#### **ORIGINAL ARTICLE**



## Prevalence of cerebral sinovenous thrombosis in abusive head trauma

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#### Abstract

**Background** Cerebral sinovenous thrombosis (CSVT) has been proposed in legal settings to be an atraumatic mimic of abusive head trauma (AHT).

**Objective** The objective of this study was to determine the prevalence of CSVT and subdural hemorrhage (SDH) in a large AHT population.

**Materials and methods** This retrospective cohort study measured the prevalence of CSVT and SDH on magnetic resonance venograms in 243 patients diagnosed with AHT at a single center. We also reported additional intra- and extracranial injuries, head injury severity and length of hospital stay.

**Results** Among 243 patients diagnosed with AHT, 7% (16/243) had CSVT. SDH was present in 94% (15/16) of the CSVT cases. Cytotoxic edema and subarachnoid hemorrhage were in 88% (14/16) and 69% (11/16) of the CSVT cases, respectively. Extracranial signs of abuse were also in 100% (16/16) of the patients with CSVT. Critical to maximal head injury severity (abbreviated injury scale >=5) was in 75% (12/16) of the CSVT population vs. 33% (82/243) in the total AHT population. Length of hospital and pediatric intensive care unit stay was greater in those with CSVT (10 vs. 21.9 and 3.5 vs. 7.3 days). **Conclusion** These findings suggest that CSVT is uncommon in AHT and is associated with additional traumatic injuries and greater injury severity.

**Keywords** Abusive head trauma  $\cdot$  Child abuse  $\cdot$  Children  $\cdot$  Infants  $\cdot$  Intracranial sinus thrombosis  $\cdot$  Magnetic resonance imaging  $\cdot$  Magnetic resonance venography

### Introduction

Child maltreatment is a global health emergency [1]. In 2018, in the United States (U.S.) alone, there were 3.5 million investigations of suspected child abuse and neglect, and 678,000 victims of maltreatment [2]. Among this population, abusive head trauma (AHT) is common [3] and is one of the leading causes of mortality [4]. Common imaging

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findings associated with AHT include skull fractures, subdural hematomas, cytotoxic brain injury and injured bridging veins [5]. These findings are often the result of direct impact or rotational acceleration/deceleration forces that result in the disruption of the cortical bridging veins that traverse the subdural space.

Some medical conditions can produce findings similar to those of abuse and result in the misdiagnosis of child abuse. For example, fractures seen in patients with osteogenesis imperfecta, a disease of bone formation, can mimic fractures that are often observed in abuse [6]. Conversely, some theories of alternative etiology for the findings we observe in child abuse have been proposed, primarily in medicolegal settings, with little or no supporting evidence [7–9]. One theory, based on case reports, suggests that the subdural hematomas associated with AHT are the result of cerebral sinovenous thrombosis (CSVT) and not inflicted trauma [10–14]. These case reports, however, failed to determine the

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Table 1Study populationcharacteristics

Study population characteristics	Total study population $(n=243)$	CSVT present ( <i>n</i> =16)
Sex (female) (%)	86/243 (35%)	4/16 (25%)
Race (%)		
White	148/243 (61%)	11/16 (69%)
Black or African American	12/243 (5%)	1/16 (6%)
American Indian or Alaska Native	10/243 (4%)	0/16 (0%)
More than one race	47/243 (19%)	3/16 (19%)
Other		
Not reported/unknown	26/243 (11%)	1/16 (6%)
Ethnicity (%)		
Hispanic or Latino	69/243 (28%)	5/16 (31%)
Not Hispanic or Latino	146/243 (60%)	10/16 (63%)
Unknown/not reported	28/243 (12%)	1/16 (6%)
Median age (months) (IQR)	4 (2–10)	3 (2–21)
Head abbreviated injury scale (AIS) (%)		
AIS 3 - serious	83/243 (34%)	3/16 (19%)
AIS 4 - severe	79/243 (33%)	1/16 (6%)
AIS 5 - critical	76/243 (31%)	11/16 (69%)
AIS 6 – maximal (untreatable)	6/243 (2%)	1/16 (6%)
Hospital length of stay (mean days)	10.0	21.9
PICU length of stay (mean days)	3.5	7.3
Fractures (%)	104/243 (43%)	8/16 (50%)
Retinal hemorrhages (%)	151/243 (62%)	9/16 (56%)

*AHT* abusive head trauma, *AIS* abbreviated injury scale, *CI* confidence interval, *CSVT* cerebral sinovenous thrombosis, *IQR* interquartile range, *PICU* pediatric intensive care unit

cause-effect relationship of CSVT and subdural hematomas or other signs of trauma [15]. If CSVT mimicked AHT by causing subdural hematomas, then atraumatic CSVT cases (i.e. patients who lack other signs of trauma such as bruising, fractures, abdominal injuries and retinal hemorrhages) should not be associated with the witness or confession of abuse. Nonetheless, this theory has impacted the work-up of AHT, specifically as it relates to the utilization of magnetic resonance (MR) venography to evaluate for CSVT. To that end, some institutions utilize contrast-enhanced MR venography in children with concern for AHT, as it is more sensitive for detecting thrombus than non-contrast MR venography [16–19]. While intracranial findings such as injured bridging veins are common in AHT, the prevalence of CSVT in AHT is unknown.

The objective of this study is to determine the prevalence of CSVT in a large AHT population and determine the relative prevalence of intra- and extracranial trauma in AHT victims with and without CSVT. Our hypothesis is that CSVT is associated with other abusive injuries and with witnessed or confessed abuse.

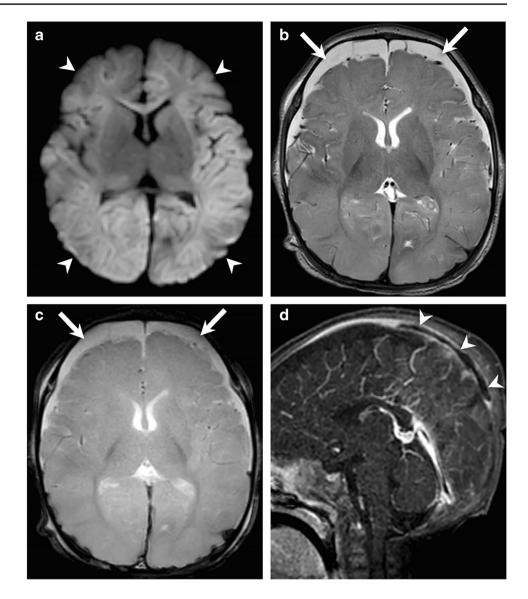
### **Materials and methods**

#### Study design

This is a single-center, retrospective study of patients who had a contrast-enhanced MR venogram during a hospitalization in which they were diagnosed with AHT. The study protocol was approved by the Colorado Multiple Institution Review Board.

# Setting, participant identification and data collection

The center is a 434-bed tertiary referral center with an established child protection team (CPT). Patients were identified from a CPT database of all children diagnosed with AHT since 2012. Inclusion criteria for this study were: diagnosis of AHT by the CPT at the Children's Hospital Colorado (Aurora, CO, USA) between Jan. 1, 2012 and Dec. 30, 2020, age <18 years at the time of presentation, and completion of a contrast-enhanced intracranial MR venogram during Fig. 1 A 3-month-old boy with abusive head trauma. a An axial diffusion-weighted magnetic resonance (MR) image shows diffuse cytotoxic edema throughout the supratentorial brain (arrowheads), largely sparing the deep gray nuclei. Axial T2-weighted (b) and (c) gradient recalled echo MR images reveal subdural hematomas about the bilateral convexities (arrows). d A sagittal contrast-enhanced, fatsuppressed, T1-weighted MR venogram image demonstrates a filling defect within the superior sagittal sinus (arrowheads)



the acute phase of their initial hospitalization. Data were extracted by clinicians and research assistants from the medical record.

Race and ethnicity were based on self-reported data from the medical record. Each patient was individually verified by a member of the CPT to confirm a diagnosis of AHT from the assessment of the multidisciplinary staffing note. Past medical history was included as documented in the initial hospitalization. Admission of harm was defined as admission from a caregiver to causing the injuries to a medical provider, or if the medical team was informed of an admission of harm per investigation by human services or law enforcement. All patients were evaluated for additional signs of trauma that included abdominal injuries, fractures, retinal hemorrhage and cutaneous injury.

#### Image and data analysis

A review of the imaging reports for all patients who met inclusion criteria was performed. The primary outcome measure was prevalence of CSVT in AHT, whereby a 95% normal proportion confidence interval (CI) was calculated. All positive or indeterminate cases of CSVT were examined by a board-certified pediatric neuroradiologist (D.M.M., 10 years of experience) to confirm the presence or absence of CSVT. While the assessors of outcomes were not formally blinded to the presence or absence of CSVT, outcomes were recorded before the planning of this retrospective study, and were recorded by different data operators than those who recorded the presence of CSVT, which minimizes the chance for confirmation bias. Any cases that remained

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1 2 3 4	у у у у	y n n	Bridging vein extending into posterior superior sagittal sinus Distal left transverse sinus	39 7
2 3 4	у	n		7
3 4	•			1
4	У		Posterior superior sagittal sinus (2 separate thrombi)	7,8
		у	Bridging veins extending into mid-superior sagittal sinus (2 separate thrombi)	5, 6
5	У	n	Left transverse sinus, sigmoid sinus, proximal internal jugular vein	103
6	у	У	Bridging vein extending into posterior superior sagittal sinus	8
7	у	У	Bridging vein extending into mid-superior sagittal sinus	4
8	у	n	Left distal transverse sinus, sigmoid sinus	82
9	n	n	Posterior superior sagittal sinus extending into the right transverse sinus; discontiguous clot within the junction of the right transverse and sigmoid sinus	98, 23
10	у	n	Posterior superior sagittal sinus	67
11	у	У	Bridging vein extending into mid-superior sagittal sinus	54
12	у	У	Bridging vein extending into mid-superior sagittal sinus	206
13	у	n	Mid- and posterior superior sagittal sinus	145
14	у	У	Bridging vein extending into mid-superior sagittal sinus	10
15	у	n	Right transverse sinus, sigmoid sinus	101
16	у	n	Right transverse sinus	30

**Table 2** Characterization of confirmed cerebral sinovenous thrombosis (CSVT) cases (present, n=16)

n no, y yes

indeterminate were adjudicated by a second pediatric neuroradiologist (N.V.S., 11 years of experience). The location and length of the CSVT was recorded, as well as whether the clot appeared to originate within an injured bridging vein and extend into the sinus (i.e. the bulk of the clot was within the bridging vein) versus originate within the sinus (i.e. the bulk of the clot was within the venous sinus). All cases were also examined for subdural hematomas, cytotoxic edema, parenchymal laceration, contusion, shear injury and subarachnoid hemorrhage.

### Results

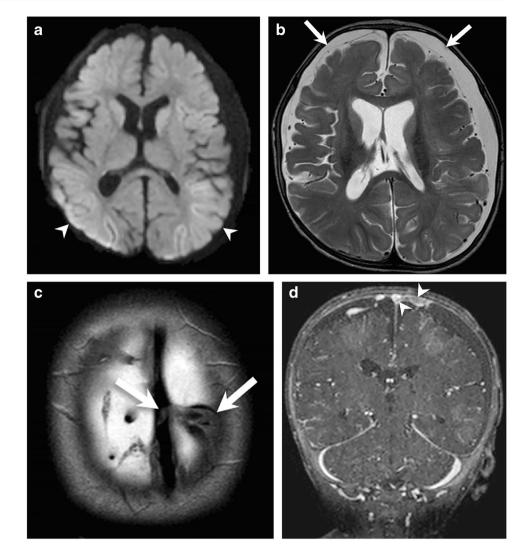
In all, 243 patients were identified with AHT who had contrast-enhanced MR venograms. Characteristics of the 243 identified participants are shown in Table 1. Participants were mostly white, with a slight male predominance (65%). CSVT was present in 7% (16/243; 95% CI, 4–11%) and indeterminate in 2% (5/243; 95% CI, 1–5%) of cases within the AHT population. The most common location for CSVT was within the superior sagittal sinus (Fig. 1, Table 2). Thrombi ranged from 4 to 206 mm in length with an average length of 53 mm (Table 2). Among confirmed cases of CSVT, 7 demonstrated evidence of the clot being centered upon an injured bridging vein and propagating back into the dural venous sinus (Fig. 2).

Subdural hematomas were present in 94% (228/243) of the total AHT population, and 94% (15/16) of the confirmed CSVT cases. Subdural hematoma was absent in one case of CSVT. There were, however, additional injuries including extracranial fractures and liver injury.

Among confirmed CSVT patients, 75% (12/16) were determined to have critical or greater (abbreviated injury scale [AIS]> or =5) severity of head injury, compared with 33% (82/243) in the total AHT population. Cytotoxic edema and subdural hematomas were reported in 88% (14/16) and 69% (11/16) of CSVT cases, respectively (Table 3). Brain contusions were present in 38% (6/16) of CSVT cases. Extracranial injuries associated with abuse were present in 100% (16/16) of confirmed CSVT cases. Specifically, 75% (12/16) with cutaneous injury, 56% (9/16) with retinal hemorrhage, 56% (9/16) with one or more fractures, and 19% (3/16) with abdominal visceral injury.

CSVT was treated with anticoagulation in 25% (4/16) of patients. The mean length of hospital and pediatric intensive care unit (PICU) stays increased from 10 to 21.9 and 3.5 to 7.3 days, respectively, for confirmed CSVT cases.

Fig. 2 An 8-month-old boy with abusive head trauma. a An axial diffusion-weighted magnetic resonance (MR) image shows faint cytotoxic edema involving the supratentorial cortices, most evident posteriorly (arrowheads). b An axial T2-weighted MR image demonstrates subdural hematomas about the bilateral convexities (arrows). left larger than right. c An axial T2-weighted MR image, at the cranial vertex, illustrates abnormal signal intrinsic to a left-side bridging vein that extends into the superior sagittal sinus (arrows). d A coronal contrastenhanced, fat-suppressed, T1-weighted MR venogram image confirms thrombus within a left-side bridging vein extending into the superior sagittal sinus (arrowheads)



#### Discussion

To the of best our knowledge, the prevalence of CSVT in AHT is not known. We found a low prevalence of CSVT and high prevalence of subdural hematoma in a large AHT population. These findings contradict the theory that CSVT is the cause of subdural hematomas [10–14]. To that end, if any subdural hematomas in our study resulted from CSVT, then these cases would likely be absent of other signs of abuse including subarachnoid hemorrhage, cytotoxic injury, brain contusions, parenchymal laceration, shear injury, bruising, fractures, abdominal injury and retinal hemorrhage; which was not the case. Our results do not support the theory that CSVT is often a nontraumatic source of subdural hematomas that mimic AHT. All cases had findings that raise significant concern for abuse, irrespective of the presence of subdural

hematoma or CSVT. Furthermore, a single case of CSVT lacked subdural hematoma. In our cohort, CSVT was associated with additional traumatic injuries and greater injury severity; increased AIS and PICU length of stay. This would suggest that CSVT is the result of critical illness and brain injury in an AHT child, not the cause of it. Compared to historical cohorts of children with CSVT that resulted from dehydration or metabolic illness, children in our cohort who had CSVT and were diagnosed with AHT had significant signs of abuse. Future studies with larger sample sizes should address this hypothesis, and look at clinical indicators (e.g., impact injury, dehydration, degree of swelling, location/size of ADH) that may predispose AHT patients to CVST. Given the low, but not insignificant, prevalence of CSVT demonstrated in this patient population, we support the use of MR venography in AHT to diagnose CSVT. Furthermore, in light

Subject ID	Age, sex	Medical history	Admission of harm	Other injuries associated with abuse	TBI	CSVT treatment
	1 month, male	None	No admission of harm.	Bruises, lacerations, abrasions, 6–10 fractures, bilateral retinal hemorrhages too numerous to count in all layers	SDH, SAH, cytotoxic edema, parenchymal laceration	None
7	23 months, male	None	GFOC made an admission of harm related to shaking the child.	Bruising, periorbital petechia, abdominal injury (liver), bilat- eral retinal hemorrhages too numerous to count in all layers	SDH, SAH, cytotoxic edema	None
e	3 months, male	Poor feeding. Multiple visits for jaundice but never placed under lights.	Father admitted to causing the fatal injuries to patient. Patient died during this admission.	1 fracture, bilateral retinal hemor- rhages too numerous to count in all layers	SDH, cytotoxic edema	None
4	9 months, male	Prematurity (<37 weeks), Com- plex history of short gut and gastroschisis. Prior reported short fall from 2.5 ft	Mother has admitted to grabbing patient's face, in frustration, to put his pacifier back in his mouth. Mother has also dis- cussed an additional fall off the couch, unwitnessed by her, when she left patient on the couch to go to the kitchen.	Bilateral cheek bruises, lesions, abrasions, bilateral retinal hem- orrhages too numerous to count	SDH, SAH, cytotoxic edema	None
S	6 years, male	Seizures, prematurity (<37 weeks), developmental delays, microschismcephaly, febrile seizures, history of shaken baby syndrome, vision impairments	Nanny admitted to abusing patient.	Bruising, poor dentition, 2 fractures, pneumomediastinum, internal bleeding	SDH, cytotoxic edema, brain contusion	None
9	2 months, male	Suspicion of exposure to sub- stances in utero	No admission of harm.	Retinal hemorrhages in all layers too numerous to count	SDH, SAH, cytotoxic edema, parenchymal laceration	None
7	3 years, female	Ongoing concern from babysitter of unexplained bruising. His- tory of burn.	No admission of harm made.	Bruising, abrasions, 6–10 frac- tures, abdominal injury (liver), optic nerve edema, oral/pharyn- geal injury	SDH, SAH, cytotoxic edema, brain contusion	None
×	22 months, female	None	No admission of harm. Inconsist- ent histories of injuries from caregivers.	Bruising, abrasions, bilateral retinal hemorrhages in all layers (20-50)	SDH, SAH	Anticoagulation
6	22 months, male	Prematurity (<37 weeks)	No admission of harm.	Bruising, abrasions, oral injuries, 3–5 fractures, abdominal injuries (liver), few preretinal hemorrhages	SAH, cytotoxic edema, brain contusion	Anticoagulation
10	3 months, male 3 months, female	Prematurity (<37 weeks) Prematurity (<37 weeks), Prior bruising	No admission of harm. MOC made an admission of harm.	Facial bruising 6–10 fractures, bilateral retinal henorrhages in all layers too	SDH, SAH, cytotoxic edema SDH, cytotoxic edema	None None

Table 3 (continued)	ntinued)					
Subject ID Age, sex	Age, sex	Medical history	Admission of harm	Other injuries associated with abuse	TBI	CSVT treatment
12	2 months, male	History of neglect	Father admitted to jerking infant 2–3 times from the floor without neck support out of frustration. Father also reported uninten- tionally dropping infant from a height of 2 ft and intention- ally dropping the infant from a height of 1 ft.	1 fracture, bilateral retinal hemor- rhages in all layers too numer- ous to count	SDH, SAH, cytotoxic edema, brain contusion, shear injury	Anticoagulation
13	2 weeks, male	NICU course after delivery and discharged home on oxygen.	No admission of harm.	Bruising, lesion, abrasion, bilat- eral retinal hemorrhages in all layers too numerous to count	SDH, SAH, cytotoxic edema, None parenchymal laceration, brain contusion	None
14	1 month, male	None	Mother states that she tried to grab him by the foot and his head slammed into the tub. Delay in seeking care.	l fracture	SDH, cytotoxic edema	None
15	20 months, male	Developmental delays, cutaneous injury	No admission of harm	Bruising	SDH	Anticoagulation
16	3 months, male	Bruising	No admission of harm	Bruising, 3–5 fractures, bilateral retinal hemorrhages in all layers too numerous to count	SDH, SAH, cytotoxic edema, None brain contusion	None
FOC father of	of child, FU follow-uj	FOC father of child, FU follow-up, GFOC grandfather of child, HIE hypoxic ischemic encephalopathy, IVH intraventricular hemorrhage, m months, MOC mother of child, NICU neonatal inten-	poxic ischemic encephalopathy, IVH	H intraventricular hemorrhage, $m$ mor	oths, MOC mother of child, NIC	U neonatal inten-

roc tand of child, rotonow-up, Or Oc granutation of child, 1112 hypoxy iscience electric checkinatopaury, 1711 intercent neuron neuron neuron age, m months, i sive care unit, POC parents of child, SAH subarachnoid hemorrhage, SDH subdural hemorrhage, SS sagittal sinus, TBI traumatic brain injury, y years

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of the unknown risks of gadolinium retention, non-contrast MR venography should suffice.

The major limitation of this study is that it represents a single-center cohort (n=243) whereby the population is comprised of a single geographical region with limited racial, ethnic and gender diversity. These findings need to be replicated across several diverse populations in order to generalize the evidence and potentially alter routine imaging protocols related to AHT.

### Conclusion

Cerebral sinovenous thrombosis was present in the minority of cases within a large abusive head trauma population; conversely, subdural hemorrhage was present in the majority of cases. All confirmed CSVT cases included additional injuries associated with abuse. CSVT was associated with greater head injury severity and a longer stay in the hospital and intensive care unit.

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#### Declarations

Conflicts of interest None

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