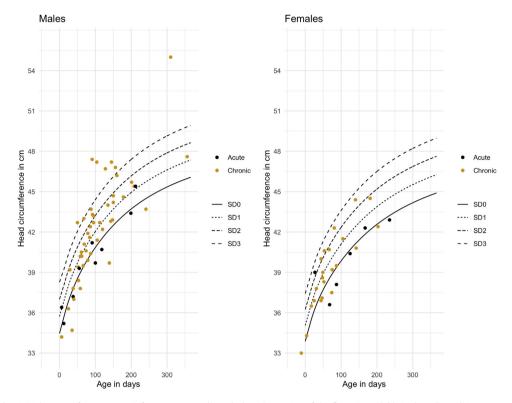


FIGURE 3. HC measured on initial CT. Data for premature infants were age-adjusted. The color version of this figure is available in the online edition. CT, computed tomography; HC, head circumference.

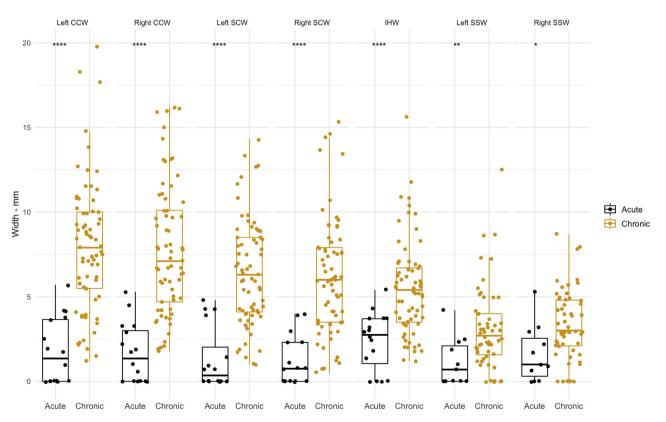


FIGURE 4. CCW, SCW, IHW, and SSW comparison between CSDH and ASDH. Asterisks represent *P*-values (*: <0.05, **: <0.01, ***: <0.001 and ****: <0.0001). Infants with CSDH (n = 69; n = 57 for SSW) had significantly higher means for all measurements than infants with ASDH (n = 16; n = 11 for SSW). The color version of this figure is available in the online edition. CSDH, chronic subdural hematoma; ASDH, acute subdural hematoma; CCW, craniocortical width; SCW, sinocortical width; IHW, interhemispheric width; SSW, subarachnoid space width.

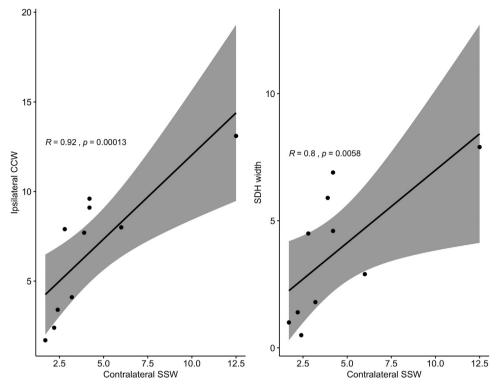


FIGURE 5. Correlation between contralateral SSW and ipsilateral CCW and SDH width in infants with unilateral SDH located at the foramen of Monro (n = 10). There was a significant correlation between contralateral SSW and ipsilateral CCW (R = 0.92, P < 0.001) and SDH width (R = 0.8, P < 0.01). SDH, subdural hematoma; CCW, craniocortical width; SSW, subarachnoid space width.

minor trauma.^{11,21,22} In the present study, SSW was significantly higher for infants with CSDH than for infants with ASDH.

Measures of CCW, SCW, and IHW are affected by ipsilateral SDH thickness, and it might be argued that the differences between ASDH and CSDH infants in this regard could be explained by average differences in SDH thicknesses, with CSDH being thicker than ASDH. However, the correlation between the contralateral SSW and ipsilateral CCW and SDH width in cases of unilateral CSDH indicates that the SDH replaced a pre-existing expanded sub-arachnoid space without causing midline shift or cortical or ventricular compression (see Fig 7). A similar presentation can be seen in Fig 8 in an infant who had a birth-related SDH and wide sub-arachnoid spaces which developed into a unilateral CSDH at the age of 27 days, as described in the study by Rooks et al.¹⁵ and also discussed by Gabaeff.¹⁶ Moreover, the greater observed SSW in CSDH than in ASDH cannot be explained by thicker CSDH.

TABLE 3.

Comparison of the Numbers of Infants With ASDH and CSDH and CCW, SCW, or IHW Measurements Over the $95^{\rm th}$ Percentile

Measurements	Acute	Chronic	P-value
Right CCW \geq 95th percentile	0	25	<0.01
Left CCW \geq 95th percentile	0	24	<0.01
Right SCW \geq 95th percentile	0	30	<0.001
Left SCW \geq 95th percentile	0	35	<0.001
IHW \geq 95th percentile	0	11	Not significant
Any value in the 95th percentile	0	42	<0.0001

Abbreviations:

 $\mathsf{ASDH} = \mathsf{Acute\ subdural\ hematoma}$

CCW = Craniocortical width CSDH = Chronic subdural hematoma

IHW = Interhemispheric width

SCW = Sinocortical width

It is possible that repetitive rebleeding into a CSDH/hygroma may cause the CSDH, and thus the HC, to grow. Several mechanisms have been described to account for rebleeding into a CSDH, perhaps such a mechanism explains why there were no infants having a purely acute SDH combined with markers of BEH.^{23,24}

Clinical implications

In addition to the standard protocol of social service investigation, clinical examination and full skeletal survey, for all infants who are diagnosed with an SDH, the investigators should have head circumference charts available and measure CCW, SCW, IHW, and SSW to not overlook a possible BEH diagnosis.

Strengths and limitations

This study covers all cases referred to the Swedish National Board of Forensic Medicine for suspected AHT during a period of nearly 25 years. This authority manages all medicolegal death investigations in Sweden. Therefore, this study can be regarded as based on the entire population of infants with SDH suspected to be caused by AHT.

This study did not aim to conclude finally whether an individual infant has been abused or did suffer from spontaneous SDH or SDH from minor trauma. A control group consisting of infants with known trauma and CSDH and known nontraumatic CSDH was not possible to construct from the present database. Thus, the true positive and true negative rate cannot be ascertained from the study design. A prospective study would be of importance to understand the association between BEH and CSDH and to investigate the possibility of causation.

Nondeceased infants with physical findings that have led to concern of possible abuse are probably referred to this authority

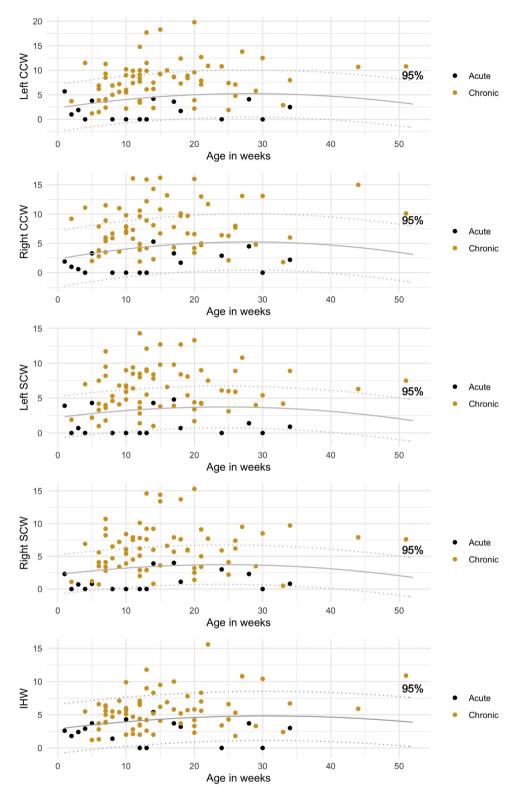


FIGURE 6. Forty-two infants with CSDH but no infants with ASDH had one or more of CCW, SCW, and IHW \geq 95th percentile. The thick gray lines indicate means, and the dotted lines show 95th and 5th percentiles plotted based on the calculations for healthy infants by Lam et al. seen in Table 1. The color version of this figure is available in the online edition. CSDH, chronic subdural hematoma; ASDH, acute subdural hematoma; CCW, craniocortical width; SCW, sinocortical width; IHW, interhemispheric width.

in most instances, but it cannot be completely ruled out that some nondeceased infants were evaluated by clinicians only. However, such a selection is unlikely to result in any kind of systematic bias. We gained access to neuroimaging in 96 of 131 cases, of which 85 had an SDH; this lack of information was mainly due to some cases being from before computerization of radiology registers; thus, it is unlikely that this caused a skewed selection.

A limitation of the present study was the lack of consensus regarding what criteria should be used to diagnose BEH when using

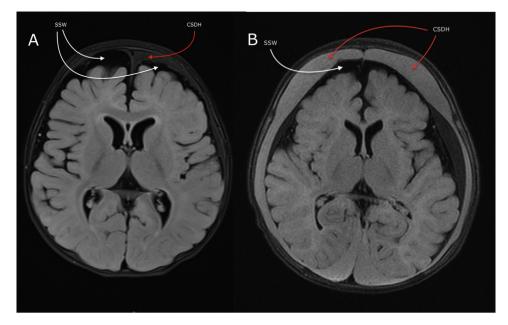


FIGURE 7. A: MRI of infant in the present study with unilateral CSDH with wide subarachnoid space contralaterally to the SDH. Note that there still is a subarachnoid space (marked "SSW") and that the CCW is the same on both sides. B: MRI of infant in the present study with bilateral CSDH with wide CSDH and visible subarachnoid space. The color version of this figure is available in the online edition. MRI, magnetic resonance imaging; SDH, subdural hematoma; CSDH, chronic subdural hematoma; SSW, subarachnoid space width.

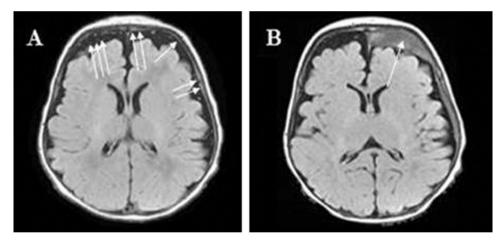


FIGURE 8. MRI of infant from Rooks et al. with birth-related SDH (A) which developed into a unilateral CSDH at the age 27 days (B). Note the relatively wide subarachnoid space and that the CSDH has filled out a pre-existing space. Reprinted with permission from the American Society of Neuroradiology. MRI, magnetic resonance imaging; SDH, subdural hematoma; CSDH, chronic subdural hematoma.

neuroimaging measurements without other clinical signs. In this study, two different definitions on neuroimaging measurements were used, one with comparison with normal values and one with measurements in combination with information on clinically increased HC. These features were present only for infants with CSDH, regardless of which definition was used. The study by Lam et al.,¹⁹ which was used to define normal values for these measurements, was performed on Chinese infants, and it is possible that the mainly Caucasian population in the present study had other normal values.

Conclusion

A substantial proportion of infants with CSDH who had been suspected of being abused had findings suggesting external hydrocephalus.

References

- Ingraham FD, Matson DD. Subdural hematoma in infancy. J Pediatr. 1944;24: 1–37.
- Högberg U, Andersson J, Squier W, et al. Epidemiology of subdural haemorrhage during infancy: a population-based register study. PLoS One. 2018;13: e0206340.
- Vinchon M, Delestret I, DeFoort-Dhellemmes S, et al. Subdural hematoma in infants: can it occur spontaneously? Data from a prospective series and critical review of the literature. Childs Nerv Syst. 2010;26:1195–1205.
- Ghosh PS, Ghosh D. Subdural hematoma in infants without accidental or nonaccidental injury: benign external hydrocephalus, a risk factor. Clin Pediatr (Phila). 2011;50:897–903.
- Ravid S, Maytal J. External hydrocephalus: a probable cause for subdural hematoma in infancy. Pediatr Neurol. 2003;28:139–141.
- Lee HC, Chong S, Lee JY, et al. Benign extracerebral fluid collection complicated by subdural hematoma and fluid collection: clinical characteristics and management. Childs Nerv Syst. 2018;34:235–245.
- Zahl SM, Wester K, Gabaeff S. Examining perinatal subdural haematoma as an aetiology of extra-axial hygroma and chronic subdural haematoma. Acta Paediatr. 2020;109:659–666.

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- 8. Wiig US, Zahl SM, Egge A, et al. Epidemiology of benign external hydrocephalus in Norway—a population-based study. Pediatr Neurol. 2017;73:36–41.
- 9. Yew AY, Maher CO, Muraszko KM, et al. Long-term health status in benign external hydrocephalus. Pediatr Neurosurg. 2011;47:1–6.
- Piatt JH. A pitfall in the diagnosis of child abuse: external hydrocephalus, subdural hematoma, and retinal hemorrhages. Neurosurg Focus. 1999;7:E5.
- 11. McNeely PD, Atkinson JD, Saigal G, et al. Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse. Am J Neuroradiol. 2006;27:1725–1728.
- **12.** Wester K. Two infant boys misdiagnosed as 'Shaken Baby' and their twin sisters: a cautionary tale. Pediatr Neurol. 2019;97:3–11.
- 13. Newton AW, Caruso PA, Ebb DH, et al. Case 5-2020: a 32-day-old male infant with a fall. N Engl J Med. 2020;382:656–664.
- Maali L, Khan S, Qeadan F, et al. Cerebral venous thrombosis: continental disparities. Neurol Sci. 2017;38:1963–1968.
- Rooks VJ, Eaton JP, Ruess L, et al. Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. Am J Neuroradiol. 2008;29: 1082–1089.
- 16. Gabaeff SC. Investigating the possibility and probability of perinatal subdural hematoma progressing to chronic subdural hematoma, with and without complications, in neonates, and its potential relationship to the misdiagnosis of abusive head trauma. Leg Med (Tokyo). 2013;15:177–192.

- Miller R, Miller M. Overrepresentation of males in traumatic brain injury of infancy and in infants with macrocephaly: further evidence that questions the existence of Shaken Baby syndrome. Am J Forensic Med Pathol. 2010;31: 165–173.
- Miller D, Barnes P, Miller M. The significance of macrocephaly or enlarging head circumference in infants with the triad. Am J Forensic Med Pathol. 2015;36:111–120.
- Lam WW, Ai VH, Wong V, et al. Ultrasonographic measurement of subarachnoid space in normal infants and children. Pediatr Neurol. 2001;25:380–384.
- Hussain ZB, Hussain AB, Mitchell P. Extra-axial cerebrospinal fluid spaces in children with benign external hydrocephalus: a case-control study. Neuroradiol J. 2017;30:410–417.
- Arkinson N, van Rijn RR, Starling SP. Childhood falls with occipital impacts. Pediatr Emerg Care. 2018;34:837–841.
- Papasian NC, Frim DM. A theoretical model of benign external hydrocephalus that predicts a predisposition towards extra-axial hemorrhage after minor head trauma. Pediatr Neurosurg. 2000;33:188–193.
 Murakami H, Hirose Y, Sagoh M, et al. Why do chronic subdural hematomas
- Murakami H, Hirose Y, Sagoh M, et al. Why do chronic subdural hematomas continue to grow slowly and not coagulate? Role of thrombomodulin in the mechanism. J Neurosurg. 2002;96:877–884.
- 24. Yamashima T, Yamamoto S. How do vessels proliferate in the capsule of a chronic subdural hematoma? Neurosurgery. 1984;15:672–678.