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ORIGINAL ARTICLE

Parasagittal vertex clots on head CT in infants with subdural hemorrhage as a predictor for abusive head trauma

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Abstract

Background Abusive head trauma (AHT) is the most common cause of subdural hemorrhage (SDH) in infants younger than 12 months old. Clot formation in the parasagittal vertex seen on imaging has been associated with SDH due to AHT. There have been very few studies regarding these findings; to our knowledge, no studies including controls have been performed.

Objective To describe parasagittal vertex clots on head computed tomography (CT) in infants with SDH and AHT compared to patients with SDH and accidental trauma, and to evaluate for parasagittal vertex clots in the absence of SDH in the setting of known accidental head trauma.

Materials and methods All infants younger than 12 months old with SDH present on CT scan were retrospectively identified from 2004 to 2014. Blinded, independent review of all CT scans for clot formation at the parasagittal vertex was performed by a pediatric neuroradiologist.

Results Ninety-nine patients were eligible for analysis. Mean age was 4 months. Fifty-seven (57.6%) were male. Fifty-five (55.6%) patients were identified as having AHT and 22 (22.2%) had accidental trauma. Forty-five (81.2%) patients with AHT had parasagittal vertex clots present on CT scan compared to 8 (36.4%) patients with accidental trauma. Compared to patients without parasagittal vertex clots, those with parasagittal vertex clots were more likely to have AHT (66.2% vs. 32.3%, P=0.001), no known mechanism of injury (69.1% vs. 32.3%, P=0.015), retinal hemorrhage (75% vs. 35.5%, P=0.002) and hypoxic-ischemic changes (25% vs. 0%, P=0.002). Patients with parasagittal vertex clots have eight times the odds of AHT compared to patients without parasagittal vertex clots. Age-matched control patients who underwent head CT scan due to a history of accidental head injury without SDH were identified (n=87); no patient in the control group had parasagittal vertex clots.

Conclusion The finding of parasagittal vertex clots on CT scans should raise suspicion for abuse and prompt further investigation, especially in the setting of no known, uncertain or inconsistent mechanism of injury.

Keywords Abusive head trauma · Child abuse · Computed tomography · Infants · Parasagittal vertex clots · Subdural hemorrhage

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Introduction

Abusive head trauma (AHT) is the leading cause of death due to physical abuse in children younger than age 5 [1] and is the most common cause of traumatic death in infants younger than 1 year [2–5]. AHT occurs in about 33–39 per 100,000 infants younger than 1 year of age [6]. Although rare, AHT causes significant morbidity and mortality with an estimated 60–70% of patients sustaining neurological deficits and an estimated mortality rate of 12–30% [3–5]. This clinical diagnosis often presents with the combination of findings of encephalopathy, subdural hemorrhage (SDH) and retinal hemor-

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rhage [7]. AHT is the most common cause of SDH in infants 1 to 12 months of age [3]. Before 1 month of age, birth trauma-related SDH is common; however, after 1 month, its presence is unlikely to be related to birthing [8]. SDH seen on head computed tomography (CT) is often one of the first clues to the diagnosis of AHT and SDH has been reported in up to 86% of infants with it [9]. SDH is associated with AHT but is not specific [3]. The diagnosis of AHT is challenging and requires a high index of suspicion as history is often vague, presenting symptoms are nonspecific and often there is no sign of external trauma [7]. Therefore, a test or finding that would help increase suspicion for AHT would be useful to clinicians.

SDH due to AHT is thought to be a result of bridging vein rupture from acceleration-deceleration and rotational forces [2, 10, 11]. The pattern of injury and SDH differs between abusive head injury and accidental head injury [12, 13]. Fall from a short distance is the most common cause of accidental head injury in infants, causing a translational force impact to the head [12]. Translational forces typically impact a localized area of the head resulting in focal skull fractures, contusions and/or subjacent SDH [10, 13]. In contrast, the angular deceleration mechanism of injury in AHT has a global impact that typically results in much more severe injury, such as shearing injury to axons and bridging veins leading to SDH [10, 13]. Bridging veins are prone to rupture during anteroposterior movements of the brain within the cranium, as they are short, non-tortuous and perpendicular to the sagittal sinus in the intradural segment [11, 14].

Bridging vein rupture in infants can be seen in up to 95% of fatal cases of AHT at autopsy [10] and is well documented in the pathology literature. Bridging vein rupture strongly suggests a traumatic mechanism of injury, which is useful in instances with vague or no history [14]. Extraluminal bridging vein thrombosis has been visualized on head imaging (CT and magnetic resonance imaging [MRI]) in patients with acute symptoms [15–18] and is suspicious for bridging vein injury. Clots are located in the parasagittal vertex region, where bridging veins drain into the superior sagittal sinus [16, 18]. Rambaud [14] identified ruptured bridging veins by looking for tubular-shaped clots at the vertex on CT or MRI. Choudhary et al. [18] describe a "lollipop sign" representing direct trauma to cortical veins referring to "a specific venous pattern that reflected parasagittal bridging veins that abruptly terminated in the region of an overlying subdural hemorrhage and hypothesized that this represents a bridging vein tear and subsequent post-traumatic thrombosis at the torn terminal end." These parasagittal vertex clots may be a useful finding to increase suspicion for AHT.

To date, there are no studies measuring the frequency of parasagittal vertex clots in infants with SDH and a diagnosis of AHT, compared to infants with SDH as a result of accidental trauma. Furthermore, no studies have looked for parasagittal vertex clots in patients with known accidental trauma in the absence of SDH Therefore, our primary aim was to record the frequency of parasagittal vertex clots on head CT in infants younger than 12 months old with SDH, and to compare those diagnosed with AHT to those without AHT. We also examined factors related to parasagittal vertex clots, and measured the frequency of parasagittal vertex clots in age-matched control patients with known accidental trauma without SDH.

Materials and methods

This study was approved by the Institutional Review Board at Children's Hospitals and Clinics of Minnesota. The study setting was an urban tertiary academic children's hospital emergency department with >100,000 visits per year between 2 campuses. The study period was Jan. 1, 2004, to Feb. 28, 2014. Before 2004, radiographic and electronic health record links were less complete.

All infants younger than 12 months old with SDH on CT scan were included in the study. Patients were identified by electronic health records using the following International Classification of Diseases, 9th edition, (ICD-9) code for SDH: (852.2). We chose CT scan as this is the recommended initial modality for imaging patients suspected of AHT. We selected infants younger than 12 months old because AHT is the most common cause of SDH [3]. Demographic data, presenting signs and symptoms, provided mechanism of injury, skeletal survey findings, presence of retinal hemorrhage and final diagnosis from a comprehensive child abuse team evaluation were abstracted from the electronic health record using a standardized case report form by M.M.R., a pediatrician with 8 years of experience including fellowship training in pediatric emergency medicine. The presence of retinal hemorrhage was established by an examination performed by a pediatric ophthalmologist. A skeletal survey was determined to be positive if a pediatric radiologist noted evidence of acute or healing fracture. Skull fracture diagnosis was determined by CT scan.

Patients were excluded if a CT scan was not available for review, the patient had a history of congenital structural or vascular neurological abnormality, history of neoplasm, previous neurological surgery, history of thrombophilia, known coagulopathy other than hemophilia, the inability to evaluate parasagittal vertex clots on CT due to obscuration of this finding by large volume SDH, or a diagnosis of sepsis.

A pediatric neuroradiologist (R.J.P., with >30 years of experience) blinded to patient history and diagnosis independently reviewed all CT scans and recorded the presence of clot formation in parasagittal vertex, as well as the number, type and location of SDH, and the presence of hypoxic-ischemic imaging

changes on a standardized case reporting form. Radiologic definitions for SDH, parasagittal vertex clots and hypoxic-ischemic changes are as follows. SDH were defined as increased attenuation within the subdural space either as linear if along the falx cerebri or falx cerebelli or crescentic if over cerebral or cerebellar convexities. SDH was differentiated from subarachnoid hemorrhage as blood extending into the sulci was defined as subarachnoid hemorrhage, while blood that was curvilinear was defined as SDH. Parasagittal vertex clots were defined as patchy extra-axial foci of increased attenuation exceeding the diameter of cortical veins and located over the cerebral convexity within 2 cm of the midline between the coronal and lambdoid sutures (Figs. 1 and 2). This location is where the cortical veins pierce the dura mater and enter into the sagittal sinus. Hypoxic-ischemic imaging changes were focal or diffuse obscuration of normal grey/white matter differentiation, accompanied by decreased attenuation in this distribution on initial CT imaging. Sagittal and coronal reformatted images were used when available; if unavailable, only axial images were used. The majority of head CT studies were performed on the two primary campuses of our institution. In 2012, a Toshiba (Canon Medical Systems, Tustin, CA) 16-slice Aquilion scanner was replaced with a Toshiba 64-slice Aquilion scanner on one campus, and in 2006, a GE (GE Healthcare, Waukesha, WI) 4-slice LightSpeed scanner was replaced with a Toshiba 64-slice Aquilion scanner on the other campus.

A diagnosis of AHT was established by the consensus of a multidisciplinary child abuse team. This multidisciplinary team included child abuse pediatricians, pediatric neurosurgeons, pediatric radiologists, pediatric ophthalmologists, pediatric nurse practitioners and social workers. To further characterize the level of certainty of diagnosis, we modeled our criteria after Kemp et al. [19]: "a definition of abuse was based upon the outcome of a legal or multi-agency child protection investigation or stated criteria where diagnosis was based upon additional features and did not rely on the neuroradiological findings, to minimize the risk of circularity in a condition where there is no 'gold standard' diagnostic test. [Accidental trauma] was defined as a witnessed traumatic event, where the cause of injury was stated or where child abuse had been actively excluded."

Previously validated criteria by Kemp et al. [19] were applied to our patient population (Table 1). Levels 1, 2 and 3 criteria were considered a diagnosis of abuse. "Stated criteria" and "multidisciplinary assessment" (levels 2 and 3, respectively) were determined as a diagnostic of abuse following the final assessment of the comprehensive child abuse team evaluation. Levels 4 and 5 were categorized as "suspected abuse only" by the child abuse team. The presence of parasagittal vertex clots was not included in the diagnosis of AHT.

Statistical analysis

Descriptive statistics were used to examine the patient population. Comparisons of baseline characteristics were analyzed using *t*-tests for continuous variables and $\chi 2$ or the Fisher exact test for categorical variables. Sensitivity, specificity, positive predictive value and negative predictive value were calculated with the 2-tailed *t*-test, with confidence intervals [CI] for presence of parasagittal vertex clots and AHT. Logistic regression was used to examine the association between parasagittal vertex clots (yes/no) or retinal hemorrhage (yes/no) and AHT (yes/no). An additional regression model examined the association between parasagittal vertex clots, retinal hemorrhage and AHT. For this model, we created a combined variable with four categories (normal, parasagittal vertex clots only, retinal hemorrhage only, both parasagittal vertex clots and retinal hemorrhage). $P<0.05$ was considered statistically significant. Data analysis was performed using SPSS (IBM, version 23, Armonk, NY).

Our primary analysis included only patients with SDH. To determine whether parasagittal vertex clots are found in patients without SDH we also reviewed CT scans for 87 age-matched infants with known accidental trauma to calculate the rate of parasagittal vertex clots in the absence of SDH.

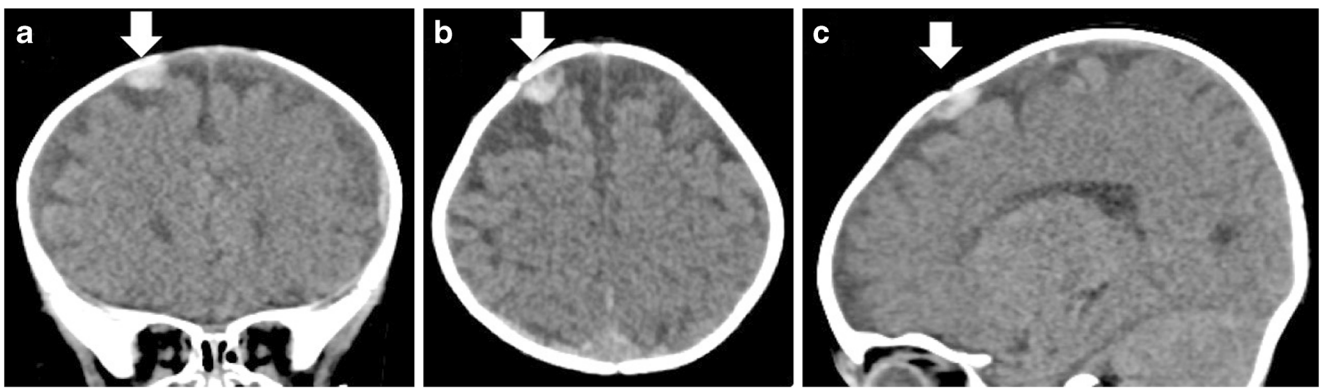

Fig. 1 Parasagittal vertex clots (*arrows*) seen on non-contrast head CT in a 5-month-old boy with abusive head trauma. Coronal view (**a**), axial view (**b**) and sagittal view (**c**)

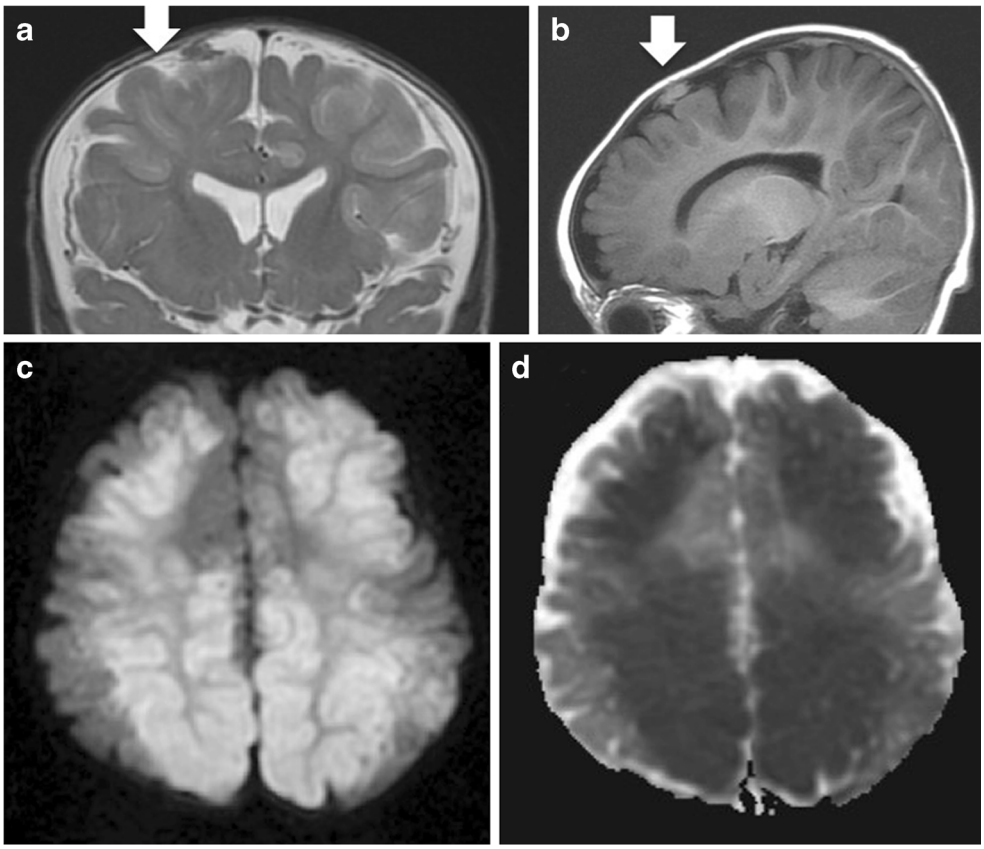

Fig. 2 Parasagittal vertex clots (*arrows*) on MRI in the same 5-month-old boy as in Fig. 1. A. Coronal fast spin echo (FSE) T2 sequence (**a**), sagittal T1 fluid-attenuated inversion recovery (FLAIR) sequence (**b**), axial diffusion-weighted image (B=1,000) (**c**) and corresponding axial apparent diffusion coefficient (ADC) map image (**d**)

Results

From 2004 to 2010, there were 137 infants with SDH on CT scan. Of these patients, 38 met exclusion criteria and 99 were eligible for analysis (Fig. 3). Demographic data and overall study characteristics of our patient population are presented in Table 2. Mean age was 4 months (interquartile range: 5 days to 11.3 months). Most patients were male (57.6%), full term (81.8%), and had no known mechanism of injury identified (57.6%). AHT occurred in 55.6% of patients (n=55) and was the most common cause of head trauma. AHT was suspected in 22.2% of patients (n=22). There were no siblings represented in the study population.

Accidental head trauma was identified as the cause of head injury in 22.2% of patients (n=22). Of patients with AHT, 81.2% had parasagittal vertex clots present on CT scan compared to 36.4% of patients with accidental trauma (n=45 and n=8, respectively). Additionally, 68.2% patients with suspicion for AHT had parasagittal vertex clots present on CT scan (n=15). We found that parasagittal vertex clots on CT scan had a sensitivity of 0.82 (95% CI: 0.69–0.90) and a specificity of 0.64 (95% CI 0.41–0.82) for AHT (Table 3). The positive predictive value of parasagittal vertex clots for AHT was 0.85 (95% CI: 0.72–0.93) and the negative predictive value was 0.59 (95% CI: 0.37–0.77).

There was no significant association between age, gender, ethnicity/race, prematurity, skeletal survey results or skull fracture and parasagittal vertex clots (Table 2). Compared to patients without parasagittal vertex clots, those with parasagittal vertex clots were more likely to have abuse (66.2% vs. 32.3%, P=0.001), no known mechanism of injury

Table 1 Definitions of child abuse [19]

Categories	Criteria used to define abuse
1	Abuse confirmed at case conference, family, civil or criminal court proceedings or admitted by perpetrator or witnessed
2	Abuse confirmed by stated criteria, including multidisciplinary assessment
3	Abuse defined by stated criteria
4	Abuse stated but no supporting detail given
5	Suspected abuse

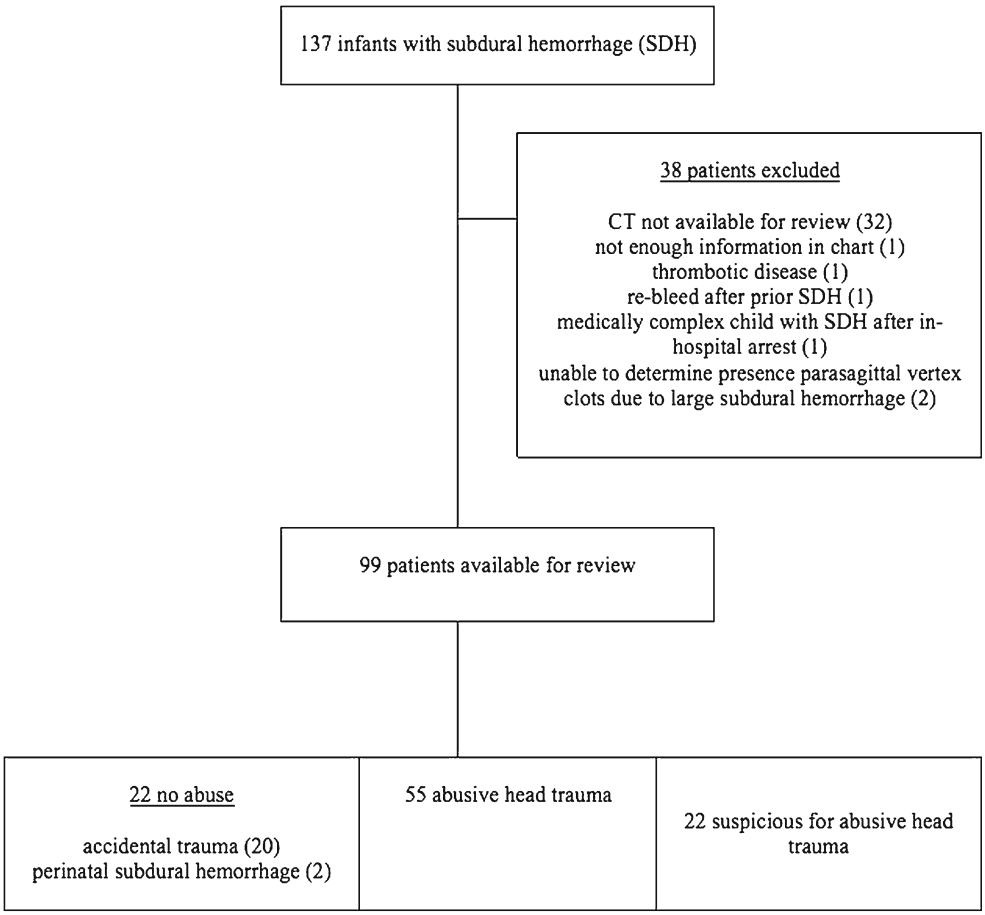

Fig. 3 Patient flow

(69.1% vs. 32.3%, *P*=0.02), retinal hemorrhage (75% vs. 35.5%, *P*=0.002) and hypoxic-ischemic changes on head CT (25% vs. 0%, *P*=0.002). Although hypoxic-ischemic imaging changes were found in only 17.2% of patients, only patients with parasagittal vertex clots and AHT or suspected AHT were found to have these changes.

The odds of having confirmed abuse were almost eight times higher in patients with parasagittal vertex clots compared to patients with known accidental trauma without parasagittal vertex clots (odds ratio [OR]=7.88, 95% CI: 2.6–23.8) (Table 4). The odds of having confirmed abuse were almost 10 times higher in patients with retinal hemorrhage compared to patients without retinal hemorrhage (OR=9.38, 95% CI: 1.9–47.2) In a model comparing patients with only parasagittal vertex clots, only retinal hemorrhage, or both parasagittal vertex clots and retinal hemorrhage to patients without parasagittal vertex clots or retinal hemorrhage, patients with the combination of parasagittal vertex clots and retinal hemorrhage had extraordinarily increased odds of having abuse (OR=152, CI: 14–1,600) (Table 5).

In the separate analysis of 88 age-matched infants who had a head CT scan for known accidental head trauma, but did not have SDH, there were no parasagittal vertex clots. AHT was ruled out in these patients by a comprehensive child abuse team evaluation. There were varying types of accidental trauma mechanisms among these infants, the most common of which included falls while being held by a parent, caregiver or sibling.

Discussion

AHT is the leading cause of death from child physical abuse and is the most common cause of SDH in infants 1 to 12 months of age [3, 7, 20]. SDH, retinal hemorrhage and hypoxic-ischemic encephalopathy are associated with AHT [20]. The American Academy of Pediatrics (AAP) recommends that all children with suspected AHT have head imaging and evaluation for retinal hemorrhage by an ophthalmologist as part of the work-up [19]. The diagnosis of AHT is challenging and takes time and resources [3, 7, 20]. Therefore, a finding that can aid in diagnosis or increase suspicion for AHT is useful to clinicians. We found that parasagittal vertex clots in infants with SDH were associated with AHT. Infants with SDH and parasagittal vertex clots were almost eight times more likely to have AHT than infants with SDH from accidental head trauma and no parasagittal vertex clots. AHT was the most common cause of SDH in our study and is

Table 2 Demographic and clinical information

Variable		Total	PSVC (−)	PSVC (+)	P^a
n		99	31 (31.3)	68 (68.7)	
Age, mean (SD), y		0.39 (0.21)	0.43 (0.26)	0.37 (0.18)	0.21
Gender	Female	42 (42.4)	17 (54.8)	25 (36.8)	0.092
	Male	57 (57.6)	14 (45.2)	43 (63.2)	
Ethnicity	White/Caucasian	60 (60.6)	20 (64.5)	40 (58.8)	0.91
	Hispanic/Latino	12 (12.1)	4 (12.9)	8 (11.8)	
	Black/African American	10 (10.1)	2 (6.5)	8 (11.8)	
	Biracial	7 (7.1)	2 (6.5)	5 (7.4)	
	Asian	3 (3.0)	1 (3.2)	2 (2.9)	
	American Indian/Alaskan	2 (2.0)	0 (0)	2 (2.9)	
	Declined/Unknown	5 (5.1)	2 (6.5)	3 (4.4)	
Mechanism of injury	No known injury	57 (57.6)	10 (32.3)	47 (69.1)	0.015
	Accidental trauma	42 (42.4)	21 (67.7)	21 (30.9)	
Prematurity[b]	No	81 (81.8)	28 (90.3)	53 (77.9)	0.14
	Yes	18 (18.2)	3 (9.7)	15 (22.1)	
Retinal hemorrhage	No	19 (19.2)	11 (35.5)	8 (11.8)	0.002
	Yes	62 (62.6)	11 (35.5)	51 (75.0)	
	No exam performed	18 (18.2)	9 (29.0)	9 (13.2)	
Skeletal survey	Negative	60 (60.6)	19 (61.3)	41 (90.3)	0.13
	Positive	28 (28.3)	6 (19.4)	22 (32.4)	
	No skeletal survey performed	11 (11.1)	6 (19.4)	5 (7.4)	
Skull fracture[c]	Negative	95 (96.0)	30 (96.8)	65 (95.6)	0.90
	Positive	4 (4.0)	1 (3.2)	3 (4.4)	
Hypoxic-ischemic imaging changes[d]	No	82 (82.8)	31 (100.0)	51 (75.0)	0.002
	Yes	17 (17.2)	0 (0)	17 (25.0)	
Abuse criteria [19]	Abuse (criteria 1, 2 and 3)	55 (55.6)	10 (32.2)	45 (66.2)	0.001
	Suspected abuse (criteria 4 and 5)	22 (22.2)	7 (22.6)	15 (22.0)	
	No abuse	22 (22.2)	14 (45.2)	8 (11.8)	

Values are expressed as *n* (%) unless noted otherwise

[a] *P*-value from *t*-test (continuous variables),Fisher exact test or χ^2 (categorical variables)

[b] Prematurity defined as gestational age <37 weeks

[c] Skull fracture present on CT scan

[d] Hypoxic-ischemic imaging changes defined as focal or diffuse obscuration of normal grey/white matter differentiation, accompanied by decreased attenuation in this distribution

PSVC parasagittal vertex clots, *SD* standard deviation

consistent with previous studies [3]. The majority of our patients' subdural hemorrhages were unexplained, which is also common among infants who are abused [7]. In our study population, only patients with SDH had parasagittal vertex clots. Our patients with parasagittal vertex clots were more likely to have retinal hemorrhage and hypoxic-ischemic imaging changes on CT than those without parasagittal vertex clots. These findings suggest that parasagittal vertex clots are related to the often identified findings of AHT: SDH, retinal hemorrhage and hypoxic-ischemic encephalopathy. Our findings suggest that parasagittal vertex clots may be a novel predictor of AHT.

Table 3 Sensitivity and specificity

	Accidental head trauma *n* (%)	Abusive head trauma *n* (%)	Total *n* (%)
PSVC (+)	8 (36.4)	45 (81.2)	53 (68.8)
PSVC (−)	14 (63.6)	10 (18.2)	24 (31.2)
Total	22 (28.6)	55 (71.4)	77 (100)

Sensitivity: 81.8% (95% CI: 68.6–90.5), specificity: 63.6% (95% CI: 40.8–82.0), positive predictive value: 84.9% (95% CI: 71.9–92.8), negative predictive value: 58.3% (95% CI: 36.9–77.2)

CI confidence interval, *PSVC* parasagittal vertex clots

This is the first study to describe the sensitivity and specificity of parasagittal vertex clots for abuse. Sensitivity and specificity of parasagittal vertex clots on CT scan for AHT were 81.8% and 63.6%, respectively. In our population, parasagittal

Table 4 Odds ratio of abuse for parasagittal vertex clots and retinal hemorrhage alone

	Odds ratio [OR] (95% CI)	*P*
Parasagittal vertex clots		
No	Reference	
Yes	7.9 (2.6–23.8)	<0.01
Retinal hemorrhage		
No	Reference	
Yes	9.4 (1.9–47.2)	<0.01

CI confidence interval

vertex clots on CT had a moderate positive predictive value (84.9%), indicating that patients who have parasagittal vertex clots on CT scan may benefit from further evaluation for abuse. The low negative predictive value in our population (53.8%) indicates that patients without parasagittal vertex clots on CT scan with history or other findings concerning for AHT should continue to get work-up for abuse. Although parasagittal vertex clots were significantly associated with abuse in our patient population, eight patients with known accidental trauma had parasagittal vertex clots on CT. Parasagittal vertex clots alone are not diagnostic for abuse but should raise suspicion for abuse. CT scan is the initial modality of head imaging recommended by the AAP in the work-up of symptomatic patients with suspected AHT [20] and allows for the ability to assess for parasagittal vertex clots without additional resource use.

There is a paucity of literature regarding the association between clots in the parasagittal vertex and AHT, however, previous smaller case series have described clots in the same area in infants with SDH due to AHT. Parasagittal vertex clots are located in the parasagittal vertex region, the region where bridging veins drain into the superior sagittal sinus [16]. This is the same region where torn bridging veins have been visualized postmortem, and where previous studies have described clot formation on head imaging in infants with SDH and AHT [16–18, 21–23]. Yilmaz et al. [15] describe two infants with AHT and bilateral multifocal signal loss at bridging veins on susceptibility-weighted imaging, and proposes that these findings represent clot formation within injured bridging veins. Similarly, Adamsbaum and Rambaud [16] describe bridging vein thrombosis on head CT and MRI, suggesting evidence for AHT as the cause for SDH in an infant with a vague history. Hahnemann et al. [17] describe bridging vein thrombosis seen on MRI in the shape of a tadpole in five infants with confirmed physical abuse and six infants with strongly suspected physical abuse.

Table 5 Odds ratio (OR) for abuse for combined variable

	OR (95% CI)	P
Odds ratio		
Normal	Reference	
Only PSVC	4.00 (0.78–20.68)	0.98
Only retinal hemorrhage	14.00 (1.86–105.27)	0.10
PSVC and retinal hemorrhage	152.00 (14.43–1,600.82)	< 0.01

PSVC parasagittal vertex clots

In our study, parasagittal vertex clots are defined as discreet, patchy extra-axial foci of increased attenuation, exceeding the diameter of cortical veins, over the cerebral convexity within 2 cm of midline, between coronal and lambdoid sutures. This definition is consistent with previous study description of bridging vein thrombosis [15–17]. In our experience, parasagittal vertex clots are best seen on coronal and sagittal CT reconstruction images through the parasagittal region. Sagittal and coronal reconstructions show the clots well and allow for the distinction between parasagittal vertex clots and subdural blood. We recommend if an infant is identified with SDH, that axial, coronal and sagittal reconstructions should be viewed to assess for parasagittal vertex clots.

We presume that the sequence of parasagittal clot formation involves injury to the bridging veins leading to perivenous clot formation resulting in venous thrombosis. While the exact sequence of events is unknown, the injured bridging vein must likely thrombose transiently in response to injury limiting the extent of bleeding. Parasagittal vertex clots are patchy and focal rather than curvilinear, which distinguishes them from SDH, which is also gravity dependent. With the exception of a very large SDH, parasagittal vertex clots can be visualized separately due to the gravity-dependent nature of subdural hemorrhages. As cortical veins are variable in diameter, it is difficult to have exact measurement specifications to define a parasagittal vertex clot. The finding relies on the location and relationship of the size of the clot compared to the diameter of the cortical vein. The parasagittal vertex clot is typically larger than the diameter of the adjacent cortical vein suggesting a component of extraluminal blood. We believe parasagittal vertex clots are the CT equivalent of the "lollipop sign" reported by Choudhary et al. [18], as the clots are in the same location and relationship to the bridging vein as the "lollipop sign" and can be seen on CT and MRI (Figs. 1 and 2).

A few specific patient characteristics warrant further discussion. It is interesting that while patients with parasagittal vertex clots more often had positive skeletal surveys, the association was not statistically significant. Another interesting finding included six patients with positive skeletal surveys but negative head CT studies for parasagittal vertex clots. These findings suggest that the mechanisms producing injury to the axial or appendicular skeleton may be different from those producing parasagittal vertex clots. It may also reflect a bias

toward performance of a skeletal survey in those infants identified with parasagittal vertex clots compared to infants without these clots. Patients lacking parasagittal vertex clots should still undergo comprehensive evaluation for abuse when clinical suspicion exists.

One study patient with hemophilia A (factor VIII deficiency) and accidental trauma did not exhibit parasagittal vertex clots. It is possible that clots may not form in patients with hemophilia. Patients with congenital or acquired forms of coagulopathy and SDH still warrant evaluation for abuse when concerns exist. In a separate evaluation of the patients that met exclusion criteria with available CT, one had thrombotic disease and one was a medically complex patient with an in-hospital cardiac arrest. Neither patient had suspicion of abuse nor parasagittal vertex clots. Parasagittal vertex clots seen in patients with thrombotic disease should still prompt concern and work-up for the possibility of abuse.

The subdural hemorrhages were so large in two patients that parasagittal vertex clots were unable to be assessed. "Large volume" is a subjective description for collections larger than the thin crescent shape of blood typically seen. Large subdural hemorrhages can obliterate the finding of patchy foci distinctive of parasagittal vertex clots. The parasagittal vertex clot may still be present, but may be hidden due to a large SDH. In this circumstance, it is likely there is associated brain edema or other factors that would raise concern for abuse.

Our study did not evaluate the presence of parasagittal vertex clots in asymptomatic term neonates with birth-related SDH. Our study included four patients who were <1 month of age, two had perinatal SDH and no parasagittal vertex clots, one had accidental SDH and no parasagittal vertex clots, and one had confirmed AHT with parasagittal vertex clots. It would be unlikely for parasagittal vertex clots to be seen in neonates with SDH secondary to birth trauma as SDH after birth trauma is usually "combined supratentorial and infratentorial usually along the tentorium, over cerebellar hemispheres, interhemispheric fissure, occipital or parietal occipital lobes [24]." Parasagittal vertex clots should prompt further work-up for AHT in neonates with SDH.

Our study has several limitations, including a small sample size. Although we had a small sample size, our study population was larger than previous case series describing parasagittal vertex clots. Yilmaz et al. [15] described two cases, Adamsbaum and Rambaud [16] described three cases, and Hahnemann et al. [17] described 81 cases. The confidence intervals for odds ratios are broad due to the small sample size; however, the magnitudes of the odds ratios are consistent likely reflecting true significance. Another limitation is our retrospective study design. The diagnosis of AHT is infrequent, and a retrospective design allowed data to be collected in a short amount of time.

In 2011, our radiology department developed a protocol for head CT scans to routinely reformat axial data to produce coronal and sagittal reconstructions. Before 2011, studies may have only included axial images; in our study, outside CT scans often only had axial images. Parasagittal vertex clots are more easily seen on coronal and sagittal reconstructions, therefore, some parasagittal vertex clots could have been missed due to image acquisition in axial format alone. It is possible that the presence of parasagittal vertex clots was overestimated in the setting of accidental trauma, as AHT may have been missed. However, these patients were reviewed by the child abuse team and AHT was unlikely. There is also the possibility that we lacked information on confounding variables, although potential confounding variables such as hemophilia, thrombophilia, neoplasm, sepsis, congenital vascular malformation or structural neurological abnormality were accounted for as these patients were excluded from the study. Finally, this is a single-center study, and results may not apply to other institutions. Further research is needed to describe the relationship between parasagittal vertex clots, retinal hemorrhage and hypoxic-ischemic encephalopathy in AHT. A multicenter prospective study design would help further characterize these relationships. Future studies should include a standardized CT protocol to include axial, coronal and sagittal imaging to enhance identification to further characterize the course for development of parasagittal vertex clots and to determine the timing for resolution on subsequent head imaging. In addition, larger studies looking at parasagittal vertex clots on MRI are needed as well as a pilot study to see if parasagittal vertex clots can be seen by ultrasound.

Conclusion

This is a unique study describing the presence of parasagittal vertex clots in patients with SDH and AHT or accidental head trauma, and in patients with accidental head trauma without SDH. The finding of parasagittal vertex clots on CT scan should raise suspicion for abusive head trauma and prompt further investigation, especially in the setting of no known, uncertain or inconsistent mechanism of injury.

Compliance with ethical standards

Conflicts of interest None

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